Ramsar Technical Report No. 7

Ramsar Wetland Disease Manual

Guidelines for Assessment, Monitoring and Management of Animal Disease in Wetlands

Ramsar WWT

This manual was prepared by the Wildfowl & Wetlands Trust (WWT) for the Ramsar Convention on Wetlands

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Cover photo: Wildlife biologist undertaking waterbird disease surveillance at Dagona Sanctuary Lake (Ramsar Wetland of International Importance), Nigeria (*Ruth Cromie*).

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Ramsar Technical Reports are designed to publish, chiefly through electronic media, technical notes, reviews and reports on wetland ecology, conservation, wise use and management, as an information support service to Contracting Parties and the wider wetland community in support of implementation of the Ramsar Convention. All Ramsar Technical Reports are peer-reviewed by the members and observers of the STRP and independent experts.

Ramsar Technical Reports are published in English in electronic (PDF) format. When resources permit, they are also published in French and Spanish, the other official languages of the Ramsar Convention.

Contents

Foreword

Perhaps there are no better settings to illustrate the importance of a ‘One World, One Health’ approach than within wetlands. Well functioning wetlands provide the services, resources and means by which people, livestock and wildlife can remain healthy. The health of these sectors cannot be viewed independently as complex interactions and their interdependence means that the health of one affects the health of the others. Wise use of wetlands helps to promote health and this has been acknowledged by the substantial body of work conducted by the Ramsar Convention’s Secretariat and Scientific and Technical Review Panel (STRP) on Healthy Wetlands, Healthy People.

In 2005, the issue of highly pathogenic avian influenza brought specific animal diseases in wetlands to the attention of the Ramsar Convention and it was quick to address some of the problems relating to wetland and waterbird conservation at the 9th meeting of the Conference of the Contracting Parties (COP) to the Ramsar Convention later that same year. Resolution IX.23 provided a clear acknowledgement that disease control operations needed to remain within the context of wise use. At COP 9, this multifaceted global issue resulted in a request for guidance from Contracting Parties which was provided as a substantive Annex to Resolution X.21 in 2008 (latterly repackaged as Ramsar Handbook No. 4). Resolution X.21 instructed STRP to develop practical guidance on the prevention and control of other diseases of either domestic or wild animals in wetlands, especially those diseases that have implications for human health, and how such guidance can be best incorporated into management plans at Ramsar sites and other wetlands.

This Manual addresses that request and provides wetland managers and other wetland stakeholders with a better understanding of their key role in health management in wetlands.

Anada Tiéga

Secretary General
Ramsar Convention on Wetlands

Preface

When confronted by a disease outbreak in a wetland, a problem analysis almost always tracks back 'upstream' and finds an anthropocentric reason – land use, pollution, abstraction, livestock, introduced species, or such like. It becomes easy to see that decisions made about the management of a wetland hold the key to disease prevention and thus that the wetland managers are the holders of this key. They, together with the decision makers, are the ones who can make a difference to health in wetlands. But do these personnel have the understanding of how to do the right thing for disease prevention and control? And do they understand the importance of the complex interactions between humans, their livestock and wildlife when managing health in wetland ecosystems?

It was heartening to see the guidance on highly pathogenic avian influenza H5N1 being well received at the 10th Conference of the Contracting Parties (COP) to the Ramsar Convention in 2008 (Resolution X.21). The request for further guidance on animal diseases in wetlands came from the Tanzanian delegate speaking on behalf of the Africa Region. Coincidence or otherwise, it was a Tanzanian wildlife manager responsible for one of the planet's high profile protected areas who said, following a taught module on wildlife health, that he "hadn't thought about wildlife in that way before" and that he would "make practical changes and do things differently". To try to better understand the nature of guidance needed for wetland health management, we conducted a needs survey of wetland professionals - there was a clear desire for practical guidance.

Although designed for wetland managers, it was difficult to know just how to pitch this Manual given the enormous range of personnel responsible for managing wetlands across the world. We hope it is helpful and provides the wetland manager with some insight and a practical manual to help to "do things differently".

Overview of this Manual

This Manual is intended to provide the reader with an overview of the principles and practicalities of managing animal diseases at wetland sites. The Manual highlights the importance of including disease prevention and control in wetland management plans and provides guidelines on how to do so. It should be used in conjunction with Ramsar Handbook No. 18 on *Managing Wetlands*[1].

This Manual draws on a range of primary sources – interpreting and re-packaging the information for the wetland manager and the wetland policy maker. This Manual is not intended as in-depth technical guidance for dealing with specific disease issues, but as a primer describing the key components of disease prevention and control strategies and directing the reader to the primary sources, where more information can be obtained.

The Manual is divided into five chapters accompanied by this Overview and a selection of Appendices ►**Figure 0-1**.

►**Table 0-1** provides a summary of what you can expect to find in each chapter.

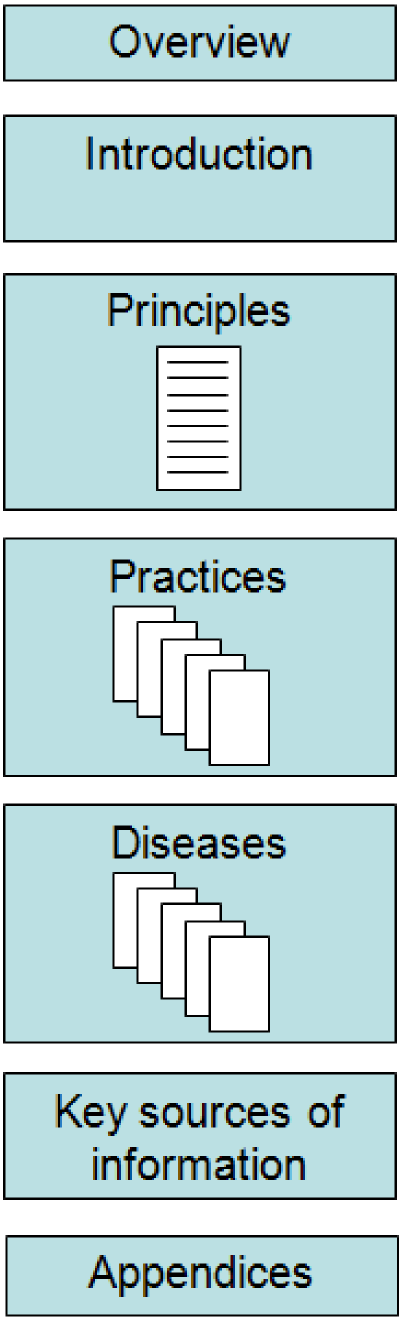

Figure 0-1.
Structure of this manual.

[1] *Managing wetlands: Frameworks for managing Wetlands of International Importance and other wetland sites.* Ramsar handbooks for the wise use of wetlands, 4th edition, vol. 18. Ramsar Convention Secretariat, Gland, Switzerland.

Table 0-1. Summary of the contents of each chapter of this Manual

1	**Introduction**	▪ A **general introduction to animal diseases in wetlands** – what they are and why they are a growing problem. ▪ A **summary of the impacts of wetland disease** on biodiversity, livestock and human health and its economic implications. ▪ The **scope, aim and objectives** of this Manual.
2	**Principles of Disease Management in Wetlands**	▪ An **introduction to disease management in wetlands** – important concepts. ▪ A look at the **disease relationship** between wildlife, livestock and humans. ▪ An introduction to taking an **ecosystem approach to health**. ▪ Why disease should be an **integral to wetland management**. ▪ **Invasive alien species** and the parallels for disease control. ▪ A **summary of current strategies** for managing animal diseases in wetlands, including proactive strategies for preventing disease and reactive strategies for controlling disease. ▪ An introduction to the role of **communication, education, participation and awareness** in disease management.
3	**General Management Practices**	▪ A standardised procedure for completing a **disease risk assessment**. ▪ Guidance on **how to incorporate disease management into management plans** for wetlands. ▪ Guidance for **reducing the risk of emerging disease**. ▪ Guidance for **detecting, assessing and responding to disease outbreaks**. ▪ Guidance for **managing disease**. ▪ Guidance for **communication, education, participation and awareness**. ▪ **Case studies:** Descriptions and photos of wetland managers' experiences responding to disease problems.
4	**Animal Diseases Currently Causing Concern in Wetlands**	▪ A summary of some of the **animal diseases currently causing concern** in wetlands. ▪ **Key questions to ask when a disease is detected**: geographic extent, wetland characteristics, host range, seasonality, transmission, field signs and potential impacts. ▪ **Factsheets** on a selection of diseases currently impacting wetlands providing a brief description of the disease and the methods used for prevention and control.
5	**Where to go for Further Assistance and Advice**	▪ A list of key international and regional **contacts**. ▪ A **bibliography** of key resources providing information and guidance on disease management.

Who should use this Manual

This Manual focuses on the information needs of:

1. **Wetland managers**, meaning persons or agencies with an interest in the continuing existence of wetlands and in protecting them. Wetland managers can be any of a number of entities, including owners of properties that contain wetlands, staff of government agencies that have regulatory power over them, and conservation organisations with an interest in wetlands or holders of conservation interests. This Manual is particularly targeted at those **wetland managers who are involved in producing or implementing wetland management plans**, from the site level to the regional level.

2. **Wetland policy makers**, meaning persons or agencies responsible for policy which may impact wetland sites or ecosystems.

How to use this Manual

Given that it is unlikely that the reader will read the entire Manual from cover to cover (indeed it is not designed for this) there is some repetition of key concepts of disease emergence, prevention and control in wetlands - *this is intentional*.

If you are a wetland manager…

We recommend that you read Chapters 1 and 2 in full, which provide an introduction to disease in wetlands and the principles of disease management in wetlands. These chapters explain the most important concepts in this Manual, namely why disease management is important, how to approach developing disease management strategies and the importance of considering disease management from an ecosystem perspective.

Whilst we recommend that you also read Chapters 3, 4 and 5, it is not necessary to read all the chapters or the sections of each chapter in chronological order. The reader is encouraged to begin with a topic of interest and follow the links and references included in the text for guidance to other chapters and sections.

►**Appendix II** provides a ‘Summary of Key Messages for Wetland Managers and Policy Makers’.

If you are a wetland policy maker…

We recommend that you read Chapters 1 and 2 in full, which provide an introduction to disease in wetlands and the principles of disease management in wetlands. These chapters explain the most important concepts in this Manual, namely why disease management is important, how to approach developing disease management strategies and the importance of considering disease management from an ecosystem perspective.

Chapters 3 and 4 provide more detailed information on specific diseases and management practices and may be of less relevance to the policy maker. We recommend, however, that the introductions to these Chapters and a sample of the other sections are read to illustrate some of the practical challenges facing wetland managers. The following sections may be of particular use:

► **Section 3.1 Assessing risk and planning for the future**

► **Section 3.2 Reducing risk of disease emergence**

As Chapter 5 contains lists of sources of further information, this will be useful to a policy maker seeking further information on any of the Manual's topics; and ►**Appendix II** provides a 'Summary of Key Messages for Wetland Managers and Policy Makers'.

In addition to text...

This Manual contains information boxes, graphics, check lists and case studies to try to make the guidance as clear and useful as possible. The following tools reappear through the text:

Key messages for wetland managers and policy makers boxes at the end of each chapter or introductory section provide wetland managers with a summary of each section's most important information, for example:

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS
▪ **'Disease'** is used to define **any impairment to health resulting in dysfunction**. There are many disease types, including: infectious, toxic, nutritional, traumatic, immunological, developmental, congenital/genetic and cancers.
▪ **Disease is often viewed as a matter of survival or death when, in fact, effects are often far more subtle**, and instead affecting productivity, development, behaviour, ability to compete for resources or evade predation, or susceptibility to other diseases factors which can consequentially influence population status.

International regulations and standards boxes highlight existing obligations under international agencies and conventions, for example:

INTERNATIONAL REGULATIONS AND STANDARDS
▪ The OIE Terrestrial Animal Health Code provides standards to assure the sanitary safety of international trade in terrestrial animals and their products. www.oie.int/en/international-standard-setting/terrestrial-code/
▪ The OIE Aquatic Animal Health Code provides standards to assure the sanitary safety of international trade in aquatic animals (amphibians, crustaceans, fish and molluscs) and their products. www.oie.int/en/international-standard-setting/aquatic-code/

Checklists to aid in disease management planning and practice, for example:

CHECKLIST	
A broad range of data should be collected at a suspected outbreak, including:	
☐ Environmental factors	☐ Number sick/dead
☐ Estimation of disease onset	☐ Clinical signs
☐ Species affected	☐ Population(s) at risk
☐ Age	☐ Population movements
☐ Sex	☐ Specific features of problem areas

Mini and full case-studies illustrate the real problems that wetland managers face, for example:

CASE STUDY

"The project area is situated in the Tongxing Village of Wucheng Township, located in the Yongxiu County of Jiangxi Province. The Wucheng Township lies at the lakeshore of Poyang Lake, covering a total area of 368 km^2, with 47 km^2 consisting of grasslands infested with snail fever…"

Definitions of main concepts or words used within the Manual [also ►**Appendix III. Glossary**], for example:

BASIC DEFINITIONS FOR THIS MANUAL

Health: a positive state of physical and mental well-being

Disease: a departure from a state of health; any impairment to health resulting in physiological dysfunction; "dis-ease" means literally a departure from a state of ease.

Boxes of specific issues of relevance providing further information, for example:

FURTHER INFO 1-1. THE IMPACT OF CLIMATE CHANGE ON ANIMAL DISEASE

"Climate change is having an unprecedented worldwide impact on the emergence and re-emergence of animal diseases, including zoonoses. The recent rise in emerging infectious diseases has included considerable increases in the number of vector borne-emerging infectious diseases during the 1990s..."

Figures, tables and other illustrations are included to illustrate key concepts.

Why this Manual was developed – the Ramsar context

The Ramsar Convention on Wetlands (Ramsar) has long recognised the multiple benefits provided by ecologically well-functioning wetlands, not just in their support of biodiversity but also in terms of services provided to human populations. Indeed, this issue was the theme of the tenth Conference of the Parties in 2008: "*Healthy Wetlands, Healthy People*". Such wetland services are especially important for impoverished communities, much of whose livelihoods or even food supplies may derive directly from wetland resources.

Should the natural ecological functioning of wetlands be impacted, the services provided can be reduced or even eliminated. The Millennium Ecosystem Assessment documents multiple ways through which this occurs and the consequences not just for livelihoods but also for human health[2,3].

Disease represents one of the many ways in which services from well-functioning wetlands may be affected. Prior to Ramsar's work on the interactions between wetlands and human health[4] and the specific case of guidance concerning highly pathogenic avian influenza H5N1 adopted by Ramsar in 2008[5], the Convention has not substantively addressed the issue of wetlands and disease before.

In 2008, CoP 10 requested Ramsar's Scientific and Technical Review Panel — in collaboration with other relevant organisations — to consider how best to develop practical guidance on the prevention and control of diseases of either domestic or wild animals in wetlands, especially those diseases that have implications for human health and further, how such guidance can be best incorporated into management plans at Ramsar sites and other wetlands. This Manual is a response to that request. It provides guidance and 'tools' for wetland managers and policy makers valuable in a range of contexts.

Disease is a 'cross-cutting' issue that has implications for a range of other wetland policy areas. Within the context of the Ramsar Convention and its national implementation, some of these other areas are indicated in ►**Table 0-2**, together with other sources of relevant Ramsar guidance.

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2 Millennium Ecosystem Assessment 2005a. *Ecosystems and human well-being: wetland and water synthesis.* World Resources Institute, Washington, DC. 68 pp. www.maweb.org/documents/document.358.aspx.pdf

3 Millennium Ecosystem Assessment 2005b. *Ecosystems and human well-being: health synthesis.* World Resources Institute, Washington, DC. 53 pp. http://www.maweb.org/documents/document.357.aspx.pdf

4 Ramsar Technical Report 6. Healthy wetlands, healthy people http://www.ramsar.org/pdf/lib/rtr6-health.pdf

5 Resolution X.21. Guidance on responding to the continued spread of highly pathogenic avian influenza H5N1. www.ramsar.org/pdf/res/key_res_x_21_e.pdf

Table 0-2. A summary of Ramsar contexts related to diseases in wetlands.

Issue	Disease implications	Source of further Ramsar or other relevant guidance
International co-operation	Potential disease spread across international boundaries Approaches for transboundary wetlands	Ramsar Handbook 17. International cooperation National co-operation through OIE (World Organisation for Animal Health)
Wetland management	Multiple means through which varying management regimes can influence risk of disease	Ramsar Handbook 16. Managing Wetlands Wetland Management Planning. A guide for site managers.
Poverty alleviation	Disease can have major impacts on livelihoods derived from wetland resources including incomes	Resolution IX.14. Wetlands and poverty reduction Resolution X.28. Wetlands and poverty eradication
Biodiversity conservation	Disease can influence the status of individual species important as reasons for the qualification of wetlands as Ramsar sites	Ramsar Handbook 17. Designating Ramsar Sites
Change in ecological character	In some circumstances, disease can influence the nature of ecological communities and hence the ecological character of wetlands	Ramsar Handbook 15. Addressing change in ecological character
Wetlands and human health	A substantive review of relationship between well functioning wetlands and human health	Ramsar Technical Report 6. Healthy wetlands, healthy people
Avian influenza and wetlands	Preparing for and managing avian influenza	Handbook 4. Avian influenza and wetlands

How this Manual was developed

User needs survey

To assess current needs, an international questionnaire survey of needs of wetland managers and other professionals in was conducted in 2010. Responses were received from 55 professionals from 17 countries (Argentina, China, India, Indonesia, Italy, Japan, Lebanon, Malaysia, Netherlands, Paraguay, Philippines, South Africa, Switzerland, Turkey, United Kingdom, United Arab Emirates and United States). These responses, such as that illustrated in ►**Figure 0-2** from the group of respondents referring to themselves as 'wardens', helped to direct the structure and content of the Manual.

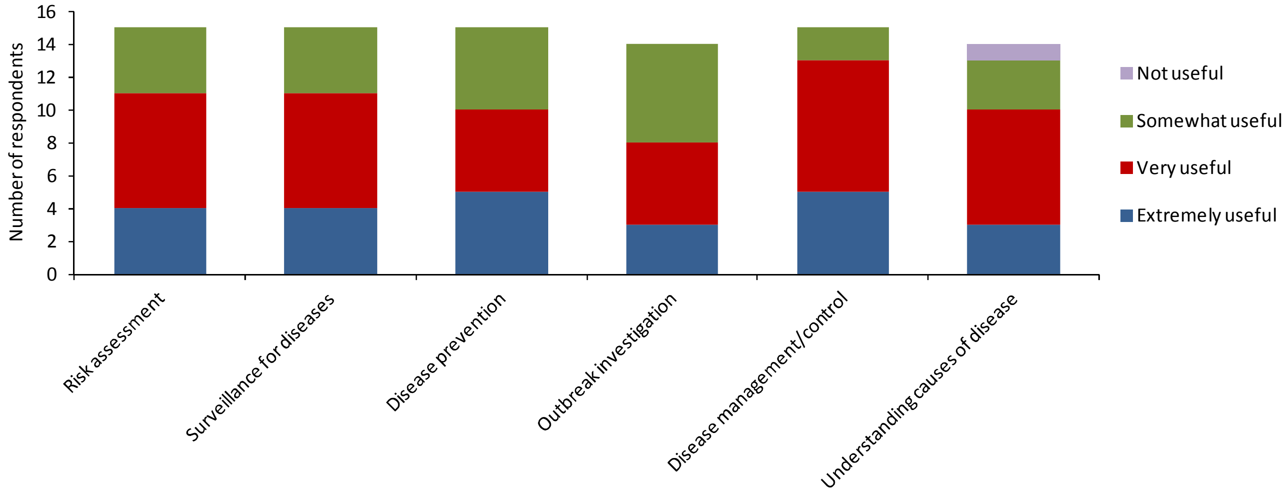

Figure 0-2. Responses from 'wetland wardens' to the question, "What are your current priority needs for information with respect to the prevention or control of wildlife disease in protected areas?"

Workshops and meetings

Two expert workshops were held to develop the Manual, one at WWT Slimbridge (2010) and one hosted by Food and Agriculture Organisation (FAO) of the United Nations in Rome, in the same year, during which the wetland disease prioritisation task was conducted.

A number of smaller planning meetings were also held.

Calls for case studies for the Manual were put out electronically and also at two international conferences: European Wildlife Disease Association 'Healthy Wildlife, Healthy People' conference held in Vlieland, Netherlands, 2010; and the `OIE Global Conference on Wildlife - Animal Health and Biodiversity – Preparing for the future' held in Paris, 2011.

Valuable input from Ramsar's STRP was provided at various stages of planning and drafting.

Sources of information

The information in this Manual was developed from the knowledge and experience of over 30 authors, contributing authors and technical editors. Additional information was gathered from:

- a review of existing sets of guidelines for managing animal disease;
- a review of guidelines for managing wetland sites;
- scientific articles in peer-reviewed journals;
- other published and unpublished documents;
- materials used in training courses; and
- the outputs and information gathered from two expert workshops.

The following websites were the main providers of information not sourced from peer-reviewed journals:

- World Organisation for Animal Health (www.oie.int/eng/en);
- UN Food and Agriculture Organisation (www.fao.org);
- World Health Organization (www.who.int);
- US Centers for Disease Control (www.cdc.gov);
- National Wildlife Health Center (http://www.nwhc.usgs.gov);
- Australian Government (www.health.gov.au/pubhlth);
- New Zealand Government (www.moh.govt.nz/moh.nsf);
- UK Government (www.defra.gov.uk);
- Wildlife Disease Information Network (wildlifedisease.nbii.gov)*;* and
- Wildlife Information Network WILDPro (www.wildlifeinformation.org).

The disease factsheets [►**Chapter 4. Animal Diseases Currently Causing Concern in Wetlands**] were largely compiled from literature produced by the veterinary, wildlife management, agriculture and public health sectors together with the technical expertise of the contributors. The information has been re-packaged (with acknowledgment) into factsheets specifically for wetland managers and supplemented, where appropriate, with information from scientific articles on wetland management and wetland management guidelines as published by the Ramsar Convention.

Chapter 1
Introduction

In this chapter you will find:

A general introduction to animal diseases in wetlands – what they are and why they are a growing problem.

A summary of the impacts of wetland disease on biodiversity and the environment, livestock and human health and its economic implications.

The scope, aim and objectives of this Manual.

Chapter contents

1.1 The state of wetlands

Wetlands are the most diverse and among the most productive ecosystems on earth. They support a high diversity and abundance of plants, birds, mammals, reptiles, amphibians, fish and invertebrates, as well as the millions of people who rely directly on wetlands for their health, livelihoods, welfare and safety. In addition, wetlands provide tremendous economic benefits, for example:

- Water supply (quantity and quality);
- Fisheries (over two thirds of the world's fish harvest is linked to the health of coastal and inland wetland areas);
- Agriculture, through the maintenance of water tables and nutrient retention in floodplains;
- Timber production;
- Energy resources, such as peat and plant matter;
- Wildlife resources;
- Transport; and
- Recreation and tourism opportunities.

Yet, in spite of their obvious importance, wetlands continue to be among the world's most threatened ecosystems, owing mainly to ongoing drainage, conversion, pollution and over-exploitation of their resources.

Many of these impacts have obvious and immediate effects, such as drainage and conversion. However, some effects, such as those from chemical pollutants, waste or excess nutrients, are more insidious, and their impacts may be more difficult to understand and quantify. One aspect which is increasingly being recognised by wetland scientists and managers as an important threat is disease. Diseases affecting wetlands have increased in both frequency and severity within the last few decades and have had major impacts on human health, livelihoods, domestic animal health and biodiversity. Yet, considering the underlying causes of disease emergence it is surprising that disease prevention is often under-recognised in management plans and actions.

1.2 What is disease?

Disease is a natural component of population ecology and ecosystems and is one mechanism by which population numbers are regulated. However, anthropogenic activities can often create novel disease problems or increases in prevalence and frequency of existing disease tipping a 'balanced' system into one where losses are increased.

DEFINITIONS 1-1. Health and disease	
Health:	a positive state of physical and mental well-being.
Disease:	a departure from a state of health; any impairment to health resulting in physiological dysfunction; "dis-ease" means literally a departure from a state of ease.

An important concept in understanding the impact of diseases on a host is that whilst diseases may cause death and/or morbidity, they may also affect the host in other ways, such as, reducing reproductive productivity and increasing susceptibility to other diseases or predation. Overall, it is important to understand that the effects of disease are often much more subtle than life or death but nonetheless can have wide ranging consequences for populations.

Figure 1-1. Canada goose *Branta canadensis* family in Greenland. Disease affects mortality and productivity, the latter in a variety of ways - a disease may delay growth so shortening the overall reproductive lifespan; the diseased host may be less able to find a mate and produce fewer young; the young may be less fit; and the diseased parent may be less fit at provisioning young and parenting effectively (*WWT*).

Types of disease

Diseases of wetlands include a wide-variety of disease types including:

Infectious: Disease due to the presence of an infectious agent that is capable of being transmitted to another host, *e.g.* avian influenza and brucellosis. This includes 'zoonotic' disease [►**Definitions 1-2. Zoonosis and related terms**].

Toxic: Disease caused by a toxin or poison, *e.g.* avian botulism and lead poisoning.

Nutritional: Disease caused by nutritional imbalance or deficiency, *e.g.* starvation and metabolic bone disease.

Traumatic: Disease caused by physical injury, *e.g.* following a hard structure collision, and electrocution.

Immunological: Disease caused by disruption or abnormal function of the immune system, *e.g.* allergy.

Developmental: Disease that interrupts normal development in growing animals. A developmental disease may affect a specific part of the body or affect multiple systems.

Congenital/ genetic: Disease that is inherited genetically or caused by loss in heterozygosity, *e.g.* infertility due to the consequences of in-breeding.

Neoplastic: Disease caused by abnormal new growth of tissue, a tumour, *e.g.* cancer.

Much of the focus of this Manual is biotic diseases: those that are caused by a living agent, such as a bacterium, fungus, virus, or protist. This category includes both infectious diseases (those that can be transmitted between host organisms) and some non-infectious diseases (those that cannot be transmitted between host organisms). An example of an infectious biotic disease is brucellosis: caused by bacteria of the genus *Brucella* and spread between animals by direct contact with contaminated body fluids. An example of a non-infectious biotic disease is avian botulism: toxins released by the bacterium *Clostridium botulinum* cause a non-infectious disease in organisms that consume it.

Other forms of disease that impact wetlands may be considered abiotic diseases: those that are caused by non-living, environmental agents, such as, toxic chemicals, heavy metals, extreme temperatures, UV radiation and nutrient imbalance. Abiotic diseases are non-infectious. An example of an abiotic disease is lead poisoning: caused by exposure to the heavy metal lead [►Table 1-1].

DEFINITIONS 1-2. Zoonosis and related terms

Zoonosis: a disease primarily of vertebrate animals that can be naturally transmitted to humans (in some instances, by a vector) and *vice versa*.

Zoonoses: plural.

Zoonotic: adjective.

A non-zoonotic disease cannot be transmitted naturally between animals and humans.

Table 1-1. Examples of how diseases can be categorised according to their ability to be transmitted between organisms, causal agents and ability to infect humans.

	Biotic *Caused by a living agent, such as a bacterium, fungus, virus or protist*	**Abiotic** *Caused by an environmental agent, such as chemicals or UV radiation*
Infectious *Capable of being transmitted between host organisms*	**Zoonotic** *Can be transmitted to humans* ▪ Brucellosis ▪ Avian influenza **Non-zoonotic** *Cannot be transmitted to humans* ▪ Chytridiomycosis ▪ Duck virus enteritis	
Non-infectious *Not capable of being transmitted between host organisms and including toxic disease, nutritional disease, trauma, genetic diseases, developmental disease etc.*	▪ Avian botulism ▪ Harmful algal blooms ▪ Aflatoxicosis	▪ Lead poisoning ▪ Trauma following hard structure collision

1.3 Wetlands and the threat of disease

Well functioning wetlands with well managed livestock with little interface with well managed wildlife should provide human wetland dwellers with the ideal healthy environment in which to thrive. Yet wetlands are at particular risk of emerging and re-emerging diseases due to a number of specific attributes:

- Their association with high population densities of people, agriculture including aquaculture, and industry;
- Pollution from the above;
- Sites providing interfaces between livestock, wildlife and people;
- Having been subject to substantial habitat modification;
- Sites rarely being isolated, instead usually being connected within catchments;
- Trade;
- The high diversity of host taxa;
- The high proportions of invasive alien species with their associated parasites; and
- The specific impacts of climate change on wetlands, their hosts, vectors and pathogens.

In effect, wetlands are 'meeting places' where humans, domestic animals and wildlife are increasingly coming into contact, creating interfaces, which together with other threats are resulting in disease emergence or re-emergence affecting public health, livestock productivity, ecosystem health, biodiversity and economies at multiple scales.

Figure 1-2. Wetlands are at specific risk of emerging and re-emerging diseases due to a range of factors including their association with people (as shown in this housing development), livestock and industry (*WWT*).

The dynamics of diseases in wetland ecosystems are changing rapidly; the most important driver is unequivocally the dramatic growth of the human population and the rapid ecological change driven, directly or indirectly, by human activity. In numerous areas of the world, infectious diseases of domestic animals that were previously endemically stable (vector, host and environment co-existing with the virtual absence of clinical disease) are now unstable due to anthropogenic changes (*e.g.* as seen with the diseases theileriosis and heartwater).

Table 1-2 provides some of the diseases that result from the specific attributes of wetlands and specific forms of rapid social and ecological change.

Table 1-2. Selected factors driving disease emergence in wetland systems (adapted from Morse, 2004).

Factor	Examples of specific factor	Examples of diseases in wetlands
Agriculture	▪ Production systems ▪ Dams ▪ Water management changes ▪ Habitat loss/degradation ▪ Pollution	▪ Highly pathogenic avian influenza, *e.g.* H5N1 ▪ Schistosomaiasis ▪ Avian botulism ▪ Harmful algal blooms ▪ Lesser flamingo *Phoeniconaias minor* toxicoses ▪ Salmon and trout sea lice
Globalisation	▪ Food production changes ▪ International trade ▪ Alien species	▪ Highly pathogenic avian influenza ▪ Amphibian chytridiomycosis ▪ Crayfish plague
Human demographics and/or behaviour	▪ Poor sanitation ▪ Wildlife interface ▪ Encroaching wildlife areas ▪ Civil conflict ▪ Non-sustainable harvesting ▪ Hunting	▪ Cholera and other intestinal parasites (micro and macro) ▪ Ross River fever ▪ Acanthocephalan outbreaks in eider ducks *Somateria mollissima* ▪ Lead poisoning
Technology and industrial changes	▪ Food production changes ▪ Breakdown in medical services	▪ Antibiotic-resistant pathogens ▪ Cholera and typhoid
Climate change	▪ Changes in rainfall and temperature	▪ Avian botulism ▪ Bluetongue disease ▪ Yellow fever

These ecological, environmental and demographic factors affect disease dynamics in three main ways:

- **Increased disease transmission risk**
  A growing human population has increased interspecies contact *i.e.* interface between wildlife, humans and domestic animals, consequently increasing the risk of disease transmission between these sectors.

Figure 1-3. Livestock market in Mali, sourced from OIE Media centre (©N.Denormandie/OIE).

- **Increased movement of pathogens**
  Globalisation (of travel and commerce) and climatic change are increasing the movement of pathogens, food, livestock, wildlife and humans worldwide. Animal translocations (of livestock and wildlife) have increased substantially in recent decades and have often resulted in serious disease outbreaks. Such movements drive disease emergence directly through the following mechanisms:

  - Infection is spread to a new area by the movement of infected animals or fomites;
  - Disease vectors are spread to a new area *e.g.* due to climate or *via* human transportation; and/or
  - Host animals are moved to a new area (exposure of immunologically naïve animals to new pathogens).

  The introduction of invasive alien species can also directly spread pathogens and indirectly drive disease emergence (*via* increased competition for resources with an invasive species increasing stress and energy expenditure rendering an animal more susceptible to disease).
  ►Section 2.5. Control of infectious diseases and invasive alien species

- **Increased susceptibility of an animal to disease**
  Stressors usually cause or result in an energetic cost and/or change in normal biological function to an animal and can increase susceptibility to disease. Populations under stress are more susceptible to disease outbreaks and length of exposure to a stressor determines how likely it is that disease will develop. Most ecological systems are exposed to multiple stressors simultaneously (or in series); subclinical stressors (*e.g.* hypoxia, pollutants) can make organisms vulnerable to other secondary stressors (*e.g.* malnutrition, disturbance) and disease progression.

  Activities that can increase stress and thus increase disease susceptibility, include the transportation and/or translocation of animals, isolation, restraint and overstocking (factors particularly relevant to the spread of disease of livestock). Other stressors are as diverse as hunting, increased genetic homogeneity and long-term toxin exposure.

  Rapid environmental changes caused by human activity have amplified the role of disease as regulation factors in species survival. These environmental stressors include the destruction or conversion of wildlife habitat by humans, resulting in habitat loss, degradation, fragmentation and macro- and microclimate changes.

Fragmentation of habitat by human encroachment can result in vulnerable isolated wildlife populations in human-made 'island ecosystems' which are at increased risk of diseases and their impacts.

Environmental pollution can also be a direct cause of non-infectious disease (*e.g.* lead poisoning or oestrogenic agents disrupting hormone function) and may also drive disease emergence. Air, water, light, noise and thermal pollution must also be considered as stressors or drivers of disease in wetland systems.

Nutritional stress (lack of, poor, or imbalanced nutrition) can lead to immunological impairment and often tip the balance between health and disease in animals (e.*g.* nutritional stress has been linked with increased prevalence of Hendra virus in fruit bats in Australasia.

These stressors and drivers of disease ***should not be considered in isolation*** *as several factors often contribute (synergistically) to the emergence of a wetland disease.*

FURTHER INFO 1-1. The impact of climate change on animal disease

Climate change is having an unprecedented worldwide impact on the emergence and re-emergence of animal diseases, including zoonoses. The recent rise in emerging infectious diseases has included considerable increases in the number of vector borne-emerging infectious diseases during the 1990s. Climate change is thought to play a significant role in this with compelling evidence of variations in climate impacting diseases such as malaria, dengue fever and plague in humans, bluetongue in livestock and other diseases of amphibians and corals.

As the climate continues to change, the effect of pathogens on wildlife, livestock and humans is also likely to change. Although there is a consensus among scientists that climate change will result in general increases in disease incidence and distribution, it is worth noting that due to the complexities of climate change-disease interactions some diseases are likely to decrease in frequency or prevalence.

How might climate change bring about animal disease expansion or change?

- **Rising temperatures** may alter the population size and/or distribution of pathogens, vectors and hosts.
  Pathogens: pathogen growth can be temperature dependent.
  - Cholera is caused by *Vibrio cholerae*, a water-borne bacterial pathogen, whose prevalence is expected to rise with global temperatures, moderate increases in temperature and rainfall may precipitate outbreaks.
  - Anomalous high ocean temperatures have been linked to coral disease outbreaks.
  - Conversely, pathogens that prefer cooler temperatures, *e.g.* fungal entomopathogens of insects, may decline.

  Vectors: temperature increases may reduce restrictions on insect distribution. Mosquitoes can now be found at Everest base camp, traditionally a place where low temperatures and high altitude have deterred the insect; annual temperature increases of 0.9° C have caused this shift in distribution. Temperature changes may also affect vectors by altering biting rates or length of the transmission period.

  Hosts: host distributions have already altered due to climate change. In the Arctic, southern species, such as white-tailed deer *Odocoileus virginianus*, are invading areas normally occupied by caribou *Rangifer tarandus*. The deer can carry ticks and therefore have the potential to distribute tick-borne parasites such as those responsible for Lyme disease.
- **Increased precipitation** – heavy rainfall, especially following drought, can cause insect population

booms by increasing larval habitats; flooding events may increase water-borne diseases such as cholera or leptospirosis; storms can increase transport of waste water diseases to groundwater.

- **Variations in rainfall/dry season patterns** – the Ebola virus is linked to unusual rainfall patterns, as climate change disrupts seasonal rainfall, increased episodes may be expected. Rodent populations are known to increase following mild/wet winters in temperate regions, rodent-borne diseases include: Lyme disease, tick-borne encephalitis and hantavirus pulmonary syndrome.
- **Increased drought and heat** – drought can cause livestock and wildlife to congregate around limited water resources increasing risk of pathogen transmission (*e.g.* bovine tuberculosis may proliferate in such a manner). Grazers would also suffer with restricted food availability due to limited vegetation growth. Such stresses would predispose animals to greater parasite load and greater risk of diseases progressing from a sub-clinical to a clinical state [► example below].
- **Forcing animals to adjust movements or migration** – climate dependent resources (*e.g.* vegetation cover) may be altered which in turn may affect movement patterns increasing the potential for introductions to, or encounters with, novel pathogens.
- **Reducing the number of long-distance migrations** – changes to habitats and weather conditions may encourage animals to remain at one site instead of undertaking traditional migrations. In China rising temperatures causing increased glacial runoff into nearby wetlands has been cited as one reason why unusually large numbers of geese are remaining at Qinghai Lake over winter instead of migrating to India. With greater concentrations of birds comes greater concern about increased transmission of avian viruses such as highly pathogenic avian influenza H5N1.
- **Human actions** – climate change may result in shifts in distribution of fertile farmland to areas that encroach upon wildlife, increasing risk of transfer of infection between livestock, people and wildlife. Local land use changes are also expected to exert temperature and rainfall changes (*e.g.* reduced vegetation could reduce evapotranspiration and consequently, rainfall). Climate models predict that such changes will alter the distribution of malaria in Africa - in tropical Africa and in parts of the Sahel the spread of malaria will decrease and the risk of malaria epidemics will shift southwards.

Example: African lions, drought and disease

An example of how increasing extreme weather may cause the expansion of animal diseases occurred in 1994 and 2001 in Tanzania. During these years there was unusually high mortality of lions *Pathera leo* due to canine distemper, an endemic disease that is not usually fatal. *Post mortem* analyses had also revealed higher than usual levels of the tick-borne parasite *Babesia leo* and it was this co-infection that had reduced the lions' immunity and caused them to succumb to canine distemper. A link was drawn between the environmental conditions and the deaths: in 1994 and 2001 there had been extended droughts that had weakened the local herbivore population and allowed the ticks that parasitised the herbivores to prosper; the lions feeding on the weakened herbivores were then exposed to greater infection by *Babesia* causing susceptibility to canine distemper. With climate change expected to increase the number of drought events in Africa, lion populations are likely to continue to suffer large losses to an already threatened population.

1.4 Effects of disease on biodiversity

It is well recognised that diseases play an integral part of ecosystems and specifically an important role in population regulation through effects on both birth and death rates. Diseases can shape population age structures and geographical range, *e.g.* distribution of cervids in eastern and north eastern America is determined by a meningeal worm. Yet the emergence of numerous and novel diseases related to human activities can negatively impact biodiversity and contribute to species declines and even extinctions. The previously discussed drivers of disease affecting the wider environment, host populations, parasites and their vectors, together with factors specific to wildlife, such as, intensive conservation management of wildlife, effects of providing supplemental food including feeding stations, and translocations have all contributed to the negative consequences of disease at a population level.

Diseases can alter wildlife communities in the short and long term. For example, the introduction of myxomatosis to control rabbits contributed to the extinction of large blue butterflies *Maculinea arion* in the UK (through knock-on effects on vegetation, vertebrate and invertebrate populations and community structure). The introduction of rinderpest virus to Africa altered abundance and distribution of herbivore populations dramatically throughout the continent.

Figure 1-4. Diseases can alter wildlife communities in the short and long term, affecting age structure and geographical distribution of species such as these African wildebeest *Connochaetes taurinus* (*WWT*).

Communities can be impacted additionally when species, such as 'keystone species', are negatively affected by disease. Perhaps this is best illustrated by effects of diseases on corals, with dramatic changes throughout communities and ecosystems.

The effects of disease on isolated or threatened species can be particularly severe. Small populations lose heterozygosity and are thus inherently more genetically susceptible to disease (and immunologically naïve isolated populations, such as island species, tend to have relatively limited genetic diversity). Additionally, individuals and populations under stress (*e.g.* caused by habitat degradation) can lose immunocompetence. The overall effect can be to create populations at greater risk of disease where the impacts can be particularly serious, causing either extinction or further loss of heterozygosity, further disease susceptibility and possibly jeopardising the survival of the population.

Figure 1-5. Whooping crane *Grus americana,* a threatened species which has suffered from diseases whilst sympatric more abundant sandhill cranes *Grus canadensis* have been relatively unaffected (*Ramsar*).

To illustrate that disease has become a cross cutting conservation issue, we have used as a proxy, an analysis of multilateral environmental agreement instruments, specifically under the Convention on Migratory Species, of the number of instruments mentioning the terms 'health' or 'disease'. As Figure 1-6 illustrates this has increased significantly over the last two decades. The issue of disease will no doubt continue to be highlighted on conservation agendas.

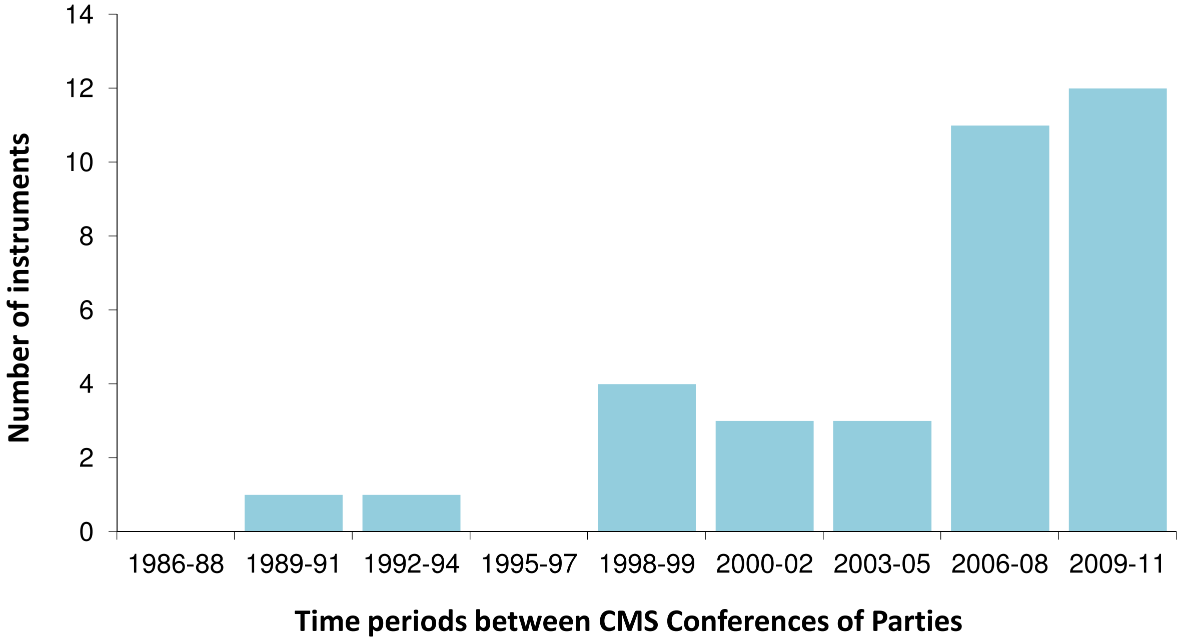

Figure 1-6. The increasing frequency of references to animal disease (or health) in formal documents of CMS (including COP Resolutions), its associated Agreements and other linked treaties for migratory species, from 1985 to 2012 (Lee, unpublished). Data are total numbers of formal documents containing the words "disease" and/or "health" by triennial periods.

As a final point to consider in this section on the effects of *disease* on *biodiversity*, it is probably worth reflecting on the effects of *biodiversity* on *disease*. Biodiversity in itself helps to provide resilience to ecosystems, buffering against disease emergence. This needs to be borne in mind when considering management of wetlands and any disease control activities negatively impacting biodiversity may have longer term poorer health outcomes.

Figure 1-7: Biodiversity in itself helps to provide resilience to ecosystems, buffering against disease emergence (*WWT*).

1.5 Effects of disease on livestock

Despite access to veterinary care and management, disease often has a far greater impact on livestock populations than wild populations, particularly in intensive systems, due to the nature of managing high densities of often genetically similar individuals in circumstances where hygiene standards may be suboptimal. Diseases in livestock create welfare issues and loss of productivity either by the fatal action of the disease itself or through an enforced cull of affected stock (*e.g.* as happens in control of highly pathogenic avian influenza). Table 1-3 summarises the impact of a number of wetland diseases on livestock.

Even when animals do not die, general unthriftiness can readily affect income, food security and human health. Such effects include reductions in reproduction and productivity (*e.g.* brucellosis or schistosomiasis), increased susceptibility to other diseases or predation, or a reduction in an animal's ability to respond to other environmental stresses (*e.g.* as is the case in African animal trypanosomiasis). Environmental stressors might be the catalyst for a disease to progress from a sub-clinical to a clinical state (*e.g.* salmonellosis). Some infections may cause infertility or spontaneous abortions (*e.g.* brucellosis and leptospirosis) whereas others may affect production by specifically affecting the young, pregnant or lactating females (*e.g.* salmonellosis or leptospirosis).

These factors together with potential impacts on trade of livestock can result in significant impacts on livelihoods. The importance of healthy livestock is illustrated in the key support of northern

African farmers in their helping to eradicate rinderpest from their cattle. They actively engaged in vaccination and other disease control measures for their cattle recognising that childhood disease vaccination programmes were of negligible value if their cattle died, as without protein security the survival of the children was in doubt.
►**Case Study 2-1. Rinderpest – eradication of a disease affecting all sectors.**

Perhaps the most important issue affecting wildlife and livestock health is the ever increasing interface between these sectors. Feeding a burgeoning human population pushes our livestock production into wild places and wildlife moves into human habitation to exploit new habitats and resources. Most (77%) infectious diseases of domestic animals are common to wildlife, so the control of a disease in domestic animals can be impeded by its presence in wildlife. Whilst culling or other disease control measures in infected livestock can reduce levels of disease, if the disease persists in a wildlife reservoir it can spillback to domestic animals at a later point. Local and global movement of domestic animals for trade and farming can help to spread disease and also introduce novel parasites to naïve livestock populations.

Production systems are also generators of new diseases, driving the emergence of novel pathogens with potential for affecting livestock, wildlife and human health. Highly pathogenic avian influenza H5N1 illustrates this very well. The use of scrapie-affected sheep as a protein source for cattle fodder provided a route for the emergence of bovine spongiform encephalopathy in the UK in the 1980s. Given the need to feed humans into the future it seems certain that livestock production systems will ensure that there are many challenges ahead for pathologists, other diagnosticians, animal and human health services and society as a whole.

Figure 1-8. Domestic ducks feeding in That Luang marsh in Laos. Wetlands provide an interface for domestic animals and wildlife as well as people allowing transmission of pathogens between these sectors (*Sally MacKenzie*).

Table 1-3. Selected wetland diseases affecting livestock.

Disease	Hosts*	Impact on livestock
Schistosomiasis	Cattle, sheep, goats. Wild mammals and wildfowl are also affected.	An estimated 165 million animals are infected in Africa and Asia, most infections are sub-clinical but the disease can still cause serious morbidity and mortality. Farmers suffer significant reductions in productivity due to disease burden. Susceptibilty to other environmental stressors is also increased.
Peste des petits ruminants (PPR)	Small ruminants - predominantly sheep and goats	PPR causes heavy losses to goat and sheep stock and thus has knock-on effects for livelihoods and food security.
Leptospirosis	Cattle, sheep, goats, pigs.	Mortality can be high in calves and young or weak piglets. Adult mortality is low and many animals exhibit mild to no clinical signs. Some infections may cause infertility and spontaneous abortion in cattle.
Brucellosis	Cattle, swine, goats, sheep, other ruminants.	High mortality of unborn animals, the disease can be debilitating and causes loss of productivity and welfare problems. Trade restrictions increase economic losses.
Duck virus enteritis	Ducks and geese	In susceptible domestic waterfowl flocks, high percentage mortality and reduced egg production can occur.
Epizootic ulcerative syndrome	Wide range of wild and farmed fish	High losses to fish farmers through mortalities, reduced productivity and market rejection due to presence of lesions affecting consumer confidence.
Lead poisoning	Mammals, poultry including waterfowl.	Lead is a common cause of morbidity and mortality in livestock, particularly for sheep and cattle. Large scale stock losses can occur if they are not removed from the source of exposure promptly.
Salmonellosis	Most commonly in poultry and pigs	Many infected animals will not show clinical disease. In mammals, clinical disease is most common in very young, pregnant or lactating animals, and often occurs after a stressful event. Outbreaks in young ruminants, pigs and poultry can result in a high morbidity rate.
African animal trypanosomiasis	Cattle, swine, camels, goats and sheep.	Trypanosomiasis threatens 50 million cattle in Africa and can reduce livestock holdings by 10-50%. The disease has a high morbidity rate and is often chronic in susceptible animals. The mortality rate can reach 50-100% within months of exposure, particularly if the animal is exposed to poor nutrition and other stresses.
Bovine tuberculosis	Cattle plus a wide range of wild and domestic mammals.	Significant importance to the cattle industry through loss of production, control measures and trade restrictions. Presence of the disease may also lead to loss of consumer confidence in milk and beef products.

** Not all hosts are listed.*

1.6 Effects of animal disease on humans

There are over 200 described zoonotic diseases and their effects, which may depend on both the pathogen and the host, are varied ranging from mild headaches to death. The majority (60%) of emerging infectious diseases in humans are caused by zoonotic pathogens. Livestock production systems provide opportunities for zoonotic disease transmission and increased human population density living with domestic livestock and pet animals is linked to a rise in the number of zoonotic infections in humans. Additionally, wildlife plays a key role by providing a 'zoonotic pool' from which new diseases may emerge. Of the 60% of emerging infectious zoonoses, 72% have a wildlife origin. Human encroachment into wildlife habitats and wildlife utilising urban settings, as well as trade and use of wildlife (*e.g.* bushmeat), increases disease transmission risks.

As well as the direct impact of animal diseases on humans, there are numerous indirect impacts mainly caused by the reduced production of livestock in terms of both food security and quality and reduced income linked to production losses and trade restrictions.

►Section 1.5. Effects of disease on livestock.
►Section 1.7. Economic impacts of animal diseases.

Figure 1-10. An increasing range of interfaces between humans and animals allows zoonotic diseases to emerge.

1.7 Economic impacts of animal diseases

The economic consequences of animal disease are numerous, varied and occur at multiple scales from local to international. There are economic losses to livestock production as disease causes direct mortality and morbidity and reduces production efficiency, *e.g.* feed conversion efficiency and/or egg/milk yields can suffer. Production efficiency is also affected if a disease forces farmers to use resources sub-optimally, *e.g.* using cattle resistant to trypanosomiasis is one option for farmers affected by the disease, however this involves a trade-off because these cattle are smaller in size and less productive (accepting that in a trypanosomiasis region it is more productive to use the trypanotolerant breeds of livestock rather than suffer losses of 'more productive' yet less resistant breeds).

Disease also causes losses of revenue from restrictions on animal movement and trade, costs of control measures (including veterinary treatments) which can be prohibitive, negative impacts on agriculture and aquaculture markets, socio-economic influence on livelihoods, public health concerns especially in the instance of zoonotic disease, and even loss of income to tourism initiatives, *e.g.* where disease reduces wildlife population numbers and therefore the likelihood of providing a tourist attraction.

For both domestic animals and wildlife, there are costs associated with disease prevention: monitoring, surveillance, preventative treatments, vaccines and vaccination programmes can all be expensive. However, the costs of disease control operations following an outbreak can be even more so: as a general principle, prevention costs provide a sound investment.

Although complicated, the economics of disease management need to be seen in the broader context of ecosystem health [►**Section 2.3. The ecosystem approach to health in wetlands**]. Viewing disease management from this perspective which includes 'all' the costs of loss of ecosystem function and benefits can help determine appropriate disease control strategies. Although disease may affect income in one sector there may be other compensations. As an example, losses and theoretical losses to livestock production in endemic African animal trypanosome areas allow areas to be left for wildlife from which other direct revenue can flow, *e.g.* through tourism initiatives.
►**Section 4.3.1. African animal trypanosomiasis.**

A cost-benefit analysis, or decision tree, for example, may be useful when comparing the relative merits of different strategies.

Table 1-4. Examples of the economic impacts of animal diseases.

Disease	Economic impact
Peste des petits ruminants (PPR)	PPR in Africa, the Middle and Near East, South West and Central Asia threatens a billion strong population of ruminants and affects economies based on losses of meat, milk, offspring and mortalities and morbidity, as well as disease control. Annual PPR losses in India alone are estimated at 1,800 million Indian rupees (US$39.4 million).
Highly pathogenic avian influenza (HPAI) H5N1	The outbreak of HPAI (HPAI) in Hong Kong in 1997 is estimated to have cost US$100s of millions and global estimates from outbreaks since 2003 run at billions of dollars.
Classical swine fever	The continued presence of classical swine fever in Haiti has been estimated to result in an annual reduction in income of US$2.7 million for the local small holder producers.
Rabies	It is estimated that the US spends a minimum of $230 million a year to control this disease.
Foot and mouth disease	The 2001 foot and mouth disease outbreak is estimated to have cost the UK £1.2 billion.

1.8 The scope, aim and objectives of this Manual

This Manual has been prepared at the request of the Conference of Parties of the Ramsar Convention (Resolution X.21) and is targeted principally at wetland managers, but also contains much information of relevance to others involved with wetland conservation either at the scale of individual wetlands or more widely.

Covering the entire range of diseases affecting wetlands is beyond the scope of this Manual. Instead, this Manual focuses on diseases primarily affecting animals[1] (with specific information for a subset of these diseases). Diseases of organisms other than animals, such as plant diseases and diseases primarily of humans, such as malaria and dengue fever, are not included. We hope, however, that additional volumes can be produced to cover these topics.

The Manual is intended for use at wetlands anywhere in the world. The practicalities and resources available may vary but the principles of disease management remain the same.

Written for wetland managers, this Manual aims to bring together what is currently known about animal diseases affecting wetlands and what options are available for managing them. Following an introduction to the issue of diseases in wetlands, we present the general principles of disease and its management in wetlands. We then provide descriptions of a selection of management practices for preventing and controlling disease outbreaks. Thereafter, factsheets present descriptions of a selection of priority diseases[2] affecting wetlands and information to assist in their management. We end with suggestions for where to obtain further information and direction.

Included in the appendices are categories of additional information which we hope will be useful, including a glossary of commonly used terms relating to disease, a list of countries which are members of the World Organisation for Animal Health (OIE) [►**Appendix IV**], a list of diseases notifiable to the OIE [►**Appendix V**], a summary of the priority disease factsheets, and a summary of the most important disease issues relevant to policy makers. Throughout the Manual key messages for wetland managers and policy makers are highlighted.

As seen in Table 1-2, the drivers for disease emergence in wetlands are closely associated with human activity and disease prevention in these habitats lies primarily with land users and managers, together with decision makers. Use of this Manual should provide managers with enhanced understanding that will help assist better informed decision making with respect to preventing and controlling disease in wetlands. This will assist with the task of maintaining the ecological character of wetlands – an essential element of Ramsar's wise use agenda. It should also materially benefit human communities dependent on wetlands by reducing disease risk either directly, or indirectly, to their livelihoods by impacts on livestock and other agricultural interests.

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[1] In this Manual 'animals' refers to non-human animals.

[2] As determined at one of expert workshops *i.e.* diseases with greatest impact on human and animal health and livelihoods in wetlands ►**Appendix VI**

Figure 1-11. The drivers for disease emergence in wetlands are closely associated with human activity and thus disease prevention in these habitats lies primarily with wetland managers and users. This Manual aims to inform these key personnel (*WWT*).

In summary…

Aim of this Manual:

Well-informed decisions by wetland managers and policy makers with regard to the prevention and control of animal diseases in wetlands so as to ensure wise use.

Objectives of this Manual:

- To explain the **principles** of disease prevention and control;
- To provide guidance on **practical measures** for disease control in wetlands;
- To provide generic information on a selection of **priority diseases**;
- To provide advice on incorporating disease control measures into **site management plans**; and
- To provide links to **further resources and information**.

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS
▪ The term '**disease**' is used to define any impairment to health resulting in dysfunction. There are **many disease types**, including: infectious, toxic, nutritional, traumatic, immunological, developmental, congenital/genetic and cancers. ▪ **Disease is often viewed as a matter of survival or death when, in fact, effects are often far more subtle**, instead affecting productivity, development, behaviour, ability to compete for resources or evade predation, or susceptibility to other diseases factors which can consequentially influence population status.

- **Well functioning wetlands** with well managed livestock, with little interface, with well managed wildlife should provide human wetland dwellers with the **ideal healthy environment in which to thrive.**
- **Disease is an integral part of ecosystems** serving an important role in population dynamics. **However, there are anthropogenic threats affecting wetlands** including climate change, substantial habitat modification, pollution, invasive alien species, pathogen pollution, wildlife and domestic animal trade, agricultural intensification and expansion, increasing industrial and human population pressures including the interface between humans and domestic and wild animals within wetlands, all of which may act as drivers for emergence or re-emergence of diseases.
- **Wetlands are meeting places** for people, livestock and wildlife and **infectious diseases can be readily transmitted** at these interfaces.
- **Stress is often an integral aspect of disease** capable of exacerbating existing disease conditions and increasing susceptibility to infection. **There are a broad range of stressors** including toxins, nutritional stress, disturbance from humans and/or predators, competition, con-current disease, weather and other environmental perturbations. Stressors can be additive, working together to alter the disease dynamics within an individual host or a population.
- **Impacts** of disease on public and livestock health, biodiversity, livelihoods and economies **can be significant**.
- **The emergence and re-emergence of diseases has become a wildlife conservation issue** both in terms of the impact of the diseases themselves and of the actions taken to control them. Some diseases may be significant sources of morbidity and mortality of wetland species and in some cases (*e.g.* amphibian chytridiomycosis) can play a role in multiple extinctions of wetland species.

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Chapter 2
Principles of Disease Management

In this chapter you will find:

An introduction to disease management in wetlands – important concepts.

Why disease management needs to appreciate the relationship between wildlife, livestock and humans, and take an ecosystem approach.

Why disease management should be integral to wetland management.

A summary of proactive and reactive strategies for managing animal diseases in wetlands.

The dual benefits of controlling emerging infectious diseases and invasive alien species.

A brief introduction to the role of communication, education, participation and awareness in disease management.

Chapter contents

2.1 Important concepts of disease management in wetlands

Disease is a natural part of ecosystems and absence of disease should be seen as neither natural nor desirable. However, with wetland habitats subject to substantial and widespread modification and with such a broad variety of anthropogenic uses, diseases have emerged or re-emerged in the last few decades at a far greater frequency than previously recorded. ►**Section 1.3 Wetlands and the threat of disease.** The dependence of all three sectors *i.e.* humans, domestic livestock and wildlife, on healthy wetlands has meant that disease management is now of significant importance in terms of public health, livelihoods, lifestyles, domestic animal production and healthy wildlife.

A million dead waterbirds in an outbreak of avian botulism is a clear indication of a major health problem. However, the wetland manager must understand that disease is usually a much more subtle process affecting body systems and functions, and creating energetic costs to the host. Morbidity or mortality may be the outcome but often there will be less obvious consequences on behaviour, reproductive success, the ability to compete for resources and evade predation, and so on. Disease, therefore, acts to shape and limit populations, affecting age structures and distribution of wild species. It is strange then, that wildlife disease has been rather sidelined as an issue by many ecologists for many years. Anthropogenic activities have now affected the environment to such an extent that wildlife disease has, in effect, 'shown itself' to the ecologists, land managers and policy makers and has now become established as a cross cutting conservation issue.
►**Section 1.4. Effects of disease on biodiversity.**

FURTHER INFORMATION 2-1. WETLAND MANAGERS AND THEIR KEY ROLE IN DISEASE MANAGEMENT

Anthropogenic activities are the drivers for 'problem' diseases. The real power for disease control and prevention is in the hands of the land managers and users. For wetland diseases, these key stakeholders are the wetland managers, local wetland users including farmers, hunters, fishers and people living in and around wetlands, and those making policies affecting wetland use. Therefore, this Manual focuses on the wetland managers and policy makers with the aim of influencing the activities and practices of all those using wetlands for their vital resources and services.

Disease management in wetlands aims to both prevent emergence of disease and, should disease become a problem, control or eradicate it. Effective disease management practiced at a landscape or catchment scale can ensure that disease does not spread and/or become endemic and cause long term problems.

The adage of 'prevention is better than cure' is fundamental to disease management. Costs of disease management must be weighed against the benefits of preventing problems, in particular long term issues negatively impacting livelihoods, public health, domestic animal production and biodiversity.

The spectrum of disease management practices is broad and may entail nothing more than routine wetland management practices through to major interventions for large scale disease control operations, depending on the issue, its scale and potential impact. Disease management practices may be focused on the environment, the hosts present in the wetland and its catchment, or, in the case of infectious disease, the parasite or pathogen, or any combination thereof.

►**Figure 2-1** illustrates some of the factors influencing disease outcomes for a host and thus provides insight into which factors can be targeted when managing disease.

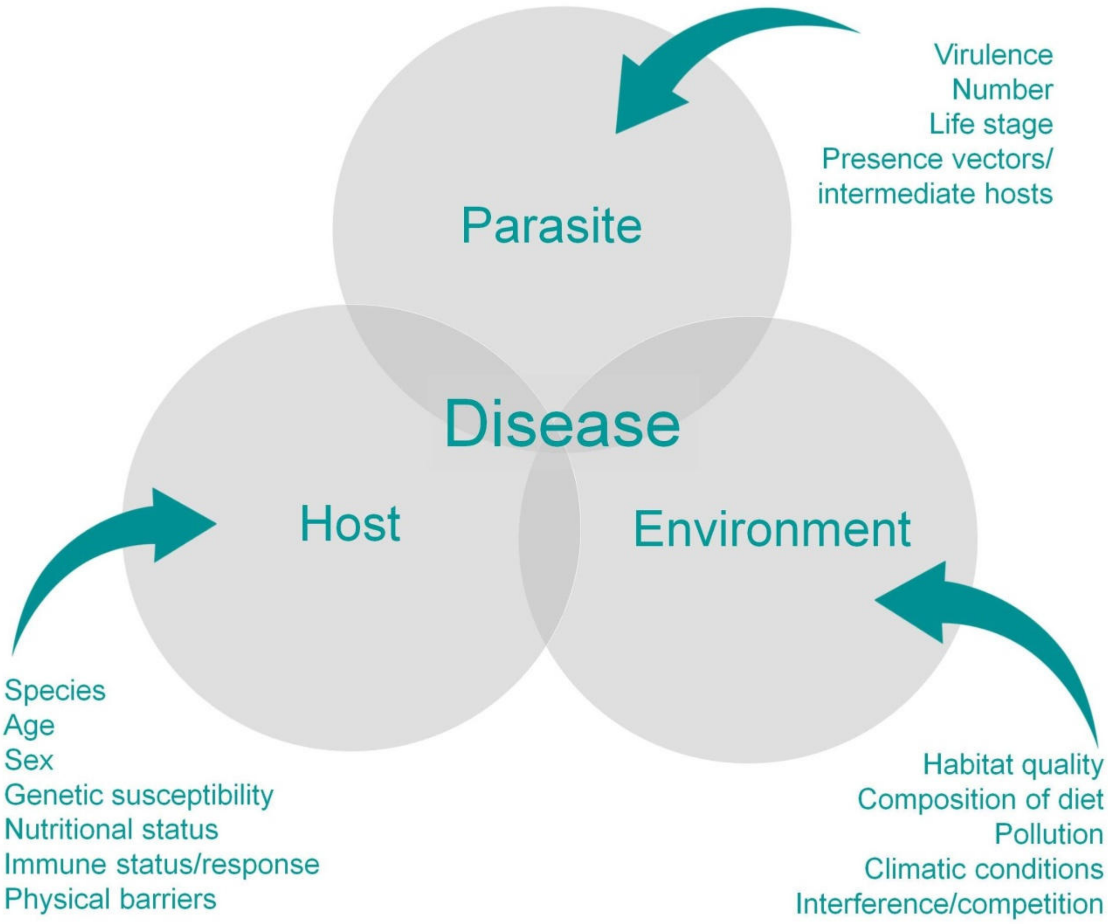

Figure 2-1. The outcome of disease is dependent on the relationship between a host and its environment, and in the case of infectious disease, the pathogen also. The figure shows some of the factors (outside the circles) which influence this relationship and thus some of the factors that can be targeted for disease control.

CASE STUDY 2-1. Rinderpest – eradication of a disease affecting all sectors

Figure 2-2. Statue of a buffalo being unveiled by Kenya's president, Mwai Kibaki, to mark eradication of rinderpest (in Roeder 2011).

Rinderpest, once described as "the most dreaded bovine plague known", became the first disease of animals to be eradicated by human intervention. This acute viral disease has been responsible for the death of domestic cattle for millennia, adversely affecting livestock, wildlife and agricultural livelihoods, bringing starvation and famine. In its classical, virulent form, rinderpest infection can result in 80-95% mortality in domestic cattle, yaks, buffalo and many other wild ungulate species. The disease has had far reaching conservation impacts affecting the abundance, distribution and community structure of many species as well as becoming a source of conflict between agricultural and wildlife interests. The disease is caused by a morbillivirus known as the rinderpest virus (RPV), which is usually spread by direct or close contact between infected and susceptible animals. Clinical signs include: fever, depression, loss of appetite, discharges from the eyes and nose, erosions throughout the digestive tract, diarrhoea and death. Weight loss and dehydration, caused by enteric lesions, can cause death within 10-12 days.

Key Actions Taken to eradicate rinderpest included the development of vaccines, disease surveillance, diagnostic tools and community-based health delivery.

- **Vaccine development:** Plowright developed a tissue culture rinderpest vaccine (TCRV) in the 1950s, a heat-stable variant of which was developed in the 1980s, which was successfully used in community-based vaccination campaigns in remote areas of Africa and Afghanistan.
- **International collaboration and coordination of eradication efforts**
    - **PARC:** The **Pan-African Rinderpest Campaign,** launched in 1986, incorporating 34 African countries, coordinated efforts to eradicate RPV from the Africa continent. Initially, mass livestock vaccination programmes were implemented followed by improved disease surveillance and focussed vaccination campaigns (containing any remaining reservoirs of disease).
    - **GREP**: FAO launched the Global Rinderpest Eradication Programme in 1994, with the aim of eradicating RPV by 2010. Initially, the GREP focussed on the extensive vaccination of susceptible livestock, later moving to disease surveillance. Research yielded sensitive tests for RPV detection, enabling rapid diagnosis and decreasing the likelihood of disease spread. The GREP coordinated rinderpest eradication efforts globally: assisting existing veterinary services through clinical disease research, disease surveillance, technical and laboratory support, awareness raising and contingency planning. The Programme worked in partnership with PARC.
- **Disease Monitoring**
    - The **Programme for the Pan African Control of Epizootics** (PACE) improved surveillance capacity in Africa.
    - The **Community-based Animal Health and Participatory Epidemiology** (CAPE) Project supported the development of veterinary service delivery and disease surveillance, particularly in remote areas. Community-based animal health workers were fundamental to disease control.
- **Accreditation of rinderpest freedom:** Finally, the FAO/OIE Joint Committee for Rinderpest Global Declaration was formed (1993) to guide and monitor accreditation of rinderpest freedom on a country-by-country basis. Disease surveillance and accreditation continued until **2011**, when on **June 28th** the world was declared free from rinderpest.

Outcomes: The benefits derived from the eradication of rinderpest are numerous and include: protected rural livelihoods, increased confidence in livestock-based agriculture, an opening of trade in livestock and their products and increased food security. Veterinary services worldwide have become more proficient as a consequence of the fight against rinderpest and the conservation of numerous African ungulates has also benefited. The socio-economic benefits of rinderpest eradication are said to surpass those of virtually every other agricultural development programme and will continue to do so. The direct economic benefits will become clearer over time but one preliminary study conservatively estimated a benefit of at least US$16 from each dollar spent on rinderpest eradication. Rinderpest was successfully eradicated due to ongoing, concerted, international efforts that built on existing disease control programmes in affected countries. Only through international coordination can other such transboundary diseases be controlled and eliminated, as isolated national efforts often prove unsustainable.

The point at which substantial interventions may be required will be related to the extent to which the problem affects or threatens livelihoods and public, domestic animal and wildlife health and welfare. It is important to note that different stakeholders will likely have different ideas about when interventions are required and ideally these can be addressed within management and contingency plans in 'peacetime' *i.e*. before a disease problem, to ensure engagement and 'buy in' of stakeholders and thus rapid responses in times of emergency.

It is important to understand that disease management may be thwarted by poor understanding of disease ecology and dynamics, and thus the appropriate management practices to mitigate. Inappropriate disease management practices can even result in counter-productive consequences and novel disease problems. Hence, a good evidence base is important, appreciating that this may be difficult to attain due to complexities or limitations of diagnosis, surveillance, and other knowledge gaps.

2.2 The disease relationship between wildlife, livestock and humans

The globalised planet currently supports some seven billion people and myriad associated livestock living across the planet in a broad range of modified habitats. As human development and livestock have encroached into wild habitats, not surprisingly infectious diseases have spread between these populations, negatively affecting all three sectors. Movements of people and extensive trade in wild and domestic animals have resulted in the global spread of a number of pathogens, causing particular problems where infectious agents are novel and new hosts are immunologically naïve.

The complexities of disease dynamics in wildlife have resulted in unpredicted disease emergence. Diseases of wildlife that affect humans or their livestock have sometimes led to eradication programmes targeted at wildlife which have not necessarily resulted in reduced disease prevalence but, instead, serious long term consequences for biodiversity, public health and well-being, and food security, whilst failing to address causal problems.

It has become common understanding that the world can no longer deal with diseases of people, domestic livestock and wildlife in isolation and, instead, an integrated 'One World One Health' approach to health has developed. Delivering integrated approaches and responses across the medical, veterinary, agricultural and wildlife sectors can be problematic given existing organisational roles and structures but demonstrating the benefits this can bring should help promote this progressive way of working. The recent global eradication of rinderpest provides an example of how one disease with impacts across all sectors requires global coordinated efforts to bring about success and benefits for all.
► **Case study 2-1. Rinderpest – eradiation of a disease affecting all sectors**.

For wetlands, which provide the 'meeting place' for people, livestock and wildlife, a mapping of a number of important wetland diseases, according to their hosts (Figure 2-3), illustrates clearly that more diseases are shared between these sectors than are specific to any one sector. Tackling disease in one sector is unlikely to be successful in the long term without consideration of the others. Moreover, not working at an ecosystem scale, and without integrated approaches, misses opportunities for broader positive health outcomes.

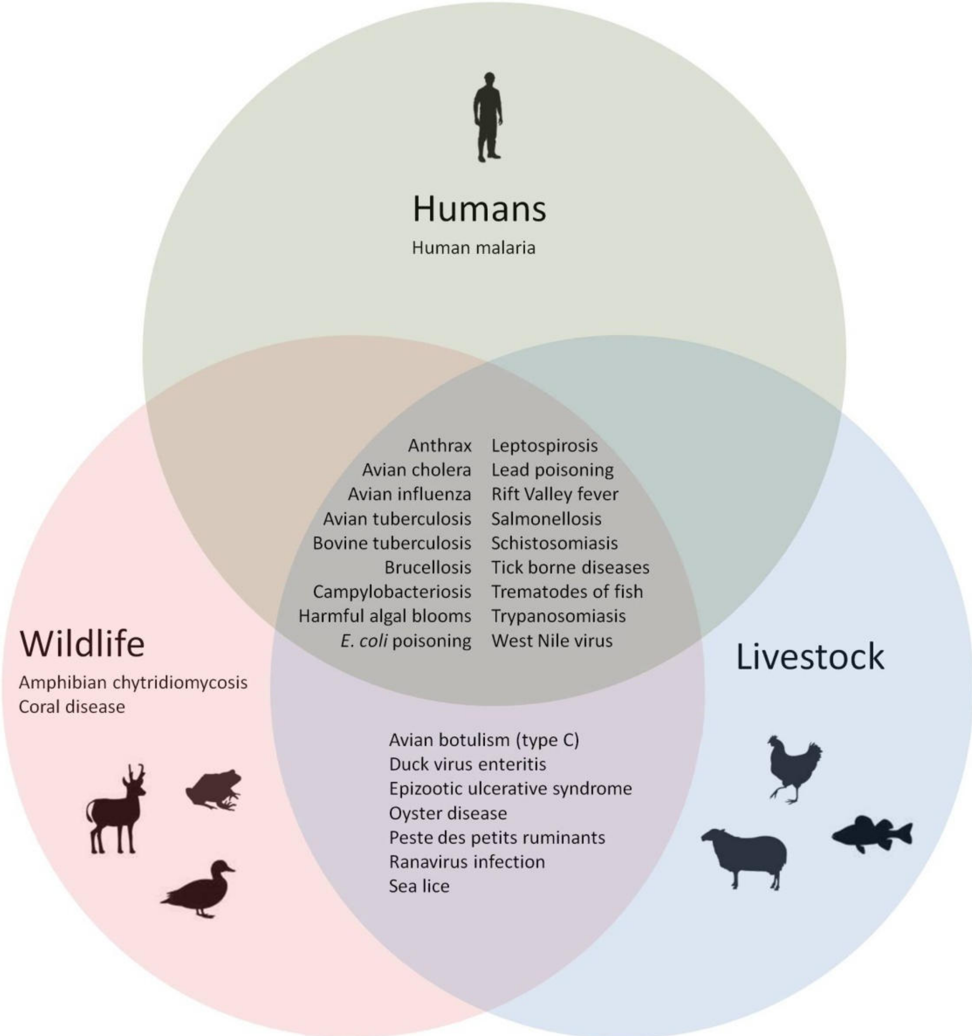

Figure 2-3. A number of important wetland diseases mapped according to the hosts they affect: the majority of both infectious and non-infectious diseases are common to all three sectors.

2.3 The ecosystem approach to health in wetlands

A perception of health with an operational context of only medical and veterinary hospitals and pharmaceutical companies comes from a societal focus on ill-health and emergency care; these dominate the thought processes, funding and expenditure in health. Whilst this focus is no doubt important, it distorts the health equation, and does not address what 'determines' health (or ill-health). That failure can result in unnecessary burdens of disease for humans, domestic and wild animals.

An ecosystem approach to health, instead, works further 'upstream' – closer to the driver of the problem. The approach is **preventative** recognising that 'prevention is better than cure' and, for wetlands, focussing at a landscape or catchment scale ensures maintenance of social and ecosystem services. This approach then seeks to establish the societal and environmental conditions for good health, bringing long-term savings for medical and veterinary costs and overall maximising benefits and minimising costs for wetland stakeholders, particularly those most likely to be affected by specific health issues.

Embracing an ecosystem approach to health in wetlands **recognises a fundamental connectivity:** the health and well-being of humans, domestic stock and wildlife is played out where wetlands are the **setting**, or the context; achieving a 'healthy wetland' through wise use, most often at a broader landscape and/or catchment scale. Managing disease within one sector without consideration of the others not only misses opportunities for improved health outcomes for more sectors, but importantly may result in negative health outcomes in other sectors, and feedback unintended consequences for the original sector in the long term. Seeing 'health' as a property of a(n eco)system, allows for more effective and widespread outcomes.

The 'One World One Health' and 'Ecohealth' movements arose due to the appreciation of this interdependence on, and connectivity between, health of humans, domestic livestock and wildlife and their social and ecological environment, **understanding disease dynamics in broader contexts** of sustainable agriculture, socio-economic development, environment protection and sustainability, and complex patterns of global change.

A fundamental aspect of taking an ecosystem approach to health is that it is **participatory** with stakeholders understanding that they can create or solve problems relating to their health and that of their livestock and wider environment. Given the complex relationships between humans and other biodiversity, the complexities of resource use, including barriers to sustainable resource use, improved health outcomes are maximised when more stakeholders are on-board and engaged. This is not an easy accomplishment and processes that allow for genuine co-operation and mutual understanding of quite different organisational sectors is required.

It is worth appreciating the consequences of not taking an ecosystem approach to health in wetlands. Wetlands as settings for lifestyles and livelihoods can deteriorate, and negatively affect health in this way. Activities which negatively affect wetland functions and services can create wetlands which actively pose health risks such as exposures to toxic materials and/or water-borne, or vector-borne diseases. Whilst steps can be taken to ameliorate these risks, the risks can increase (sometimes dramatically) if disruption to ecosystems, and the services they provide, continues.

2.4 Why disease management should be integral to wetland management

Control of disease in wetlands brings broad scale benefits even if these benefits may be somewhat intangible as 'absence' of a problem is often not fully appreciated nor costed appropriately. Current wetland management practices focussed at maintaining wetland function and wetland benefits usually also address disease prevention and control. However, there will be strategies for disease management that are additional to traditional management practices that once integrated, provide additional gains.

To view disease management as separate to other forms of land and wildlife management ensures that opportunities for good disease prevention will be missed. Wetland managers are the key stakeholders in delivering healthy wetlands and, as such, all efforts should be made to integrate disease management thoroughly within wetland site management plans and other stakeholder activities at wetlands.
►Section 3.1.3. Integrating disease management into wetland management plans.

2.5 Control of infectious disease and invasive alien species

It has been estimated that damage caused by invasive alien species worldwide amounts to almost five percent of the world economy. Invasive alien species of flora and fauna are considered the second biggest threat after habitat loss and destruction to biodiversity worldwide, the greatest threat to fragile ecosystems such as islands, and are a major cause of species extinction in freshwater systems. Climate change may also exacerbate the spread of non-native species as warmer temperatures may allow currently 'benign' non-native species to potentially extend their ranges and become invasive.

Invasive species impact native species in a wide range of ways, including competition, predation, hybridisation, poisoning, habitat alteration and disease. With respect to the latter, invasive alien species can carry novel pathogens non-symptomatically, to which native species may have no natural immunity. This can lead to population reduction and extinction in native species *e.g.* crayfish plague carried non-symptomatically by introduced North American signal crayfish *Pacifastacus leniusculus*, causes disease and mortality in European freshwater crayfish *Austropotamobius pallipes* [►**Section 4.1.12. Crayfish plague**], and amphibian chytridiomycosis carried non-symptomatically by introduced species such as American Bullfrogs *Lithobates catesbeianus* causes population declines and plays a role in amphibian extinctions [►**Section 4.1.2. Amphibian chytridiomycosis**].

There are many parallels between prevention and control of invasive alien species, and of infectious diseases, such as the proactive measures of:

- Risk analysis and assessment ►**Section 3.1.1. Risk assessment**
- Contingency planning ►**Section 3.1.4. Contingency planning**
- Surveillance (in particular horizon scanning) ►**Section 3.3.1. Surveillance and monitoring**
- High standards of biosecurity ►**Section 3.2.4. Biosecurity**
- Information and public awareness campaigns e.g. public education on measures to reduce the risk of introducing invasive species and how to recognise those species if they are present in an area ►**Section 3.5. Communication, education, participation and awareness**
- Training regarding management of those species ►**Section 3.5.2 Building capacity by education and training**
- Communication between governments and national organisations as invasive species often spread from one country to the next and advance warning allows time to increase biosecurity and surveillance strategies ►**Section 3.5.1 Communication and public awareness**

Given the dual benefits of reducing risk of invasive alien species and the pathogens they carry, these are worthwhile measures to take.

2.6 Strategies for managing animal disease

Proactive strategies

Proactive strategies aim to prevent disease introduction or an outbreak of existing disease and will always be more cost effective than dealing with the consequences of disease emergence. In general, to apply the concept of wise use and maintain biodiversity and ecological function *i.e*. maintain healthy wetlands, will provide the greatest contribution to health.

Figure 2-4. Maintaining healthy wetlands by applying the concept of wise use is the single greatest contributor to health in wetlands (*WWT*).

Although a good understanding of disease dynamics is needed for the most effective proactive disease control strategies, there are some basic generic principles which, if implemented, are likely to reduce risks of disease emergence. For example, strategies for biosecurity (including prevention of introduction of invasive alien species), reduction of stresses on hosts and environment, and prevention of pollution, will bring obvious health benefits. Table 2-1 provides a list of proactive practices for disease prevention and control and the locations of further information in Chapter 3.

Table 2-1. Proactive practices for prevention and control of disease in wetlands.

Practice	Section of Manual for further information
Healthy wetland management	*Wise use of wetlands*
Site-specific risk assessments	►**Section 3.1.1**
Formation and utilisation of multidisciplinary advisory groups	►**Section 3.1.2**
Integrating disease management into site management plans	►**Section 3.1.3**
Contingency plans which are tested regularly	►**Section 3.1.4**
Reducing stressors at a site	►**Section 3.2.1**
Disease zoning and use of buffer zones	►**Section 3.2.2**
Standards for movements of domestic livestock and restrictions where appropriate	►**Section 3.2.3**
Biosecurity measures	►**Section 3.2.4**
Surveillance and disease monitoring programmes	►**Section 3.3.1**
Identifying a disease problem	►**Section 3.3.2**
Control of vectors where appropriate	►**Section 3.4.3**
Vaccination programmes	►**Section 3.4.4**
Communication, education, participation and awareness raising programmes for wetland stakeholders in disease prevention and control to help develop a 'culture' of disease management	►**Section 3.5**

Reactive strategies

Reactive strategies, once a disease has emerged and/or been identified, aim to: reduce spread; impact; and potential for disease to become established and create a longer term problem.

Reactive strategies may include determining an evidence base, conducting surveillance, animal movement restrictions and instigating various other control measures. Reactive strategies for complete disease eradication may involve substantial intervention.

With such a wide variety of wetland stakeholders, it is important to appreciate that there is the potential for differences in opinions over reactive disease control strategies and thus cross-cutting education, awareness raising and communication about these activities is advisable, particularly where rapid responses to disease emergence are required. Table 2-2 provides a list of reactive practices for disease control.

Table 2-2. Reactive practices for control of disease in wetlands.

Practice	Section of Manual for further information
Utilisation of multidisciplinary advisory groups in response to outbreaks	►**Section 3.1.2**
Further integrating disease management into site management plans	►**Section 3.1.3**
Implementation of contingency plans which are tested regularly and refined as necessary	►**Section 3.1.4**
Reducing stressors at a site	►**Section 3.2.1**
Disease zoning and use of buffer zones	►**Section 3.2.2**
Standards for movements of domestic livestock and restrictions	►**Section 3.2.3**
Biosecurity measures	►**Section 3.2.4**
Surveillance and disease monitoring programmes	►**Section 3.3.1**
Investigation of outbreaks	►**Section 3.3.5**
Disinfection and sanitation	►**Section 3.4.1**
Control of vectors	►**Section 3.4.3**
Vaccination programmes	►**Section 3.4.4**
Habitat modification	►**Section 3.4.6**
Movement restrictions	►**Section 3.4.7**
Eradication, elimination, stamping out and lethal intervention	►**Section 3.4.8**
Communication, education, participation and awareness raising programmes for wetland stakeholders in disease prevention and control to help develop a ‘culture’ of disease management	►**Section 3.5**
Risk communication and dealing with the media	►**Section 3.5.1**

All these practices are detailed in Chapter 3. Their application is illustrated in the case studies throughout the Manual and in the ‘Prevention and Control in Wetlands’ sections of the disease factsheets in Chapter 4.

2.7 Communication, education, participation and awareness (CEPA)

The vision for the Ramsar Convention’s CEPA programme is “people taking action for the wise use of wetlands”. All wetland stakeholders (such as wetland managers, local wetland users including farmers, hunters and fishers, and local government agencies, community leaders and NGOs) should understand the basic principles of healthy habitat management and the action they can take for disease prevention and control. Wetland users do not need to become disease experts but communication and awareness raising programmes should aim to increase motivation to become engaged and ‘do the right thing’, with respect to disease management. This will likely only come from becoming informed about the problem, understanding the issues and implications, and participating in the solutions.

Developing capacity to undertake disease management may involve formal education and training of key personnel *e.g.* land managers or appropriate authorities. Ideally disease training should be part of other wetland management or wetland-related training to convey its integral nature and to avoid it becoming detached from day to day practices. Frequency of training will depend on the disease issue *e.g.* there may be merit in provision of brief annual refresher training for a seasonal disease, or to coincide with changes in wetland management practices. Education and training for those involved in high risk activities *e.g.* a large-scale disease control operation, are essential to protect public health (if the disease is zoonotic) and potential for further spread of disease.

Communication networks of key wetland stakeholders, including disease control authorities, should be established in 'peacetime' to facilitate rapid disease control responses should the need arise.

This Manual aims to provide some of the information as a foundation for communication and public awareness programmes.

►Section 3.5. Communication, education, participation and awareness (CEPA)

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- The **greatest power to prevent disease** emergence in wetlands **lies in the hands of wetland managers** and other wetland users.
- The concept of **'One World One Health'** has arisen due to the appreciation of the **fundamental connectivity in health of humans, domestic livestock and wildlife**.
- Embracing an **ecosystem approach to health in wetlands** involves **recognising the dependence of health and well-being on 'healthy wetlands'** which can only be achieved through wise use, most often at a landscape and/or catchment scale.
- **'Prevention is better than cure'** and an ecosystem approach to health, maximises benefits and minimises costs for wetland stakeholders.
- **If wetland stakeholders understand** both the impacts of diseases and how to prevent and control them, **they will feel motivated and empowered** to take action. Stakeholder understanding must be built through effective communications or training but action will also be influenced by capacity to respond.
- **Understanding disease** in its broadest terms (*i.e.* not just in terms of life and death) and its overt and subtle effects on individuals and populations, and the factors that affect this, **allows a better appreciation of how to manage them** effectively.
- **To view disease management as separate** to other forms of land and wildlife management **ensures that opportunities for good disease prevention will be missed**. Therefore**, integrating disease management into wetland management** means putting disease consideration at the heart of the wetland management planning process.
- **Effective management of any disease** is dependent on **a good understanding of its epidemiology and the ecology of host populations**. The dynamics of disease in wildlife populations can be highly complex, and disease management interventions can have unpredictable outcomes.
- **Invasive alien species and novel pathogens and parasites have many parallels** in their biology, the risks they pose, and **in the measures required to prevent their establishment** and control. Prevention of their introduction is preferable to subsequent control, and wetland management practices aimed at prevention of any of these can provide additional benefits and protection from all.

- **A broad range of proactive and reactive strategies and practices are available** to the wetland manager and other wetland stakeholders to achieve or maintain the health of the ecosystem including:

   1. **Targeting the environment and land use** *e.g*. healthy habitat management including wise use; maintaining appropriate water quality and quantity; reducing risk from pollutants and toxicants; and manipulation of habitat to reduce disease agents or their invertebrate vectors.

   2. **Targeting host populations** *e.g*. maintaining good nutritional status; reducing stressors; managing density of domestic animals and wildlife; reducing contact between domestic animals and wildlife (including zoning); and vaccination or veterinary treatment.

   3. **Targeting pathogens and parasites** *e.g*. managing biosecurity; hygiene, disinfection and sanitation; and interrupting transmission by exploiting weaknesses in a parasite's life cycle, such as targeting intermediate hosts and/or their preferred habitat.

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Chapter 3
General Management Practices

In this chapter you will find:

A standardised procedure for completing a disease risk assessment.

Guidance on how to incorporate disease management into management plans for wetlands.

Guidance for reducing the risk of emerging disease.

Guidance for detecting, assessing and responding to disease outbreaks.

Guidance for managing disease.

Case studies: descriptions and photos of wetland managers' experiences responding to disease problems.

Chapter contents

3.1 Assessing risk and planning for the future

Many ecological and anthropogenic factors may affect the risk of disease emergence or re-emergence in wetlands, including:

- presence of human populations
- presence of domestic livestock
- presence of non-native species
- seasonal influxes of wild or domestic species
- trade
- industry
- agricultural activities
- habitat loss or modification
- weather
- impacts of climate change.

Dealing with such a diversity of factors can seem daunting. However, a risk assessment approach can be used to quantify and/or qualify risks and so help the wetland manager to identify an appropriate course of management actions.

The wetland manager is not expected to be a disease expert. Understanding diseases of wildlife and domestic stock, and their public health implications, within wetlands requires a multidisciplinary approach. Advisory groups reflecting a diverse range of knowledge and understanding for specific or general issues, can significantly improve risk assessments and advise on best courses of actions that safeguard both livestock interests and wildlife protection appreciating that these may sometimes seem to be at odds. This is illustrated by the use of advisory groups to deal with highly pathogenic avian influenza H5N1 where it is important to appreciate human health issues, poultry trade and economic issues, alongside factors relating to wildlife such as the behaviour and movements of migratory birds.
►Case study 3-1. UK Ornithological Expert Panel – integrated expertise for dealing with highly pathogenic avian influenza H5N1.

To ensure the principles and specific actions for disease management and control are embedded within the management practices at a wetland site, they should be written into management plans and updated, as and when, disease risks change.

Disease control in endemic disease situations depends upon engagement of all key stakeholders together with their sustained participation and cooperation. Disease control in outbreak situations is similarly dependent on stakeholder engagement but also requires preparedness for a rapid response. Appropriate contingency planning helps to reduce response times and promotes the likelihood of an effective resolution. Contingency plans should be developed in ‘peacetime’ *i.e*. in advance of disease problems, when it is possible to fully consider current or potential issues, determine appropriate courses of action, develop relationships and communication channels, and test plans (*e.g.* by means of exercises) to evaluate whether or not they are fit-for-purpose. It is sensible to build a degree of flexibility into contingency plans as unexpected local conditions may be important in determining the action to take. Following implementation of contingency plans, they should be critically reviewed and updated.

This section contains further information on the following topics:

- *Risk assessment*
- *Advisory groups*
- *Integrating disease into wetland management plans*
- *Contingency planning*

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- To ensure consideration for **disease prevention and control** is **at the heart of wetland management**, activities need to be **integrated into wetland management plans**. Clearly defined roles and responsibilities are required to ensure effective management which can deliver a range of benefits to stakeholders.
- **Risk assessments are valuable tools for animal health planning** and serve to identify problems/hazards and their likely impact thus **guiding wetland management practices**. From these assessments, risk management and communication actions can be taken. **Good** local, national and regional **surveillance data** are needed **for robust risk assessments**. Risk assessments are living documents which require regular revision.
- **Multidisciplinary advisory groups provide a broad range of benefits** for disease prevention and control. Their role is to review epidemiological and other disease control information, inputting to the activation of agreed contingency plans and advising the appropriate decision makers on future contingency planning. As appropriate, wetland managers can play a key role in these groups.
- **Contingency plans aim to consider possible emergency disease management scenarios** and to integrate **rapid cost effective response actions** that allow the disease to be prevented and/or controlled. It is advisable to develop bespoke contingency plans for specific high-risk/high-priority diseases and also generic standard operating procedures (SOPs) that may be common to many situations. Plans and SOPs should be documented and tested with a broad range of stakeholders in 'peacetime' (*i.e*. outwith any emergency situation), and subjected to periodic review.

3.1.1 Risk assessment

Risk assessment is a tool for the identification of potential problems and/or hazards, evaluation of their likelihood and probable magnitude. Risk assessments provide data to permit the effective management of risks.

Risk assessments should be based on the best available data, which may be quantitative or qualitative. Quantitative assessment of risks associated with wildlife diseases is often difficult due to complex disease dynamics and absence of robust biological data. Qualitative assessments of risk are more usual, within which, risks may be described as “extreme”, “high”, “medium” or “low” or a simple scoring system may be employed. Risk assessments should be revised in the light of new data or changing circumstances.

The Ramsar Convention’s Wetland Risk Assessment Framework (Ramsar Convention Secretariat 2010) provides a mechanism for predicting and assessing change in the ecological character of wetlands. This framework (Figure 3-1) provides an appropriate general approach to problem identification, impact prediction, estimation of the extent of impacts, and overall assessment of the risk of adverse impacts.

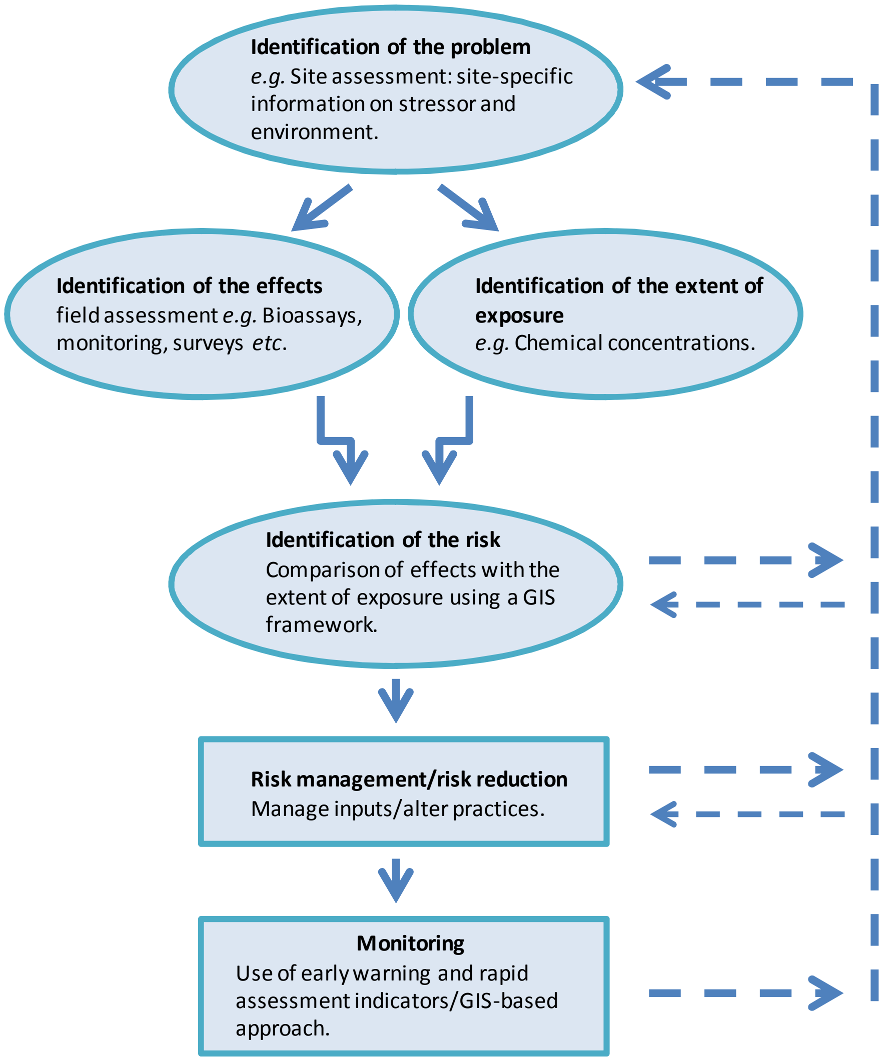

Figure 3-1. Model for wetland risk assessment (*Ramsar Convention Secretariat 2010*)

Problem or hazard identification

A system to help identify potential problems or hazards including current disease trends and outbreaks should be put in place. This may include site-specific information or surveillance data, or searching scientific literature and using sources of contemporary disease information and outbreaks, such as:

- The World Organisation for Animal Health (OIE), which provide up to date disease information and annual disease reports. Information for diseases and associated findings can be found at http://www.oie.int/our-scientific-expertise/reference-laboratories/annual-reports/ and information on aquatic diseases can be found at http://www.oie.int/international-standard-setting/specialists-commissions-groups/aquatic-animal-commission-reports/.

- Food and Agriculture Organization (FAO), which can provide additional disease intelligence, for example in the EMPRES transboundary animal diseases bulletin, which is published quarterly at http://empres-i.fao.org/empres-i/home.
- The Programme for Monitoring Emerging Disease (ProMED) which is a forum for rapid dissemination of official and unofficial information on animal, plant and human disease occurrences globally (available as Internet server and mailing service) at http://www.promedmail.org/pls/apex/f?p=2400:1000.

Useful key information relating to potential disease problems includes:

- Species known or likely to be affected.
- If infectious, the pathogenicity of the pathogen or parasite (and *e.g.* how this varies across species).
- If toxic, the nature of the toxin.
- Zoonotic potential.
- Potential for transmission between domestic/captive and wild animals.
- Routes of transmission.
- Potential for pathogen or toxin persistence in the environment.
- Presence and movements of vulnerable wildlife in and around the site.
- Activities of vulnerable domestic animals in and around the site.
- Human activities contributing to potential problems or hazards in and around the site (*e.g.* agriculture, tourism, industry).

Identification of the adverse effects and/or extent of the problem

For each problem or hazard identified, the effect and extent of the potential impact needs to be evaluated and described. This process can be difficult in view of the scarcity of information relating to wetland disease epidemiology, however, the following sections help to provide a general framework for making these evaluations:

What sort of effects may occur and to what extent?

For example:

- The most obvious consequences may be direct mortalities or morbidities of varying scales.
- There may be indirect mortalities and morbidities, *e.g.*, loss of prey may impact on predator populations.
- Livestock losses may impact livelihoods.
- There may be possible economic consequences.
- If the problem is an OIE- notifiable disease outbreak, a site may be subject to severe restrictions including possible closure.
- There may be possible welfare issues.
- Tourism revenue may be affected.
- If the disease is zoonotic then measures may have to be put in place to reduce human exposure.

Which species or individuals are at risk and to what extent?

For example:

- Range of species potentially affected (including domestic animals, wildlife and humans).
- Species at higher risk due to behaviours (*e.g.* congregating at water holes) or ecological characteristics (*e.g.* residing in certain areas at particular times of the year).
- Status of hosts present (*e.g.* some diseases may only have an adverse impact on hosts in poor condition).
- Threatened species (present in low numbers and/or fragmented populations) may be at particular risk from disease.

When and for how long is the problem likely to occur

For example:

- Disease risks may be seasonal and the range of wild or domestic species present may vary accordingly (*e.g.* there may be seasonal grazing of domestic livestock; wild populations may be residents, breeding visitors, non-breeding visitors, passage migrants, nomadic or irruptive species).
- There may be relatively predictable times of increased risk due to human and livestock activities. For example, during times of livestock movements, when people or vehicular access to the site is greater or when there is application of fertilizer which may contain potentially infected manure.

Where is the problem likely to occur and how widespread would it be?

For example:

- Wetlands tend to be connected so potential for aquatic spread of toxins or pathogens needs to be assessed.
- Species are likely to be unevenly distributed within a site due to different habitat preferences and daily behavioural patterns (feeding, roosting, resting, bathing/drinking). Some species will be present in dense flocks or herds, some in loose aggregations, and others as small groups or individuals, and different species may mix with one another to varying degrees.
- For infectious diseases it is worth appreciating how infection may be moved into and out of a wetland into surrounding areas by animal movements. For example, waterbirds may feed on adjacent agricultural fields and fish-eating birds like cormorants may commute between wetlands, rivers, farmed fishponds and coastal areas. So called ‘bridge species’ (*e.g.* birds and mammals, such as rodents associated with human habitation) have the potential to carry disease between wetlands and agricultural premises and have been speculated to be involved in spread of avian influenza.
- Some wildlife species will remain far from human habitation whilst others are attracted as it offers benefits such as food sources, shelter, nesting and safety from predators.

Identification of the risk

This involves integrating the results from the assessment of likely effects with those from the assessment of the likely extent of the problem and generating an overall assessment of the level of risk, for example:

Risk	Impact
Negligible	Impact so low so does not merit consideration
Very low	Impact very low but cannot be excluded
Low	Impact low but requires consideration
Medium	Impacts and requires consideration
High	Impacts and requires great consideration and mitigation
Very high	Impacts greatly and requires great consideration and mitigation

A range of techniques exist for estimating risks, including spatial approaches using Geographic Information Systems (GIS) to link different components (*e.g.* poultry facilities on or near the site, other human activities, distribution of key species at the site across different seasons, seasonal changes in water levels leading to concentrations of wild bird species or other wild and domestic animals, important resting/roost sites, wetland margins and crop patterns in adjacent landscapes).

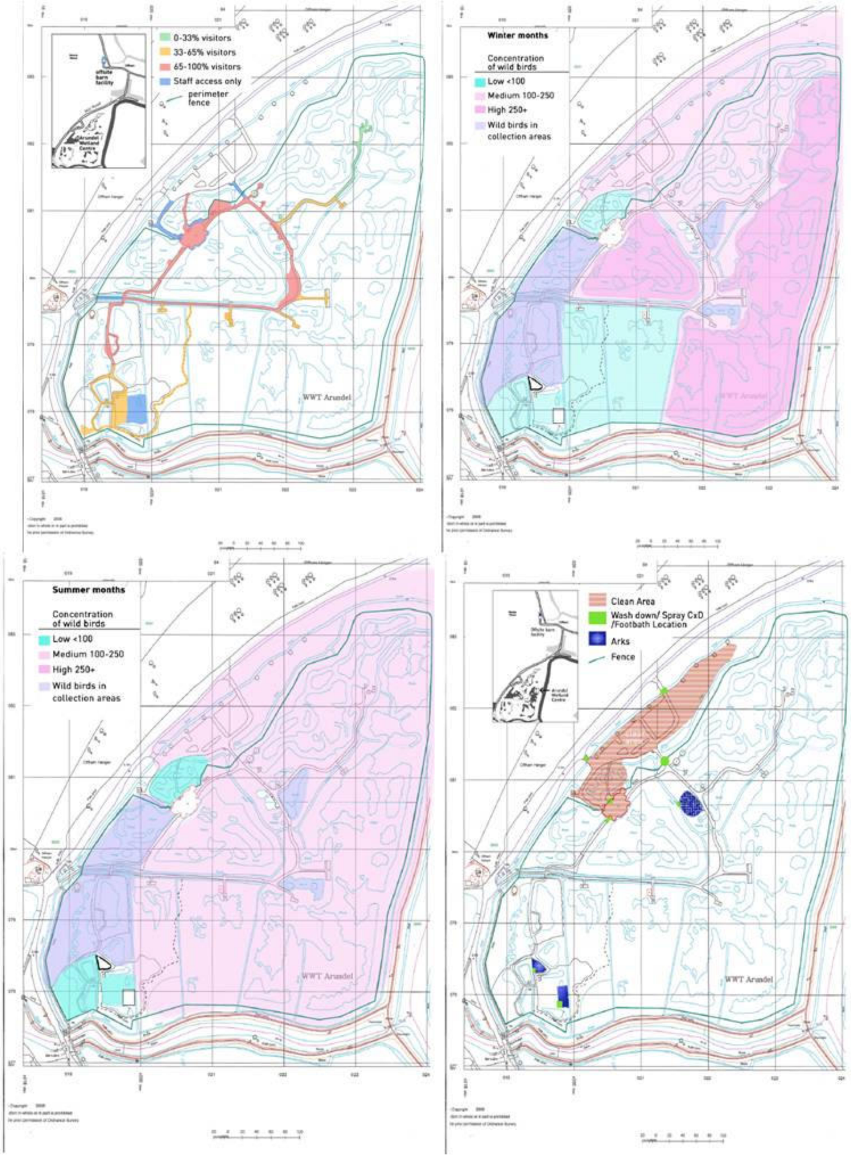

Figure 3-2. GIS maps of a wetland site indicating layers for seasonal bird densities, people movements and specific facilities: GIS offers a valuable tool to aid development of risk assessment.

Risk management is the practice of identifying, documenting and implementing measures to reduce risks and their associated consequences. Although risks can rarely, if ever, be totally eliminated the aim is to implement actions that reduce the risks to an acceptable level. These following sections illustrate the types of practical risk management practices that can be implemented at wetland sites:

►**Section 3.2 Reducing risk of disease emergence.**
►**Section 3.3 Detecting, assessing and responding to a new disease.**
►**Section 3.4 Managing disease**.

Risk communication is the route for stakeholders (everybody that could be affected) and risk analysts to exchange information and outlooks on risks. Stakeholders should be consulted throughout each process to ensure ownership of decisions.

Public health communication may require knowledge of points of contact and a strategy to disclose information.

Further information and sources

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World Organisation for Animal Health (OIE) **Reference Laboratory – Annual Reports**. http://www.oie.int/our-scientific-expertise/reference-laboratories/annual-reports/. [Accessed March 2012].

World Organisation for Animal Health (OIE) – **Aquatic Animal Commission and Reports.** http://www.oie.int/international-standard-setting/specialists-commissions-groups/aquatic-animal-commission-reports/. [Accessed March 2012].

3.1.2 Advisory groups

Dealing with outbreaks of disease in wetlands, particularly where wildlife is involved, requires a multidisciplinary approach. Multidisciplinary advisory panels of 'experts' (or perhaps, in the absence of a panel, a small number of personnel providing a range of expertise) can help to fulfil this requirement. Advisory groups should be integrated with any government (local or national) disease response, where appropriate.

The role of the advisory group is:

- to review epidemiological and other disease control information
- to input to the activation of agreed contingency plans
- to maintain oversight of the disease campaign
- to advise the appropriate decision makers on future contingency planning and on implementation of the plans.

Such groups may include expertise from human, animal and wildlife health professions, together with wildlife managers and the wetland manager. The scale at which advice is sought will depend on how government/local authorities are structured but advice should be available to key decision makers whether they are at national or sub-national (*e.g.* provincial) scales.

Composition

Advisory groups should comprise best available expertise drawn from both governmental and non-governmental sectors, including wetland managers, experts from research institutes, universities and other key groups as appropriate. There should be close collaboration with relevant species monitoring schemes, in order to facilitate rapid analysis of data and information from relevant databases and other information sources.

Establishment

Advisory groups should be established in advance of disease outbreaks as part of forward contingency planning and should be integrated into existing governmental processes or disease control systems.

The group should preferably be part of any epidemiological team that has responsibility to investigate disease outbreaks, or sufficient communication structures to allow easy and rapid information exchange. The relationship between the advisory group within other government disease response processes and structures should be explicitly established from the outset.

Modes of working

Contingency planning should include means of bringing together relevant experts at short notice in order to provide timely advice to decision-makers. Experts on advisory groups should be kept informed on the epidemiological features of any outbreak involving impacts on wetland wildlife, livestock and humans, and on the progress of such investigations. Planning should include alternative mediums of communication, such as teleconferencing, in anticipation that not all experts will be able to physically attend meetings.

Emergency field assessments

Emergency field assessments may be necessary to rapidly establish the nature and extent of a disease outbreak and their requirement should be considered in contingency plans. Such assessments may involve collecting information on animals affected by disease and disease-carrying vectors. Field assessments should be complemented by rapid desk-based data assessments that aim to analyse available data sources and thus to inform risk assessments.

International networking

Risk assessments, evaluations and relevant data should be shared between neighbouring countries or within wider geographic regions. Therefore, national advisory groups should collaborate together at regional scales to develop collective international assessments and understanding.

Lessons learned

Following the activation of the advisory group in the event of an outbreak, it is important afterwards to undertake a formal review to identify any problems or areas of operation where there may be scope for improvement of activity. The outcomes of such a review should inform future contingency arrangements.

CASE STUDY 3-1. UK Ornithological Expert Panel – integrated expertise for dealing with highly pathogenic avian influenza H5N1.

The spread of highly pathogenic avian influenza H5N1 of Eurasian lineage towards Europe in late 2005 and early 2006 stimulated the UK's Ministry responsible for animal health issues (Department for Environment, Food and Rural Affairs – DEFRA) to set up an 'Ornithological Expert Panel' (OEP) made up of ornithologists and others from the statutory conservation agencies and relevant non-government organisations to provide technical advice to DEFRA on a continuing basis. It operates responsively, with members being available at any time to provide advice and information on wild birds and their movements in the context of avian influenza. Among other tasks, the OEP has advised on:

- the risk of HPAI H5N1 occurring in the UK given known presence in nearby countries[1];
- the significance of risk factors, such as periods of extreme cold weather, which may heighten the risk of movements of birds carrying HPAI H5N1 to the UK;
- the development of risk-based national surveillance programmes for avian influenza (*e.g.* Snow *et al.* 2007);
- the generic design of routine ornithological assessment procedures to be undertaken at locations where infection with HPAI H5N1 may be suspected;
- the undertaking of emergency ornithological field assessments as part of immediate epidemiological investigations at suspected infection sites;
- interrogating organisational databases and other information sources to provide summaries of bird species most likely to be in the area of outbreak sites, and/or moving to or from that area; and
- using networks of personal contacts in neighbouring countries to provide 'real-time' information on movements of birds and other relevant international information and contexts to the developing situation within the UK.

Seeing outbreak sites with ornithologists' eyes

In order to assist epidemiological investigation, emergency field assessments were undertaken to establish the nature of, and collect information on, populations of wild birds near outbreak sites. Two examples are summarised below:

Turkeys in Eastern England, 2006

An outbreak of HPAI was reported on 1 February 2006 from a turkey production facility associated with a slaughterhouse and two large processing plants for poultry products in eastern England. Genetic sequencing of the virus showed it to be virtually identical (almost 100% homologous) to that recovered from a Hungarian outbreak in farmed geese the previous month. This level of similarity suggested the virus was either transferred directly between the Hungarian geese and the eastern England turkey outbreak, or that they shared a common source.

Rapid consultation with the OEP confirmed a lack of known mid-winter movements of wild birds between Hungary and England that might have been a vector for the infection.

An emergency field assessment of the infected premises identified the following factors:

- lack of proximity of the infected premises to areas used by migrant waterbirds;
- however, potential access to the infected premises by small non-migrant birds, rats and mice, and use of the area by a significant local population of gulls (*Larus spp*.); and
- presence of waste meat from the processing plant, and potential access to it by gulls that were moving between the factory area and roofs of the production sheds.

The final epidemiological assessment was that infected turkey meat had been transported from Hungary

[1] 'Qualitative Risk Assessments' - for example at http://webarchive.nationalarchives.gov.uk/20080108002802/http://defra.gov.uk/animalh/diseases/monitoring/pdf/hpai-h5n1-developments060706.pdf. [Accessed March 2012].

to England in January 2006, with discarded meat being scavenged by local gulls, probably carrying infected scraps onto the roof of a nearby turkey production shed. Heavy rain then washed virus into the shed (which was in a poor state of maintenance) infecting young turkey poults.

Ornithological assessments both of the site and through desk studies of the surrounding area were critical to rapidly ruling out wild birds as the vector which transferred the virus from Hungary to England, and also to identifying the probable means by which the virus was transferred from external waste meat containers at the factory to turkey sheds *via* the agency of gulls, rats or mice[2] — an ultimate consequence of poor biosecurity.

Mute swans on The Fleet, England, 2008

HPAI H5N1 was confirmed from three dead mute swan *Cygnus olor* carcases collected in late December 2007 and January 2008 from a population of 750 largely resident swans using a lagoon known as The Fleet on the coast of southern England. The virus was later detected in several further swan carcases from the same area.

Key elements of OEP advice were that:

- swans, and/or other wildfowl using The Fleet could potentially move within the lagoon to adjacent nature reserves and poultry, but that, as the hinterland of this wetland was rather dry, movements of >3 km were unlikely. This influenced the shape and size of the statutory control zones (Wild Bird Control and Wild Bird Monitoring Areas)(Figure 3-3);
- analysis of the movement records of the infected swans (shown by previously reported observations of individual rings) showed little evidence of off-site movements, thus indicating that they were unlikely to have been the vectors which bought the virus to the area;
- that there was potential for human exposure in some areas used by gulls which potentially may have carried the virus. This led to the erection of warning signs in those areas where people may have come into contact with gull faeces;
- there was a potential risk that local wildfowling might disturb and disperse infected birds. This led to the establishment of a no-shooting area centred on The Fleet (Figure 3-3).

The mute swans present were largely resident and so were unlikely to have been the vectors that brought the virus to The Fleet and unfortunately, the ultimate vector was never determined[3]. However, the disease control operation was successful and the statutory control zones were appropriate as no cases were found outside these areas.

Lessons learnt from the UK experience

- **Establishment and organisational placement:** the OEP was established in advance of disease outbreaks as part of forward national contingency planning and as part of the wider epidemiological team that had the responsibility to investigate HPAI outbreaks. This integration greatly assisted in the identification of achievable objectives, and in making explicit the formal relationship between the OEP and other UK government disease response processes and structures.

- **Composition:** the OEP comprised best available ornithological expertise drawn from both governmental and non-governmental sectors, including ornithological experts from research institutes. Staff from the UK national bird ringing and waterbird monitoring schemes were involved to facilitate rapid analysis of data and information drawn from relevant databases and other information sources.

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[2] http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/ai/documents/epid_findings070405.pdf. [Accessed March 2012].

[3] http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/ai/documents/epireport-080212.pdf. [Accessed March 2012].

- **Mode of working:** in order to facilitate the rapid convening of advisory expertise from multiple geographically separated experts, teleconferencing was used extensively and successfully. A significant effort was made by DEFRA to ensure that OEP members were made aware and kept up to date on the epidemiological features of outbreaks involving domestic poultry and the progress of the epidemiological investigations. This helped the quality of the advice provided by OEP to the benefit of refining epidemiological assessments.

- **Field assessments:** these proved invaluable in assessing local concentrations of wild birds and their degree of access to domestic poultry and so focussed wider epidemiological investigations. In some cases field assessment enabled the rapid exclusion of wild birds as probable sources of infection and the more rapid identification of other factors (*e.g.* trade in poultry meat, as described above) as the vector for HPAI H5N1 infection. Further ornithological advice on additional and specific surveillance was frequently sought following these assessments.

- **Desk-based assessments:** field assessments should be complemented by desk-based rapid ornithological data assessments that seek to interrogate available data sources and, thus, to inform risk assessments.

- **Reviewing lessons learnt:** following the activation of the OEP in the event of an outbreak, formal 'lessons learnt' reviews were undertaken to identify any problems or areas of operation where there was scope for improvement of activity[4]. The outcomes of such reviews were then implemented by modifying contingency arrangements. This enabled progressive 'learning from experience'.

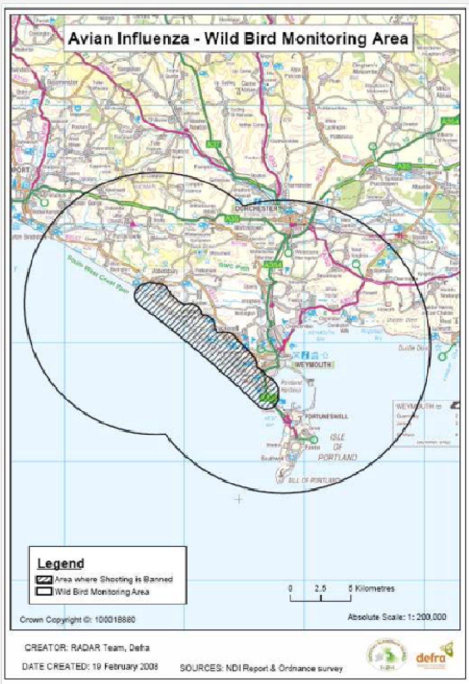

Figure 3-3. Extent of the statutory Wild Bird Monitoring Area established in January 2008, and of the area where shooting was banned, both centred on The Fleet, England (*Defra*).

Reference

Snow, L.C., Newson, S.E., Musgrove, A.J., Cranswick, P.A., Crick, H.P.Q. & Wilesmith, J.W. (2007). **Risk-based surveillance for H5N1 avian influenza virus in wild birds in Great Britain**. *Veterinary Record,* 161: 775-781.

Case study provided by David Stroud, Joint Nature Conservation Committee, UK

[4] for example, http://archive.defra.gov.uk/foodfarm/farmanimal/diseases/atoz/ai/documents/holtonlessonslearned-070803.pdf. [Accessed March 2012].

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3.1.3 Integrating disease management into wetland management plans

Wetland management plans are usually 'living' documents tailored to a specific site and aimed at delivering a clear set of objectives. Figure 3-4 provides a recommended structure and content for such a plan (Ramsar Convention 2002).

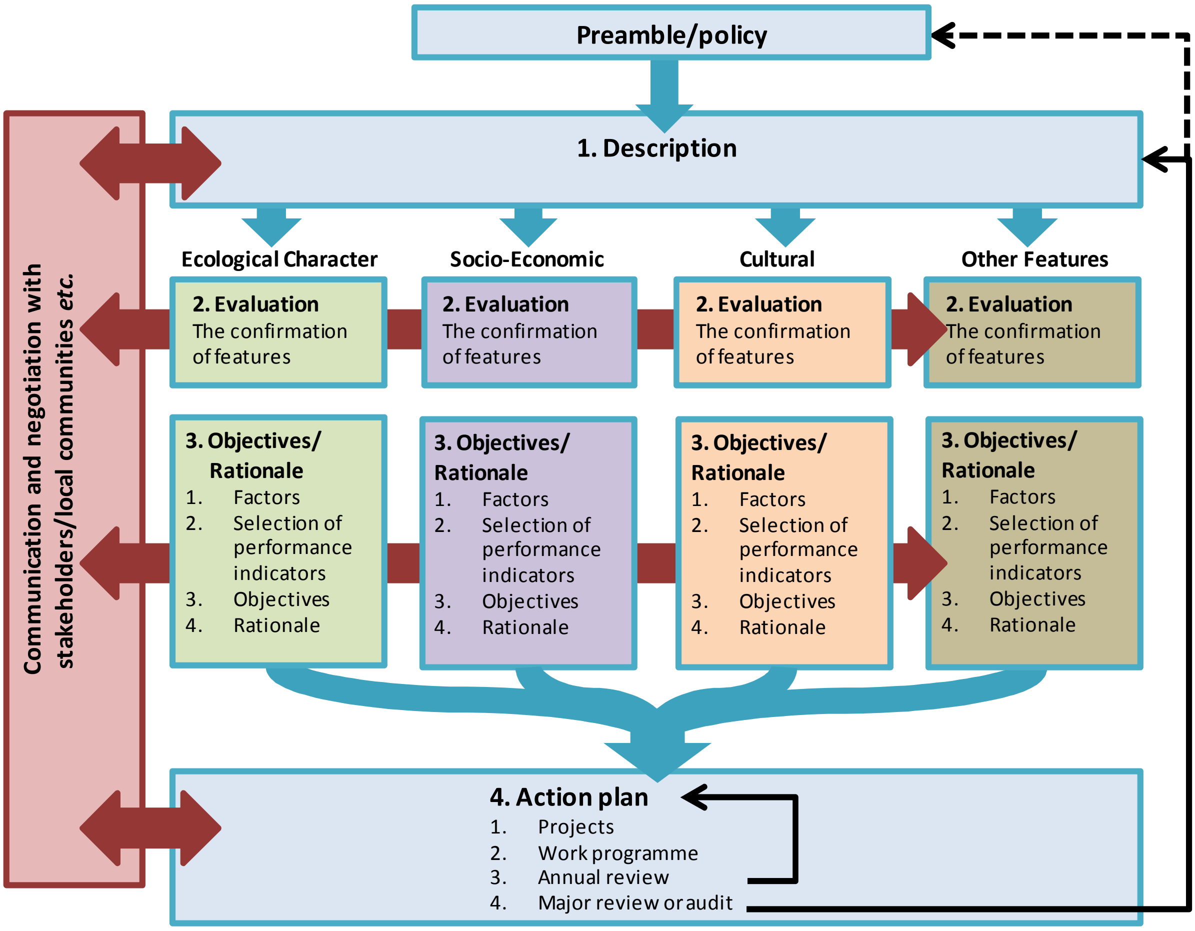

Figure 3-4. Recommended structure and content of a management plan for a Ramsar site or other wetland (*Ramsar*).

Why integrate disease management into management plans?

This bringing together within one document of information about biological characteristics, (*e.g.* climate, geology, hydrology, habitat type, biodiversity, ecological linkages), socio-economic factors (*e.g.* agriculture, consumptive use), cultural factors (*e.g.* recreational activities) and other attributes (*e.g.* boundaries, legal land ownership, surrounding land use *etc.*) of sites for the purpose of management, also provides a framework for informing disease risk assessments and contingency plans. Integration within the management plan ensures that disease management is at the heart of site management by a wide range of stakeholders.

Integrating disease management within a wetland management plan brings a number of benefits:

1. It ensures that disease management is not, nor is it perceived as, a set of activities that are distinct from site management.

2. It identifies disease prevention as a specific objective of site management.
3. As such plans are often used by other stakeholders (*e.g.* land users, and local and regional decision makers) in addition to wetland managers, it ensures that disease management is brought to their attention.
4. As management plans are not static documents they allow for updating in response to changes in the risk of disease and lessons learned. This can then inform the review of risk assessments, contingency plans and disease surveillance activities.
5. A single integrated document is useful for informing specific 'problem disease' contingency plans.
6. Integrating disease management within the management plan reduces the likelihood of new activities being incorporated which are at odds with disease control objectives.
7. As such plans are used to inform budgetary requirements for a site, incorporation of disease management objectives increases the likelihood that these activities will be routinely funded.
8. As such plans are used to inform personnel workplans for a site, to incorporating disease management increases the likelihood that the required activities will be routinely scheduled into work planning.
9. As such plans are used to inform training requirements for a site, incorporation of disease management increases the likelihood of investment in building capacity and maintaining appropriate expertise.

Figure 3-5. Wetlands provide the interface for wildlife and domestic stock: managing the diseases of both should form part of an integrated site management plan (*Sally MacKenzie*).

How to integrate disease management into management plans

When integrating disease management into wetland management plans, the following practical aspects should be included:

What: Ensure the disease management objectives are clearly defined (*e.g.* prevention of specific diseases, or maintenance of status of particular diseases). The management plan should specifically describe those diseases of known concern or with potential for emergence. It is also important to specify which activities should be avoided or amended if the disease management objectives are to be met.

Who: Within the management plan, ensure it is clear who is responsible for each disease management activity, both in terms of project management and implementation. Also, it is important to highlight which stakeholders are involved in activities with key roles to play in disease prevention and control (*e.g.* farmers with livestock using the wetland). The authorities involved in disease control should also be identified within the management plan.

How: The management plan should describe the specific disease management practices required. The logistics and practicalities of their implementation should be explicit or sources of this information should be provided.

When: The timing of disease management activities should be described, both in terms of when to be implemented and their duration. For example, specific disease management activities may be required to coincide with seasonal use of the wetland by domestic livestock or migratory wild animals, or in response to 'seasonal' diseases. Similarly it should be explicit when to cease or reduce other activities which might have a negative impact on disease prevention or control. For example, during periods where there is a high risk of disease outbreak, anthropogenic stressors should be reduced or restricted to less sensitive areas of a site. Consideration should be given to when extra vigilance for disease is required.

CASE STUDY 3-2. Managing avian botulism at Wildfowl & Wetlands Trust reserves in the UK

The nine UK WWT wetland reserves are sites managed for their biodiversity and raising awareness of wetlands and their value with the visiting public. Following a number of outbreaks of avian botulism at two WWT wetland reserves the following activities were integrated into management plans.

Staff awareness and training

The outbreaks are seasonal in nature (in response to factors including hot weather) hence a training presentation is provided to all grounds staff (*i.e.* those working in and managing the wetland sites), one month prior to the highest risk period in the (northern hemisphere temperate) summer. Training includes information about the disease, recognising disease signs in the field, principles of disease control and the annual action plan. All appropriate staff with a role to play in the prevention and/or control of outbreaks are, therefore, aware of the actions to be taken and their responsibility for their implementation.

Figure 3-6. Paralysis of the neck muscles is a common sign of avian botulism and results in an inability to hold the head erect. (*USGS Field Manual of Wildlife Diseases*).

Summary of management actions

During the next eight weeks (or whatever period is considered appropriate *i.e.* 'high risk') staff should:

1. Prevent environmental conditions that can lead to an outbreak
   - Keep water levels stable.
   - Maintain sufficient flow of water/oxygenation.
   - Wherever possible prevent addition of organic matter to water bodies.

2. Break carcase-maggot cycle by immediate removal of carcases.
   - Thorough daily searches to collect carcases.
   - Collection of maggots associated with carcases.
   - Daily search for sick/dead birds and other animals.

- Cut back vegetation at the water's edge to aid searches.
- Document findings: dates, species, numbers, location, *etc.*
- Searches to begin two weeks before previous outbreak date.

3. If an outbreak occurs be prepared to remove birds/scare birds away from the site.

Specific details of management actions at one wetland site - WWT Slimbridge, Gloucestershire, UK

1. Environmental factors
   - Maintain water pump in 'South Lake' (area of high risk and previous disease outbreak).
   - Keep high volume of water moving through the 'South Lake' (replace in-flow pipe with one of larger diameter).
   - Efforts to be made to keep water levels stable in 'South Lake' area.
   - The pipe bringing water from the canal to the 'Swan Lake' to be continued to be kept clear, including regular clearing of grids at either end.
   - A portable oxygenating pump to be made available for problem areas.
   - Care to be taken when strimming/cutting vegetation to prevent organic matter entering water bodies.
   - Wherever possible, care to be taken to eliminate excess organic input to water.

2. Carcase and maggot removal
   - Vegetation at water's edge will be strimmed/cut to allow easier searches for sick and dead animals.
   - Active searches for carcases of all species (including fish) to begin immediately, with extra searches in priority areas. Searches to be done early in the morning to reduce effects of the disturbance on visitors. Frequency to be increased in the face of an outbreak.
   - All grounds staff and volunteers to be extremely vigilant – looking for any birds showing early stages of paralysis, obviously sick birds and carcases.
   - Sick birds to be reported immediately to staff capable of catching birds.
   - All grounds staff to carry basic equipment *e.g.* plastic bags with them for picking up any carcases immediately.
   - Double bagging to collect carcases (a single bag can be knotted, inverted and knotted again to create double bag).
   - Recording: details of species, ring number and location of sick and dead birds to be recorded. Bags containing carcases, maggots and substrate containing maggots to be put into freezer to kill maggots.
   - Reserve Manager to ensure canoes are available for use in carcase searches.
   - Consideration given to scaring techniques in case birds need to be scared from specific sites.
   - If the need arises, one half of isolation area to be set up to as a hospital unit for sick birds. Animal Health Officer to ensure supplies available for treating birds.

Case study from the Wildfowl & Wetlands Trust, UK

Further information and sources

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3.1.4 Contingency planning

Project and site managers should develop site specific contingency plans to address disease problems that could arise at any time. The aim is to consider possible emergency disease scenarios and to integrate rapid cost effective response actions that allow outbreaks to be controlled and prevented in the future. Contingency plans should be considered, 'bought into' and agreed upon by all major stakeholders, and have appropriate resources and legislative backing where necessary.

It is important that these plans are practiced in 'peacetime' (*i.e.* before disease problems occur) both as desktop exercises and also in a practical sense so site personnel are familiar and experienced at response activities. Regular simulation exercises will also serve to highlight any modifications required in contingency plans where aspects are subject to change such as incorporating new staff, new emerging disease threats and legislation and regulations [**►Section 3.5.2 Building capacity by education and training**]. Plans should include clear objectives and guidelines and be written in language that is understandable to all relevant stakeholders. Above all, plans should provide sufficient information to allow the relevant authorities and managers to make informed decisions on appropriate policies and measures used to control a disease outbreak.

It is advisable to develop contingency plans for specific high-risk/high priority diseases which incorporate generic standard operating procedures that may be common to several different specific plans. These should be supported by additional financial and resource plans and supportive legislation to ensure enforcement of contingency plans when needed. Ethical issues should also be considered when creating a plan. The following contingency plan structure is recommended by the Food and Agricultural Organization of the United Nations (FAO).

Technical contingency plan

Specific disease contingency plans detail the management measures that should follow detection of an outbreak in order to control spread. These documents are likely to need to make reference to generic operating procedures for activities and programmes that may be common to several or all disease management strategies, such as modes of internal and external communication and organised public awareness campaigns [**►Section 3.5 Communication, education, participation and awarness**], plans for compensation for wetland stakeholders affected by disease control activities and biosecurity measures [**►Section 3.2.4 Biosecurity**], and methods for surveillance and monitoring [**►Section 3.3.1 Surveillance and monitoring**]. Reference may also be made to manuals that provide zoosanitary guidelines for enterprises deemed at risk of a disease outbreak (*e.g.* areas where animals congregate such as wildlife parks, poultry or other farming around and within wetlands, markets, and fisheries). The contingency plan should clearly identify assigned roles and responsibilities of personnel taking part in the response to a disease outbreak.

A contingency plan should be developed for each of the diseases that have been identified as being of high risk in a particular wetland site [**►Section 3.3.2 Identifying a disease problem**]. Although the format and content of a contingency plan should be tailored to suit the needs of each site and disease, the following factors should be considered for inclusion in the plan:

1. Nature of the disease

- Aetiology (cause).
- Susceptible domestic and wildlife animal species.
- Distribution and history of occurrences in the country and wetlands.
- Epidemiology (including likely pathways for introduction and transmission). An epidemiological investigation will help determine the impact of a disease and understand the infection risks to others and the environment. Outcomes will help determine the extent of infected areas/zones and guide disease prevention and control measures in each area/zone.
- Clinical signs and pathology.

2. Risk assessment

►Section 3.1.1 Risk assessment

- Risk 'profile' of the disease.
- Likely methods of introduction and transmission and defined areas at high risk.
- Potential consequences for people, wildlife and livestock, including food security and poverty alleviation, production losses, trade losses and public and animal health.

3. Diagnosis and surveillance

►Section 3.3 Detecting, assessing and responding to new disease

- Early warning mechanisms for disease introductions and outbreaks.
- Disease reporting procedures.
- Field and laboratory diagnostic strategies.
- Linkages with national and international reference laboratories.
- Surveillance strategies during different phases of a disease management programme.

4. Principles and standard operating procedures for control and elimination

►Section 2.6 Strategies for managing animal diseases
►Section 3.4 Managing disease

- Methods for preventing and controlling disease spread and eliminating disease from the target area. Detailed instructions for disease control activities should be included where possible.
- Factors that may affect control and elimination.
- Feasibility of control and elimination in the target area.

5. Communication

►Section 3.5 Communication, education, participation and awareness

- Identification of key stakeholders needing to be informed and/or involved in a disease outbreak and control action.
- Means by which these stakeholders can be contacted and engaged.
- Strategy for dealing with specific stakeholder groups such as the media.
- Procedures by which stakeholder groups can be engaged in lessons learnt assessments following an outbreak.

6. Policy and rationale

- Overall policy.
- Zoning policy where appropriate [►**Section 3.2.2 Disease zoning, barriers and buffer zones**].
- Disease prevention, control and elimination strategies and activities in defined areas/zones.
- Alternate disease control and elimination strategies and the general circumstances in which these other options would be used.
- Strategies for dealing with special circumstances (*e.g.* disease in wildlife or feral animals, areas with nomadism, and difficult or relatively inaccessible areas).
- Criteria for proof of disease-free designation.

7. Appendixes/annexes

- Full contact details of personnel needed before, during and after a disease outbreak.
- Full contact details of relevant bodies involved in outbreak responses (*e.g.* local and national veterinary authorities and diagnostic laboratories).
- Full contact details of relevant stakeholders.
- Criteria for defining infected areas and disease control zones.
- Justification for chosen methods through assessments of their cost and benefits.
- Summary of disease control actions in infected areas and other zones.
- Relevant national and international legislations and regulations for the disease if applicable (*e.g.* OIE International Animal Health Code).

Annex 2. Activities which need regulating / implementing with HPAI H5N1 in the UK.

	Visitor Activities					WWT Activities						Access										Biosecurity				
	Hand-feeding birds	Pond dipping	Waterbird ringing demos	Passerine ringing demos	Decoy demos	Non-waterbird ringing	Waterbird ringing including for AI surveillance	Wild bird scaring	Entering water bodies	Dead bird surveillance patrols/vigilance	Pest control	Media entry to bird areas at WWT centres	Contractor access to WWT centres	Feed lorry access to WWT centres	Tenant access to WWT reserves	Staff access to WWT reserves	Non-groundstaff access to grounds	Bird movements between centres	Staff movements between centres	Staff access to poultry farms	Staff access to zoos and waterfowl collections	Bird arking plan	Bird vaccination plan	CENTRE CLOSURE PLAN	Only essential staff on site	Fieldwork
HPAI H5N1 not known to be in UK																										
HPAI H5N1 on UK mainland, but:						INC	INC			↑		C						•*		•						F
Centre within Wild Bird Monitoring Area	•*	•*	•*	•*	•*	•*	INC			↑		C	INC	INC	INC	INC		•*	E	•	•	I*	D			F
Centre in Surveillance Zone/WBCA	•	•	•	•	•	•	INC	•		↑	INC	C	INC	INC	INC	INC	•	•	E	•	•	I*	I*	I*		•
Centre suspected/confirmed Infected Premise or H5N1 in wild bird at centre	•	•	•	•	•	•	INC	•	•	↑	•	•	INC	INC	INC	INC	•	•	E	•	•	I	I	I*	•	•

Key: • – Activity ceased; •* – Activity ceased if AITAG consider necessary; INC – activity continues with increased PPE and disinfection; INC – only emergency access with increased PPE and disinfection; ↑ - increase, C – no access to captive or wild bird areas but asses on a case-by-case basis; E – essential staff only; D – depends on advice from Defra; I – implement plan; I* - implementation of plan dependent on AITAG response, F – depends on AITAG approval, WBCA – Wild Bird Control Area.

Figure 3-7. An example annex from a contingency plan for avian influenza at a Ramsar Site. The plan includes activities to be initiated or stopped (columns) according to level of risk (rows) ranging from absence of known disease (white) to outbreak at the site (black).

Contingency support plans

Support plans are vital for ensuring that sufficient money, resources and legislative instruments are available to make the implementation of disease contingency plans possible. Although they can be specific for each region or country, they should contain broadly similar components.

1. Financial plans

Ensuring that you have sufficient funds to finance your disease management strategy is of critical importance as any delay in obtaining finances will hinder the speed of response to an emergency disease outbreak. The rapid implementation of disease control activities will ultimately reduce the overall cost of the disease control campaign.

Financial plans must include the immediate provision of funds to respond quickly to an outbreak but also provide for other phases of the disease management strategy, where necessary. Where appropriate, funds should be approved at governmental level. Criteria should be agreed for the release of funds, for example, when an outbreak has been identified or the presence of disease is strongly suspected, when effective control and/or elimination of the disease is possible and when there are approved plans to implement such measures.

If the funding and resources of a disease management strategy is limited in a country or area, potential international donor sources should be identified (*e.g.* support from the FAO or appropriate international agencies). It may be wise to include procedures for applying for funding from various 'back-up' sources in the financial plan. If possible, funds for compensation of wetland stakeholders who have incurred financial losses as a result of disease control activities should also be included where this is national policy.

2. Resource plans

It is important to make an inventory which lists all the resources that will be needed during a disease outbreak, including capacity of personnel (their qualifications, expertise and experience) and equipment (quantities, specifications and locations). This should be compared with an inventory list of existing resources and any deficiencies should be rectified. Resources for each stage of a disease outbreak should be incorporated into plans.

All staff should be thoroughly trained in their roles, duties and responsibilities, and a contingency plan should allow for 'back-up' staff [**►Section 3.5 Communication, education, participation and awareness**].

Legislation

A contingency plan should include information on legislation and regulations that may or may not give permissions to conduct various disease prevention and control activities, in the event of an outbreak at or around your site. This should include information about the compulsory notification of certain animal diseases and may also include authorisations for the declaration of infected areas and disease control zones, movements of animals and people, the destruction and safe disposal of infected or potentially infected animals and objects, compensation for those financially affected by disease control activities and authorisation for any other relevant activities.

Simulation exercises

It is important to ensure that your contingency plan is practically achievable and for this, simulation exercises should be carried out in advance of their implementation. Lessons learnt from such exercises should be used to further refine and improve your contingency plan. These exercises are essential for building effective teams, ensuring that there are adequate resources and for training staff [**►Section 3.5.2 Building capacity by education and training**].

Disease outbreak scenarios should be realistic and real data should be used if possible. Each stage of an outbreak response may need to be tested before a full-scale disease scenario is attempted.

Reviewing and refining your contingency plan

Above all, contingency plans should be working documents that are subject to periodic review. Changing circumstances may require that a contingency plan be updated to retain its effectiveness in preventing and controlling disease. The effectiveness of a contingency plan in preventing and/or controlling a disease in a wetland should be thoroughly evaluated after a disease outbreak response has ended, and recommendations for improvement should be incorporated where necessary.

Further information and sources

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3.2 Reducing risk of disease emergence

An understanding of patterns of the use of a wetland by people, livestock and wildlife, coupled with an appreciation of risk factors for disease emergence, can provide a sound foundation for disease risk management. Disease surveillance and monitoring [▶**Section 3.3.1**] may identify diseases of particular concern for the wetland. The specific actions required to reduce risks associated with these diseases should be identified within risk assessments [▶**Section 3.1.1**] and included in wetland management plans [▶**Section 3.1.4**]. More generally, 'healthy habitat management' and reducing stressors at a site will benefit disease prevention and/or control [▶**Section 3.2.1**]. Additionally, following standardised protocols for releasing and moving animals into, within and out of wetlands will help to mitigate disease risks [▶**Section 3.2.3**] as will the consistent application of sensible precautionary biosecurity measures [▶**Section 3.2.4**].

This section contains further information on the following topics:

- *Reducing stressors*
- *Disease zoning, barriers and buffer zones*
- *Standards for releasing and moving animals*
- *Biosecurity*

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- An **understanding by the wetland manager of the uses** of a wetland and its catchment by people, industry, agriculture including livestock, and wildlife, coupled with an appreciation of risk factors for disease emergence, can **provide a sound foundation for disease risk reduction**.
- It is **important that wetland managers identify stressor risks** within their site and the broader catchment/landscape, and understand that these may change over time. Once these factors are identified, **they can be managed and/or their impact mitigated,** as appropriate.
- **Disease zoning** (although challenging in wildlife and/or aquatic systems) **can help control some infectious diseases** through the delineation of infected and uninfected zones defined by sub-populations with different disease status. Buffer zones separating infected and uninfected zones may consist of physical barriers, an absence of hosts, an absence of disease vectors or only immune hosts *e.g*. following ring vaccination. **Appropriate levels of surveillance are required** to accurately define zones and for prevention of disease spread to occur, the movements of animals between zones needs to be restricted.
- **The movement of infected animals to new areas and populations represents the most obvious potential route for introduction of new/novel infections**. The risk of transmission and spread of disease can be **minimised by conducting risk assessments and following certain standardised national and international guidelines and regulations** for moving, relocating and/or releasing animals. A disease risk analysis should be conducted for any translocations for conservation purposes.
- **Biosecurity** in wetlands refers to the **precautions taken to minimise the risk of introducing infection (or invasive alien species)** to a previously uninfected site and, therefore, preventing further spread. Infectious animal diseases are spread not only through movement of infected hosts but also their products *e.g*. faeces, saliva *etc*. or *via* human and fomite (inanimate object) contact with animals and their products. **Constructed treatment wetlands can assist greatly in reducing risks** from contaminated wastewaters.
- Where possible, **biosecurity measures should be implemented routinely** as standard practice whether or not an outbreak has been detected. A regional/supra-national approach to biosecurity is important for trans-boundary diseases, particularly those where domestic and international trade are considered as important pathways for disease spread, *e.g*. trans-boundary aquatic animal diseases.
- **If wetland stakeholders understand the principles and value of biosecurity** and what measures to take, this will **encourage the development of an everyday 'culture' of biosecurity** which can help disease prevention and control.
- **Implementing biosecurity measures in the natural environment can be extremely challenging**, particularly in aquatic systems, and although eliminating risk will be impossible, **a substantial reduction in risk may be achievable**, particularly where several complementary measures are employed.

3.2.1 Reducing stressors

Stress in its various forms can affect the ability of the immune system to protect the host from infection and disease. Stressors may not in themselves cause disease but their effects can be subtle and can influence disease dynamics and the likelihood of a disease outbreak. Stressors can be additive or synergistic, working together to shift the balance between health and disease within individual hosts or populations.

Consequently, stressors at wetland sites should be identified and managed to reduce disease susceptibility. Identification of potential stressors requires a thorough knowledge of the site and a reasonable understanding of the biology and ecology of the animal species present. It is important to periodically re-assess the stressors at a given site as they may change over time.

Common stressors

Toxins: environmental pollution (*e.g.* heavy metals and pesticides) may become concentrated in certain areas with negative impacts on vertebrate physiology and immune function. Mitigation measures need to focus on eliminating or reducing such effects.

Nutrition: malnutrition (deficiency, excess or imbalance of nutrients) of animals may result in increased disease susceptibility. Consideration can be given to providing supplementary high quality food and/or water, although artificial provisioning brings its own disease risks (*e.g.* concentrating wildlife at 'unnaturally' high population densities and hence increasing opportunities for transmission of infection).

Human disturbance: ideally this should be reduced/kept to a minimum where possible, especially at sensitive times in the life cycles of wildlife, at times when other stressors are known to occur or when risks of disease outbreaks are high. Consideration could be given to closing public footpaths/access during key times. Zoning human activities such as recreation and agriculture may also be of value in managing human disturbance. Herding and capturing animals (*e.g.* chemical immobilisation, corralling, netting, holding, transport, and restraint and sampling) are generally considered acutely stressful activities, so careful planning and preparation, and the use of established protocols and well trained teams are essential.

Predators: depending on the management priorities of a site, measures could be considered to minimise stress from predators (*e.g.* by methods of deterrence).

Interspecific and intraspecific competition: depending on the management priorities of a site, measures could be considered to reduce competition from other animals (*e.g.* by controlling stocking density to reduce psychological and nutritional stress).

Con-current disease: if it is known (*e.g.* from surveillance activities) that significant con-current disease is present, particular attention should be given to reducing other stressors.

Extreme weather and other environmental perturbations: during periods of extreme potential stress (*e.g.* extreme hot or cold weather, drought, flood) other stressors should be kept to a minimum to help to reduce the likelihood of disease outbreaks. For example, a voluntary ban on shooting activities during extended periods of cold weather may be advisable. Such actions need to be the subject of advance agreement amongst site managers and other stakeholders.

CASE STUDY 3-3. Nutritional and other stressors? Common Eider *Somateria mollissima* mortality in the spring and winter of 1999/2000 in the Wadden Sea.

Although debatable, there is evidence to suggest the 1999/2000 mass mortality of common eider ducks in the Wadden Sea was due to nutritional stress and simultaneous heavy parasite loads. It has been suggested that the eiders suffered starvation resulting from poor foraging conditions linked to over exploitation of mussels by the commercial industry. This disrupted food intake combined with parasite loads two to three times higher than apparently healthy eiders may have led to compromised body condition and function.

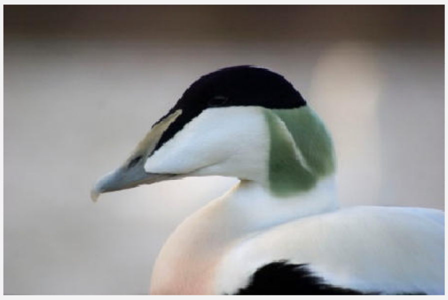

Figure 3-8. Common eider (*WWT*)

One explanation for the elevated parasite loads could be derived from the shore crabs which the eiders were apparently 'forced' to prey upon given the scarcity of mussels. Shore crabs harbour multiple parasites and, therefore, present higher risk of infection to eiders. Although in this case the high parasite loads were not directly correlated with poor body condition they may have contributed as an accelerating or secondary factor. Parasitic infections may have increased energetic costs for eiders and enhanced their susceptibility to other stressors such as concurrent nutritional disease and environmental conditions.

Sources: Blomert & Reinekeg 2001 and Christensen 2008

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3.2.2 Disease zoning, barriers and buffer zones

Disease zoning

Disease zoning can be used to control infectious diseases by delineating infected and uninfected zones, and allowing measures to be implemented to prevent the spread of infection between them. An infected disease zone is an area or local population in which disease has been detected. Zoning may be particularly useful where disease elimination is not feasible [►**Section 3.2.4 Biosecurity**].

Buffers and barriers

A buffer zone is an area of uninfected status (under surveillance) which surrounds the infected zone. Its purpose is to facilitate prevention of disease spread into an uninfected sub-population.

The buffer zone may be identified on the basis of:

- an absence of hosts
- an absence of disease vectors
- only immune hosts (*e.g.* following ring vaccination).

An effective buffer zone may take the form of a geographical, hydrological or climatic barrier. These barriers may be natural such as rivers and lakes (for terrestrial hosts) or terrestrial habitat (for aquatic hosts), or unnatural features in the landscape such as roads, fences or cleared habitat. Such barriers have been shown to be effective in control of disease by either slowing or preventing spread. Complications are introduced when dealing with highly mobile hosts (*e.g.* migratory, semi-migratory or nomadic animals), and where the epidemiology of a disease (particularly the identification of reservoir species) is poorly understood.

Artificial barriers can also be used to inhibit movements of hosts but can themselves have adverse ecological consequences, such as the prevention of movements of wild animals caused by foot and mouth disease fences in parts of southern Africa.

Specific considerations for water-borne diseases

Within wetlands, zoning for the control of water-borne diseases is particularly challenging but may still be a useful approach. Wetland zones may be defined by catchment areas and rivers and coastal zones. The simplest zone is that of an area that derives its incoming water from an unshared source and thus may continue to function independently of any infected areas. In the instance of an inland area that shares common water sources, the minimum zone would apply to the entire catchment area. Larger catchment areas may require multi-national and transboundary cooperation and jurisdictions as disease management relies on all aspects of the water catchment zone being managed accordingly.

Surveillance for defining zones

The definition of 'infected' and 'uninfected' zones relies on adequate surveillance and the effective use of zones for disease control relies on an understanding of modes of transmission. Restrictions on domestic and international trade of animals and derived products, may apply to infected zones. Continued surveillance is needed to confirm the absence of infection in uninfected areas.

Movement of animals between zones

Conditions applying to the movement of animals (either domestic or translocated wildlife) between zones should be comprehensively described in a zoned management strategy. Conditions should also apply to movement of other materials which could facilitate mechanical transfer (*e.g.* slurry, bedding substrates, other fomites or animal products).

Examples of barriers and buffer zones

Foot and Mouth Disease: Several countries including Botswana and Zimbabwe have implemented effective disease control strategies which include dividing the country into risk zones. These zones are managed by means of appropriate disease surveillance, movement restrictions, livestock identification and vaccination. Ring vaccination may be required as an emergency measure for animals within a certain radius of a confirmed outbreak.

Anthrax: Following an outbreak in cattle a buffer zone of a specified width can be established around infected areas. All animals inside this area which have been exposed can then be vaccinated and quarantined.

Further information and sources

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3.2.3 Standards for releasing and moving domestic animals or translocating wildlife

Movements of wildlife and domestic animals may facilitate the spread of disease by introduction of pathogens to formerly disease-free areas. In order to control disease spread, it is therefore crucial to understand movement patterns of potential disease hosts at a national and international level and the associated disease risks. The risk of transmission and spread of disease can be minimised by following certain guidelines for releasing and moving animals. Such measures should be supplemented by an efficient surveillance network involving the health screening of animals, particularly when they are to be moved to another area. Given the global scale of animal movements in wildlife populations and the livestock and pet trades, international cooperation in maintaining standards of moving and releasing animals is vital in preventing and controlling disease spread and reducing the risk of outbreaks.

Legislation and regulations

National and international legislation and regulations are in place to control the movement of animals, although disease outbreaks still occur regularly as a result of both legal and illegal movements. It is, therefore, important to familiarise yourself with legislation and regulations and their enforcing regulatory bodies, where they relate to not only a wetland site, but also to the exporting country, the transit country and the importing country [►**Section 3.1 International regulations and standards**].

Certification requirements for moving animals should also be fulfilled and should clearly outline the wishes of the importing country. For this, prior consultation between veterinary authorities of importing and exporting countries may be needed. Special permits or licences may be required for moving and/or releasing wildlife. For example, CITES-listed species will require licences for movements of the whole animal, or derivatives thereof, across trans-national boundaries of signatory countries.

INTERNATIONAL REGULATIONS AND STANDARDS 3-1. Movement and trade.

The following international organisations and regulatory bodies are concerned with the movement and trade of animals and may be able to provide further guidance.

International organisations

- Food and Agriculture Organization (FAO): www.fao.org
- World Health Organisation (WHO): www.who.int
- World Organisation for Animal Health (OIE): www.oie.int
- World Trade Organisation (WTO): www.wto.int
- African Union-Inter African Bureau for Animal Research: www.au-ibar.org
- Organization for Economic Co-operation and Development: www.oecd.org

International regulatory bodies

- Convention on International Trade in Endangered Species of Wild Fauna and Flora: www.cites.org
- World Conservation Union: www.iucn.org
- Convention on Biological Diversity: www.biodiv.org
- International Air Transport Association: www.iata.org
- Council of Europe: www.coe.int
- European Union: europa.eu

TABLE 3-1. Legislation, regulations and guidance relevant to the trade and movement of domestic and wild animals (from Fèvrea *et al.* 2006).

Level	Animal health	Animal welfare	Endangered species
International	World Trade Organisation and Sanitary and Phytosanitary Agreement International Animal Health Code and International Aquatic Animal Health Code (World Organisation for Animal Health – OIE)	International Air Transport Association regulations International Animal Health Code and International Aquatic Animal Health Code (World Organisation for Animal Health – OIE)	Convention on the International Trade in Endangered Species (CITES) Convention on Biological Diversity (CBD) IUCN guidelines
Regional	European Union directives (numerous)	European Union Regulation (transport of animals) Council of Europe Convention (transport of animals)	European Union and CITES regulations
National	Laws on control of disease and movement	Anti-cruelty laws, welfare codes	Laws implementing CITES and CBD, species protection
Sub-national	Local restrictions on animal movement		

Source: Fèvrea et al. 2006

Protocols for relocating animals

Prior to any relocation of wild animals a risk assessment should be conducted [►**Section 3.1.1 Risk assessment**]. Information should be available from government agencies, as well as other sources, to help inform the risk assessment and protocols for relocation. The latter may include:

1. Thorough examination and **health screening** of animals prior to their relocation and routine surveillance and monitoring of animals for the early detection of disease [►**Section 3.3.1 Surveillance and monitoring**].

2. **Movement restrictions** for diseased/susceptible animals to prevent the spread of infection. This may include **quarantine** of animals before their release to ensure that they are disease-free. Once animals have been moved to a new area, a routine 'standstill' period may also apply, preventing the movement of certain animals on and off that site for a specified number of days [►**Section 3.4.7 Movement restrictions**].
3. **Methods to protect** animals to be translocated from exposure to infection at their destination (*e.g.* through vaccination).
4. Methods to ensure **animal welfare** during transportation. Animals must be moved in a way that will not cause them injury or unnecessary suffering and additional stress that may affect their health. When transporting animals:
    a. plan journeys well and keep the duration to a minimum
    b. ensure that animals are fit to travel and check them frequently
    c. ensure vehicles are designed to avoid injury and suffering
    d. handlers should be experienced and competent and understand the behaviour patterns of animals
    e. provide sufficient floor space and height
    f. provide water, feed and rest as needed.
    g. It is advisable not to transport animals that are considered unfit to travel, and it is illegal to do so in many countries. This includes individuals which are sick or injured, newly-born, heavily pregnant or have recently given birth.
    h. It is important to **avoid mixing of animals from different sources.**

5. Methods for **recording animal movements** which will make it easier to trace and identify infected animals in the event of a disease outbreak.

In some countries, it is a legal requirement that livestock keepers retain individual records and notify authorities of livestock movements, births and deaths. Animals can be individually identified by a variety of methods (*e.g.* ringing, tagging, micro-chipping). Government agencies may visit premises or require records be sent to them directly. Licenses may be used to record the movements of livestock (*e.g.* where animals are kept, where they originated and, if appropriate, the final destination) which in turn are logged onto a national database that records and monitors the movement of all animals across a country. In the event of a disease outbreak such as foot and mouth disease or avian influenza, movement records will inform the investigation and so it is vital they are accurate and up to date.

Tools for recording animal movements may significantly improve the effectiveness of the management of disease outbreaks and food safety incidents, vaccination and animal medication programmes, animal husbandry, zoning, surveillance, early response and notification systems, animal movement controls, and animal inspection and certification.

Most importantly, **follow guidelines** as outlined in the relevant regulations and legislation to ensure that standards for releasing and moving animals are effective and maintained. Further information can be found in the OIE's Terrestrial Animal Health Code (2010).

Specific considerations for conservation translocations

All of the above guidance is applicable to conservation translocations including reintroductions.

Every translocation project should be accompanied by a comprehensive disease risk analysis [►**Section 3.1.1 Risk assessment**] aimed at summarising and managing the risks to wild source populations, captive populations, the released population and any species present in the release area or likely to encounter the released population in other areas.

Temporarily captive or captive-reared animals involved in conservation translocations may be particularly vulnerable to disease due to the stresses of both captivity and transport, and due to reduced genetic diversity often found in threatened species, and captive populations thereof. Thus, extra care must be taken to reduce stressors throughout any translocation [►**Section 3.2.1 Reducing stressors**].

The range of diseases to screen for and manage will be outlined in the disease risk analysis. The soft release technique of temporarily holding released animals within a release enclosure allows a period of time in which released animals can acclimatise to the new environment and endemic diseases (to some extent), and provides a period of time, during which, veterinary intervention can be given, if necessary.

The risks of disease translocation together with the logistical and administrative aspects, and potential for delays, may provide sufficient reason to attempt to rear animals *in situ* within natural disease range and within country of origin.

Figure 3-9. A release enclosure for the UK's Great Crane Project: in addition to standardised health monitoring, this soft release measure allows a period of time during which birds can encounter some 'endemic' diseases and allows veterinary intervention if necessary (*WWT*).

Further information and sources

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3.2.4 Biosecurity

Biosecurity refers to the precautions taken to minimise the risk of introducing infection to a previously uninfected site and, therefore, preventing further spread. In the context of this guidance it refers to measures taken to reduce the likelihood of introducing infection into a wetland. Infectious animal pathogens are usually spread through movement of infected hosts or their products (*e.g.* faeces, saliva *etc.*) or *via* human and fomite contact with animals and their products.

Robust surveillance [►**Section 3.3.1 Surveillance and monitoring**] can provide early warning of emerging risks and inform the level of biosecurity required. Routine cleansing and disinfection regimes [►**Section 3.4.1 Disinfection and sanitation**] can minimise opportunities for the introduction and spread of diseases *via* infected animals or contaminated objects such as clothes, boots, vehicles, equipment and water.

Biosecurity measures should be implemented routinely as standard practice whether or not an outbreak has been detected. However, the stringency of biosecurity measures may be altered in response to changes in the perceived level of risk. A regional/supra-national approach to biosecurity is important for trans-boundary diseases, particularly those where domestic and international trade are considered to be important pathways for disease spread.

Biosecurity in 'wild' settings can in some circumstances seem impossible to attain; although the *elimination* of risk is unlikely to be attainable, *reduction* of risk may be sufficient to make a significant contribution to disease control.

It is important that wetland stakeholders understand the principles and value of biosecurity. Developing a 'culture' of biosecurity in managed wetlands can make a substantial contribution to disease control.

General biosecurity measures

Wetland managers should try to ensure that the movement and/or introduction of livestock, people, vehicles or equipment into wetland areas is minimised or at least controlled, particularly so during periods of increased risk. Attention should also be focused on hazardous/high risk substances such as slurry and faecal-contaminated materials.

Information on the diseases present within a wetland and its surrounding area, and the routes by which these are spread, will help to dictate the level of risk and, therefore, the biosecurity required. Ideally, when entering and leaving a wetland area (within reason), vehicles, equipment, and protective footwear and clothes should be cleaned and disinfected [►**Section 3.4.1 Disinfection and sanitation**]. This is particularly important for those items in contact with animals and their products. Where appropriate or possible, footwear and equipment should also be disinfected before being used again on a different part of the wetland site. Facilities for disinfection should be available on entry to and exit from the area.

In some circumstances it may be appropriate for protective clothing and footwear to be worn (*e.g.* rubber boots and gloves). Items must be easily cleaned (*e.g.* waterproof clothing and boots) or disposable. If practicable, equipment should be protected with plastic bags. Where possible, vehicles should be parked on hard standing ground, away from animals, and kept visibly free of mud, slurry and animal products.

Other means by which infection risk can be reduced involve: 'resting' domestic animal holdings to allow a period of time in which contaminated materials can decompose; and reducing stocking density to reduce likelihood of disease transmission.

Artificial water supplies for domestic animals (*e.g.* cattle in enclosed areas and fish and shellfish in aquaculture) should be clean and waste effectively disposed of where possible, through efficient treatment systems. New domestic animals should be quarantined before being introduced to a wetland area. Where possible, domestic animals should be sourced from specific pathogen-free certified stock or following pre-movement testing.

During an outbreak of infectious disease, only essential persons should visit areas with infected animals and they should adhere to appropriate biosecurity measures. Non-essential visits, including public access, should be suspended at such times.

Wetland treatment systems

Both natural wetlands and specifically designed constructed wetlands, can play an important role in sanitation and treating wastewater, sewage and run-off. They function through a combination of physical, chemical, and biological processes, reducing pathogenic agents such as helminth eggs, bacteria, viruses, and heavy metals, as well as removing and storing nutrients. As such, they can provide a sustainable, and highly effective, means by which to reduce risks from both point-source and diffuse contaminated wastes. If designed and managed correctly, as well as treating wastewater, they can also provide additional benefits in terms of maximising biodiversity, providing stormwater and floodwater detention, and providing livelihoods. It should be recognised that if using an area of natural wetland for treating waste, this designated area must be monitored and managed appropriately to ensure no detriment to the wider wetland environment.

Expert guidance should be sought to ensure the wetland type is fit for the waste treatment purpose required, as effectiveness of such wetlands to treat contaminated wastewater, will depend on a number of factors including:

- Plant and substrate type.
- Type of wetland or constructed wetland (whether it is a surface flow wetland or a constructed sub-surface flow wetland as the latter is more efficient at pollutant removal per m^2).
- Hydrological regime (including the wetland water balance as wastewater needs to remain within the wetland for sufficient time to allow 'cleansing' processes to occur).
- Wetland background water quality (the ability of the wetland to treat wastewater will be dependent on the existing water quality).
- Area and depth of wetland (generally the larger the wetland the more treatment it can provide but if the wastewater flows directly through the wetland rather than spreading across the wetland then even a large wetland may not provide total treatment).
- Climate (higher temperatures and UV radiation levels provide more treatment but even in cold temperatures wetland treatment systems can be effective).

- The volume of wastewater and type and concentration of pollutants and suspended solids within the wastewater input (high concentrations may 'overload' the capability of the wetland).
- Management of the wetland (poor management will reduce the capacity to treat wastewater).

Figure 3-10. Constructed treatment wetlands are an effective means of treating contaminated wastewaters (*Martin Senior*).

Further information and sources

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CASE STUDY 3-4. Protocol to protect amphibian populations from chytridiomycosis by disinfection of workers and equipment on WWT reserves.

Within the UK, chytrid fungus *Batrachochytridium dendrobatidis* has now been found in a number of separate locations.

The following protocol applies to WWT sites.

Figure 3-11. Natterjack toad *Epidalea calamita*: biosecurity protocols are being followed at wetland sites to reduce risk of spread of chytrid fungus (*WWT*).

1. Disinfection of work-related equipment and footwear

To prevent infection being introduced to a WWT site, people* (staff, students, contractors, *etc*.) who will be coming into contact with water or amphibians on a wetland site, *e.g.* during surveying, must first disinfect (see disinfection protocol below) both footwear and sampling equipment (*e.g.* footwear, boats and nets) if they have been previously used at another site.

To prevent infection being carried from one WWT site to another wetland site (including WWT sites), the same disinfection protocol (below) should be followed for footwear and equipment coming into contact with water or amphibians.

Where possible footwear and equipment should be disinfected before being used again on a different part of the reserve.

2. Disinfection of personal equipment and footwear

Members of staff that use their own hiking boots *etc.* at work must take care if they have either entered any water bodies or travelled to high risk areas such as the Lake District or Yorkshire (infected sites) on leisure trips. Boots should be fully disinfected (below) before being used back on a WWT reserve.

Ideally staff should try to use one set of footwear for the site on which they work and have a separate set for use at home or on other sites.

3. Animal release and movement

There is a standard protocol not to accept or release amphibians/spawn or pond plants onto sites and those currently on our sites should not be moved elsewhere.

DISINFECTION PROTOCOL

To properly clean footwear and equipment:

- First use a brush to clean off organic material (*e.g.* mud and grass).
- Rinse with clean water.
- Soak in fungicidal disinfectant for one minute.
- Rinse with clean water and allow to dry. Drying thoroughly is important and will act to kill any chytrid fungus present.
- If any clothing is particularly soiled during fieldwork, then washing at 40°C with detergent will be sufficient to remove any contamination with chytrid fungus.

*Only people coming into direct contact with the water or amphibians need to disinfect their boots *etc.* so this does not apply to farmers coming onto land to check stock. Similarly this does not currently apply to staff/visitors in the public areas and on paths.

Further information for fieldworkers is available at: www.arguk.org/advice-and-guidance/view-category

Source: Wildfowl & Wetlands Trust, UK

3.3 Detecting, assessing and responding to new disease

Globalisation and climate change have greatly enhanced opportunities for the emergence and spread of diseases throughout the world, giving rise to serious threats to human and animal health. The mobility of wildlife also increases the potential for infectious disease to spread to new locations and populations. It is, therefore, becoming increasingly important to not only reduce the risk of disease emergence, but to effectively detect, assess and respond to new diseases as they arise. Timely and accurate diagnosis of a disease problem and appropriate actions during the first stages of an outbreak are especially critical to achieving effective disease control and prevention. The importance of enabling rapid responses cannot be over-emphasised.

Disease control would of course not be possible without its detection in the first instance. Disease surveillance and monitoring programmes enable a greater understanding of disease patterns, therefore enhancing capacity to detect and control infectious diseases that may emerge in the future [**►Section 3.3.1 Surveillance and monitoring**]. Such programmes are critical for the 'early warning' of disease presence and the planning and monitoring of disease control programmes thereafter and will be more cost-effective than managing a large scale disease outbreak [**►Section 3.3.2 Identifying a disease problem**].

Surveillance and monitoring for diseases in wildlife in and around wetland areas should be considered, as wildlife are inherently at risk of negative impacts from emerging diseases and can serve as reservoirs of pathogens important to human and livestock health. Given that many diseases that have emerged in recent years have originated in wildlife, surveillance and monitoring of wild animals may be an important tool for the protection of public health, livestock health and the conservation of endangered populations. Therefore, although programmes for surveillance and monitoring diseases in wildlife are less likely to be as well developed as programmes for livestock, they are nevertheless recognised as being of increasing importance. Samples taken during routine and targeted surveillance and monitoring are valuable resources so it is essential that they are stored, preserved and transported appropriately [**►Section 3.3.3 Sample collection and preservation** and **►Section 3.3.4 Sample transport and shipment**].

Following a disease outbreak, it is likely that animal health professionals will conduct an epidemiological investigation to try to determine why the disease has arisen and where it may occur next. The wetland manager will not be responsible for this investigation but has an important role to play in recording as much relevant information as possible during a suspected outbreak [**►Section 3.3.5 What data to collect at a suspected outbreak**] as this represents a unique opportunity to collect potentially valuable epidemiological evidence.

Following the detection of a disease in a wetland, the next challenge may lie in assessing whether or not it constitutes a 'real' problem. Disease is a part of our natural world and may not always pose a risk to people and/or animals. However, measures must be in place to help identify when a particular disease becomes a problem and these are discussed here.

This section contains further information on the following topics:

- *Surveillance and monitoring*
- *Identifying a problem*
- *Sample collection and preservation*
- *Sample transport and shipment*
- *What data to collect at a suspected outbreak*

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- The **detection** of new, emerging disease, robust **risk assessments**, and effective **disease control** in and around wetlands, **all rely on effective disease surveillance and monitoring**. Surveillance programmes should be well designed with clearly defined aims and objectives. Robust surveillance requires appropriate methods for sample collection, recording, storage and transportation, which in turn depend on well trained personnel and adequate resourcing.
- **Timely and accurate diagnoses and early warning systems for disease emergence are critical for swift responses**, achieving effective disease control and minimising losses and costs. Early warning systems may depend on a comprehensive understanding of a wetland site and catchment, good disease intelligence from a range of stakeholders (including crucially the wetland manager, as well as data from local and national disease surveillance programmes), and clear systems and networks for communication and reporting.
- **Identifying** when a disease presents a '**problem**' is complex and **requires** thorough disease investigation and existing **good long term surveillance** information.
- In the event of a suspected outbreak of disease, **wetland managers are not expected to be the final disease diagnostician**. However, they should **play a key role in an outbreak investigation** team being ideally placed to provide the crucial contextual epidemiological information about timing of events, the populations at risk, the effects on these, land use and environmental conditions at the time and leading up to the outbreak, and other relevant local information.

3.3.1 Surveillance and monitoring

The detection of emerging disease, robust risk assessments and effective disease control in and around a wetland area rely on effective disease surveillance and monitoring. Surveillance and monitoring are terms often used interchangeably but surveillance generally refers to observing a population for signs of a disease over time. Monitoring, on the other hand, can be used to refer to measuring disease prevention or control programmes and providing the information to evaluate whether or not interventions have worked or how improvements can be made. Above all, surveillance programmes should aim to evaluate the health status of a group or population and help to prevent or limit the spread of diseases by informing disease control activities.

Surveillance is a continuous and systematic process which involves the collection of relevant data for a specified population, time period and/or geographical area, meaningful analysis of the data and dissemination of the results to appropriate stakeholders. Collected data should include observed clinical signs, diagnostic test results and any associated risk factors identified.

Surveillance and monitoring are vital for:

- establishing base-line data on the health of a population or group
- determining temporal and spatial variation in disease prevalence
- identifying the point at which there is a departure from ‘normality’ and hence the point at which action should be triggered
- detecting disease problems before they have adverse consequences
- predicting future disease outbreaks
- determining the potential role of wildlife in the ecology of the disease
- helping to plan and monitor control programmes if needed.

Information obtained from disease surveillance may need to be communicated to stakeholders representing public and animal health, wildlife conservation and management and environmental management interests. Disease surveillance and monitoring should form an integral part of any disease management strategy.

Importance of wildlife surveillance

Surveillance for wildlife diseases is an important tool for conservation management necessary for assessing risks to wild populations. Wildlife can also be important reservoirs or sources of zoonotic infections (*e.g.* leptospirosis) and diseases affecting domesticated stock (*e.g.* bovine tuberculosis). As humans and their livestock increasingly move into wildlife areas and as wildlife moves into urban areas to exploit novel resource opportunities, the likelihood of contact and spillover of infections from wildlife to humans and domestic animals has increased so enhancing the need and value of wildlife disease surveillance.

Designing a disease surveillance strategy

A disease surveillance strategy should have clearly defined objectives, sound epidemiological justification and should involve appropriately trained personnel with sufficient technical skills to perform both field and laboratory exercises [►**Section 3.5.2 Building capacity by education and training**]. Appropriate human health and biosafety precautions should be followed during surveillance and monitoring activities. Activities should focus on collecting only the information that is needed to achieve the objectives, noting that this information may differ between diseases.

Surveillance may involve collecting various samples from the environment, the health screening of living and dead specimens, remote screening and/or the introduction of sentinels [►**Checklist 3-1**].

CHECKLIST 3-1. Information commonly collected during surveillance activities

Timing

☐ Dates of findings, sampling, results *etc.*

☐ Estimation of timing of any change in health status

Host information

☐ Species involved

☐ Numbers affected

☐ Numbers sampled

☐ Population(s) at risk *i.e.* contextual information about species present at the site

☐ Ages (*e.g.* juvenile/adult)

☐ Sex

☐ Condition (*e.g.* from fat score or biometric measurements)

☐ Clinical signs

☐ Signs of trauma or injury

☐ Additional observations (*e.g.* whether animals seen prior to outbreak appear under-nourished or healthy)

☐ Other contextual information (*e.g.* population movements)

Samples

☐ Sample types taken (*e.g.* whole carcase, whole blood, serum, plasma, faeces, buccal swab)

☐ Storage and transport methods

Environmental information

☐ Location/s

☐ Type of habitat/area

☐ Environmental factors (*e.g.* weather conditions)

☐ Land use and human activities

☐ Specific features of an areas

Additional information

☐ All other relevant case related notes and comments

Laboratory findings

☐ Laboratory results and diagnosis

☐ Laboratory or diagnosticians conducting the work

Personnel

☐ Name and contact information of individuals involved in collecting information

Wherever possible, assessments should be made to ensure that an appropriate portion of a population is examined and that the correct types of data are collected, in order to fulfil the surveillance objectives as defined in the strategy. An accurate assessment is reliant on a thorough understanding of the disease and its lifecycle, notably, transmission [►**Case study 3-5. Surveillance strategies for highly pathogenic avian influenza (HPAI) H5N1**].Therefore, a multi-disciplinary approach to surveillance involving a variety of professionals (*e.g.* wetland stakeholders, human and animal health professionals, epidemiologists, wildlife ecologists, mathematical modellers, geographic information specialists and statisticians) is often most effective. In some cases, reports about sick wildlife from the general public can be the first indication that a larger incident of morbidity and/or mortality is about to occur.

To a large extent, the robustness of a surveillance strategy relies on sampling an appropriately sized sample of the appropriate portion of the population. Skilled animal health personnel will be needed to determine sample sizes although for wildlife the wetland manager is likely to have a *relatively* good understanding of structures of wild populations and thus can help in the design and practicalities of achieving this target sample size.

The problem of bias in surveillance strategies is less of an issue for domestic animals where it can be *relatively* straightforward to sample individuals randomly and in a stratified manner *e.g.* individuals of different ages, sexes, at different times of year *etc.* Surveillance in wildlife, however, can be fraught with problems of bias. All wildlife trapping techniques have their own biases, surveillance from carcases may introduce a range of biases, *e.g.* such strategies are often biased towards larger bodied animals (smaller ones being over-looked or scavenged before retrieval), or there may be other non-random reasons why carcases are found (*e.g.* road-killed animals may not be representative of the population at large but instead a sub-set of perhaps younger, less experienced or diseased individuals less able to remove themselves from danger).

CASE STUDY 3-5. Surveillance strategies for highly pathogenic avian influenza (HPAI) H5N1.

Active surveillance programmes for free-ranging healthy wild birds should be targeted at species with the following characteristics:

- Species known to have been infected in the past with the HPAI H5N1 virus
- Species known to be epidemiological reservoirs for low pathogenic AI viruses
- Social species that are known to aggregate seasonally at breeding, roosting, migration stopover and non-breeding (wintering) sites
- Species that potentially share habitats with poultry farms, integrated livestock-aquaculture systems, backyard poultry flocks and croplands such as rice fields (sometimes called 'bridge' species)
- Species whose seasonal movements or migratory patterns may explain disease dispersal and/or emergence. Selection of sampling sites will primarily be dictated by the habitat preferences of the species to be sampled and occurrence of outbreaks in poultry although other factors such as bird and researcher safety, and project logistics should also be considered.

Source: Food and Agriculture Organisation, 2007.

Between disease cases, there may be differences in:

1. **Surveillance approaches**

   - *Passive or 'scanning' disease surveillance***:** this involves examination of only clinically affected individuals, with no special effort being made to 'seek out' infected or diseased cases. This may involve the routine gathering of information on disease incidents from the general public, medical or veterinary professionals and laboratories dealing with routine cases. Passive surveillance may lead to significant under-reporting of diseases and should, therefore, be supplemented by active disease surveillance particularly for important animal diseases.
   - *Active disease surveillance*: this involves proactive examination of individuals to actively seek out infection or disease, and targeted searching for evidence of disease in populations. Programmes may be broad-scale to capture any significant disease occurrences, targeted against specific high-threat diseases (*e.g.* diseases of particular public and animal health or agricultural significance), or designed to monitor the progress of individual disease control or eradication strategies. International trade may also guide surveillance schemes to establish national and regional disease status, especially where it relates to public health and economic initiatives. For livestock diseases which are spread by the movement of infected animals, areas where animals are moving should be targeted for surveillance (*e.g.* livestock markets, trading routes and border areas). Such areas or routes should also be carefully controlled during an outbreak.

2. **The speed of information flow** between different components of the disease surveillance system (immediate or routine).

3. **The rapidity of response required:** immediate investigation of disease incidence or routine and regular analysis of data with subsequent adjustments to control activities when required.

For a disease surveillance strategy to act as an early warning system, reporting, decision-making and response must be rapid. However, for endemic diseases, it may be more appropriate to evaluate the routine data collected to adjust or target control activities. National surveillance systems should include an integral approach and accommodate all needs.

Surveillance systems

All surveillance systems involve similar components (Figure 3-12). It may be possible to link and integrate several different surveillance systems.

The following functions may support surveillance systems:

- setting of standards (*e.g.* disease case definitions)
- training and supervision
- laboratory support
- communications
- resource management.

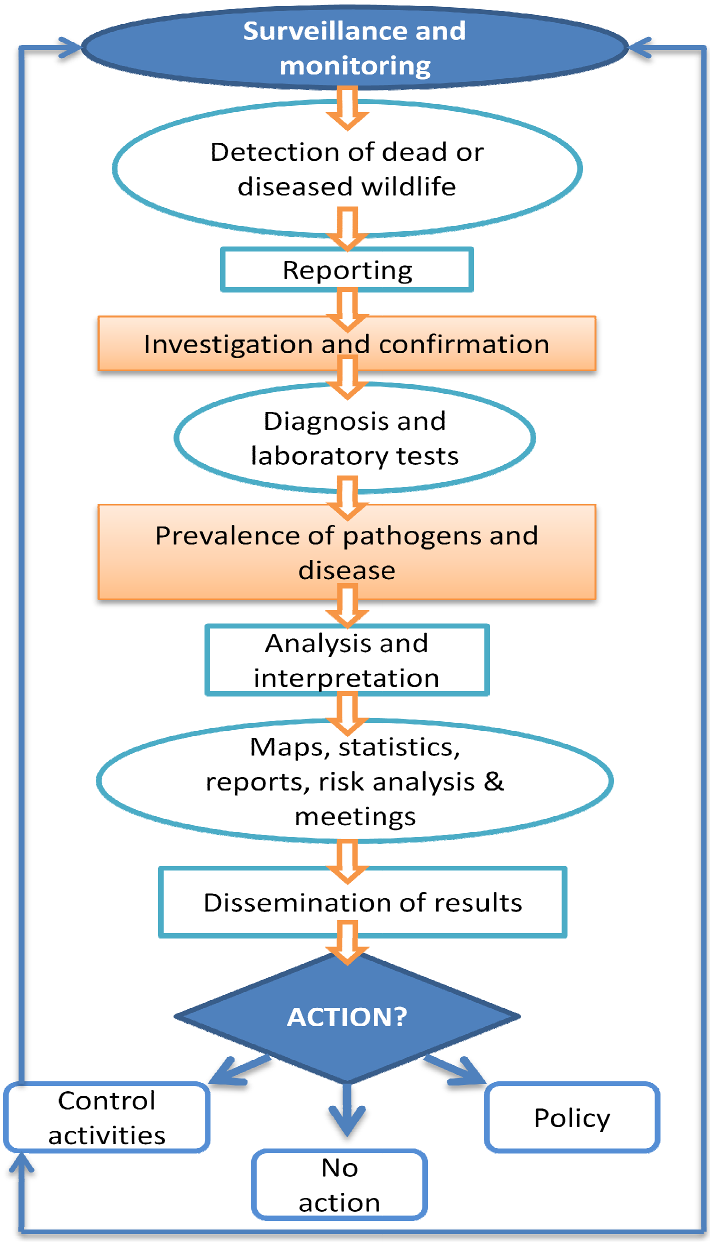

Figure 3-12. The key components of a surveillance and monitoring system.

The following tasks are recommended for improving animal disease surveillance:

1. **Identify key stakeholders and organisations relevant to the site**
    - state or local veterinarian or animal health officer (will most likely be lead person in regional surveillance effort)
    - public health contact
    - veterinary diagnostic laboratories.

2. **Identify relevant animal diseases for the site**
    - notifiable animal diseases
    - wildlife animal diseases
    - zoonoses.

3. **Familiarisation with country responses with reference to potential disease outbreaks at the site.**

4. **Establish standardised report forms for disease surveillance including definitions such as "confirmed" and "suspected".**

5. **Identify and collaborate with ongoing animal disease surveillance efforts at other wetland sites and government Ministries or Departments *e.g.* in the Departments of Agriculture or Health.**

6. **Identify efficient and effective communication channels with the relevant health authorities and laboratories and other wetland stakeholders and include opportunities for feedback.**

Prioritising diseases for surveillance

The following factors should be considered when determining which diseases to prioritise for surveillance:

- Whether the disease is of public health or agricultural importance.
- Whether the disease has a potentially severe impact (*e.g.* using indicators such as morbidity, disability, mortality).
- Whether the disease has significant epidemic potential.
- Whether the disease is a specific target of a local, regional, national or international control programme.
- Whether the information to be collected will lead to significant successful human/animal health action.

Figure 3-13. Conducting disease surveillance in an attempt to understand why water voles *Arvicola amphibius* in UK have suffered population declines (*WWT*).

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3.3.2 Identifying a disease problem

Disease is a natural part of any ecosystem. Identifying a departure from 'usual', 'natural' or 'expected' levels of mortality or morbidity can be complex and measures need to be put in place to help this process. Many of the other sections of this Manual will help in identifying a disease problem [*e.g.* **►3.1.4 Contingency planning, ►3.1.1 Risk assessment, ►3.2 Reducing risk of disease emergence,** and **►3.3.1 Surveillance and monitoring**].

Figure 3-14. Apparently healthy wildlife: identifying when a problem is emerging relies on a good understanding of what constitutes 'normal' mortality and morbidity and good early warning systems (*Sally MacKenzie*).

Early warning systems

Identifying a problem early before it becomes fully established or widespread can bring a wide range of potential benefits such as preventing loss of productivity from livestock, loss of biodiversity, loss of livelihoods, loss of disease-free status for trade, and reducing disease control costs. Thus, early warning systems are vital for swift responses.

CHECKLIST 3-2. Capacity requirements for identifying disease problems and informing early warning systems
☐ A good understanding of the use of the site by wild and domestic animals throughout the year and an understanding of their biology, abundance, behaviour and movements.
☐ A reasonable understanding of the epidemiology of particular diseases and of the stressors and other factors associated with disease outbreaks.
☐ An appreciation of possible routes of disease introduction (*e.g.* new livestock, migration, people movements).
☐ An understanding of times of greatest risk of disease outbreak at a site (*e.g.* a particular season or agricultural activity).
☐ Robust disease surveillance (both active and passive) in wildlife and livestock at a site. Ideally this should include regular visual checks of animal groups to screen for unusual behaviour, reduced body condition or productivity of domestic stock, signs of disease and/or mortality.
☐ Clear systems for reporting concern to a site manager and from the site manager to the local disease control authority.
☐ Use of these systems for immediate reporting of an unusual animal health problem to the local disease control authority.
☐ An understanding and capability to provide information and samples from a site to aid disease diagnosis [**►Sections 3.3.3 Sample collection and preservation, ►3.3.4 Sample transport and shipment, ►3.3.5 What data to collect at a suspected outbreak**].
☐ A communication network established between surveillance diagnosticians, site managers and disease control authorities both for two-way information flow about surveillance at the site but also from authorities about disease in surrounding areas including neighbouring countries.
☐ A communication network between site users in particular farmers and those working and living within wetlands.
☐ Awareness amongst wetland stakeholders of disease issues and an understanding of how to respond if there is an apparent problem.
☐ Training of site personnel to deliver the above [**►Section 3.5.2 Building capacity by education and training**].

Communication networks

Communication networks, developed ahead of an emergency situation, are vital not only between local and national authorities but also with other stakeholders, including local communities and landowners to ensure awareness raising and risk communication.

Early identification of a disease problem and the ability to respond are dependent on clear and well established channels of communication and formal or informal networks. A problem disease may manifest itself in various subtle ways and a site manager should have available a communication network that allows rapid synthesis of seemingly disparate information. For example, a flow of information should allow a site manager to become aware that there has been a recent incursion of wildlife due to disturbance in surrounding areas, that there has been some loss of productivity in the livestock using the site, or that a higher than expected number of dead or sick wild animals has been observed. Although these may all be entirely unrelated it should prompt the site manager to investigate further. This sort of approach to disease intelligence is key as it supplements disease surveillance data by making full use of additional qualitative information, enhancing awareness of disease related issues that may otherwise remain undetected.

Once a disease problem has been identified the response plan can then be put into action.
►**Section 3.3 Detecting, assessing and responding to new disease**
►**Section 3.4 Managing disease**

Further information and sources

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3.3.3 Sample collection and preservation

The wetland manager is not expected to be the final disease diagnostician, however they may play a key role in maintaining the quality of data and samples collected for surveillance and disease investigation purposes. All samples should be collected as fresh as possible and undamaged. Samples may include carcases, tissues, parasites, whole blood, serum, swabs, environmental material, faeces or ingested food *etc.*

Choosing a specimen

The most useful sample to collect is an entire carcase, which is fresh and undamaged by decomposition or scavengers. Such a sample allows a pathologist to carry out gross examination, take a variety of samples and perform a range of tests.

It is important to note that carcases of certain species such as fish and aquatic invertebrates, decompose more rapidly than those of birds or mammals and, therefore, examination or chemical-fixation (*e.g.* in alcohol) must occur as soon after death as possible. Collection of both healthy and diseased tissue from the same chemically-fixed specimen for comparison can prove invaluable in certain circumstances (*e.g.* for investigation of diseases of coral).

To help to reduce bias, samples should be representative of the range of species/individuals affected and several specimens of each species or class (*e.g.* age or sex) should be collected.

Personal protective equipment

The primary concern when collecting carcases or other diagnostic samples must be personal safety. Many animal diseases are zoonotic and every carcase or other diagnostic sample must be treated as a potential hazard to human health. Gloves (either plastic or disposable), coveralls, rubber boots and potentially masks, should be worn where possible and/or appropriate. If gloves are not available, inverted plastic bags can be used to protect the hands of the person collecting the carcase.

Each carcase should be double-bagged whilst using gloves and coveralls and the outside of bags and footwear should be disinfected before leaving the area. Any other specimens should also be double-bagged in plastic before leaving the area. Disposable protective equipment should also be double-bagged and incinerated at high temperature where possible.

Tissue collection

If submitting an entire carcase for analysis is impractical, it may be necessary to remove appropriate samples from specimens. It is advisable to first consult disease specialists about the method they require for sample preservation. The collection of parasites and their preservation should also be discussed (most parasites can be preserved in 70% ethanol). It is valuable to become familiar with these specialists, their fields of expertise and potentially the sample preservation methods they prefer, before an emergency situation occurs.

It is important to collect separate tissue samples where possible for microscopic examination, microbiology, toxicology and other types of analysis. For most tissue samples the following is appropriate: with a sharp knife or scalpel cut a thin (3-6 mm) section of tissue. If lesions are present include all or part of this affected tissue and adjacent apparently healthy tissue. Take care not to crush the tissue and place in a volume of preservative at least ten times the volume of the tissue to ensure adequate preservation.

Supplies

Basic supplies and equipment required will vary depending on the species and samples in question. It is advisable to keep a small kit packed for ease of ready sampling. Samples can be stored in appropriately sized plastic bags with a sterile interior as they are easily transported and labelled. Wide mouth plastic bottles with threaded caps are useful for sample storage. Indelible markers and pencils are necessary for sample labelling. Tape to prevent leakages is also advisable.

Photography

Photographing the site and carcases *in situ* can be extremely helpful to a diagnostician. Photographing any lesions (both external and internal) can provide useful information on their position and appearance. Include a ruler or other readily recognised objects in the photograph to provide scale, and keep a written record of contextual information on each photograph.

Labelling

For maintaining sample identity, proper labelling of samples is vital, together with preventing loss of readability of labels or their separation from samples. Where appropriate, affix a label directly to the sample (*e.g.* tie directly to a leg of the carcase). Write directly onto sample tubes or keep labels as close to the specimen as possible.

Double labelling is advisable, for example, directly label the sample or sample tube and also the bag in which the sample is placed. This helps prevent confusion and possible errors when multiple samples are received at the same laboratory. Use of pencil or waterproof ink on tags is advisable. The most durable tags are those made of soft metal that can be inscribed with a pencil. Waterproof paper can also be used when dealing with specimens from marine environments.

Information marked on carcase tags should include:

- name, address and telephone number of the person submitting the carcase
- collection site
- date
- reference number
- whether the animal was found dead or euthanised (plus method of euthanasia)
- brief summary of clinical signs.

Each carcase should then be place in a separate bag which should also be labelled.

Tissue samples taken into plastic bottles should be labelled on the outside of the bottle or a piece of masking tape placed around the tube. The label should include:

- date
- type of animal from which the sample came
- the type of tissue
- reference number.

If the sample is in a plastic bag the bag should be labelled in this way. Do not insert tags into bottles or bags with samples as they may contaminate the sample.

Preservation of specimens

Chill or freeze all specimens depending on the length of time it will take for them to reach a diagnostic laboratory (understanding that chilled is preferable), unless they are chemically fixed, in which case samples can be kept at ambient temperature. Freezing can damage tissue or kill pathogens and hence reduce options for diagnosis. However, if samples must be held for more than a few days they should be frozen on the day of collection to minimise decomposition.

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3.3.4 Sample transport and shipment

If diagnostic samples are spoiled during shipment then all the effort involved in their collection will have been wasted. Where samples need to be chilled or frozen an understanding of the concept of the 'cold-chain' is required. This refers to the need for samples to remain at the desired temperature and not to experience cycles of change (*e.g.* freezing and thawing) which can damage some samples irreparably.

The requirements for sample packaging and shipment vary between countries and diagnostic laboratories. It is, therefore, essential to contact the laboratory that will analyse samples to find out any specific shipping requirements as early as possible in the procedure. This will help with processing samples upon their arrival at the laboratory and reduce the risk of sample quality being compromised.

Transporting and/or shipping samples must not pose a biosecurity or human health risk. Seek advice from veterinary authorities about safety and regulations for transporting and shipping samples.

The most important considerations for successful sample transport and shipment are:

- prevent cross-contamination between specimens
- prevent decomposition of the specimen
- prevent leakage of fluids
- preserve individual identity of specimens
- properly label each specimen and the package in which they are sent.

Prevent breakage and leakage

Isolate individual specimens in their own containers and plastic bags. Wrap these samples with protective material where possible (*e.g.* bubble wrap or newspaper). Protect samples from direct contact with coolants such as dry ice or freezer blocks. Ensure that if any sample breaks or leaks the liquid does not leak to the outside of the package by containing all materials inside plastic bags, or other leak-proof containers, where possible.

Containing specimens

The plastic bags for containing specimens need to be strong enough to resist being punctured by the materials they hold and those adjacent to them. Polystyrene boxes within cardboard boxes are useful for their insulating and shock absorbing properties. The polystyrene should be at least two centimetres thick when possible. These boxes are strongest and least prone to break when their sides are straight. If polystyrene boxes are not available, sheets of this material can be cut to fit inside cardboard boxes with a similar effect (though the package is less leak-proof). The strength of the cardboard box needs to be sufficient for the weight of the package. If hard plastic or metal insulated boxes are used for transport, cardboard boxes around them can be used for protection and to attach labels.

Cooling and refrigeration

Keeping samples chilled

When it is necessary to keep samples cool during shipment (*i.e.* at refrigerator temperature of approximately 4^{o}C), chemical ice packs are preferable to wet ice due to less leakage when they thaw. It is possible to make ice packs by freezing water inside a plastic bottle that is sealed (not filled completely and taped closed to prevent the top coming off in transit) and then placed in a sealed plastic bag to further prevent leakage. If frozen carcases are being transported they can act as a cool pack for other samples sent in the same container. When using ice packs they should be interspersed between samples to achieve a uniform temperature throughout.

When submitting dead fish for *post mortem* examination they should be wrapped in moist paper to prevent them drying out and then refrigerated but not frozen. Fish decay very quickly but a fish refrigerated soon after death may be held for up to twelve hours before examination and sample fixation.

Keeping samples frozen

Dry ice (solid carbon dioxide) or in some circumstances liquid nitrogen can be used to ship frozen specimens. The gaseous carbon dioxide given off by dry ice can also damage some disease agents and this must be considered before using it for tissue transport. As the volume of both dry ice and liquid nitrogen expand as they change to gas, specialist containers that allow for this expansion are needed for their transportation.

Note: Shipment of formalin, dry ice, liquid nitrogen and alcohol is regulated in many countries and must be cleared with a carrier before shipping.

Samples preserved in formalin, other chemical fixative or alcohol can be transported without chilling.

Shipping

It is important to pack any space within packages with a substance such as newspaper which will prevent movement of containers, act as a shock absorber and may also soak up any potential leakages. It also has insulating properties.

Packaging and labelling

Packaging and labelling of specimens must conform to the regulations of the country from which the package is sent and also those of the country in which it will be received (if it is being sent to a laboratory in another country). It is important to mark the outside of the package with the required labelling regarding the type of specimen being transferred and where necessary the method of cooling (*e.g.* packages containing dry ice should be marked with specific symbols).

Permits

Permits or licences may be required for collection and transportation of some samples such as CITES-listed species, and invasive collection of samples from living animals, and these permits and licensing regulations may vary between locations. Animal health permits will likely be required for crossing national boundaries. Advice from national authorities about permit requirements must be sought prior to collection and transportation of samples.

Carriers

Samples should be shipped where possible by carriers that can guarantee 24-hour delivery to the diagnostic laboratory. Where possible arrange for collection of sample packages from the point of origin to avoid delays. When shipping arrangements have been made, contact the diagnostic laboratory to provide them with further details including estimated time of arrival and any shipping reference numbers.

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3.3.5 What data to collect at a suspected outbreak

The wetland manager will not be responsible for leading an epidemiological investigation but can play a key role in this. Detailed field observations during the course of an outbreak and information about events preceding it, may provide valuable data on which to base a diagnosis and corrective actions. It is important for the information gatherer to keep an open mind about the potential cause of the problem. Some information which may seem irrelevant in the field may become very important when piecing together the events leading up to an outbreak. A thorough chronology of events is key to diagnosis and disease control operations, and is almost impossible to obtain some time after the outbreak has occurred.

A key concept is that of explaining to the diagnostician how the affected individuals relate to the whole *population at risk*. As an example, 100% of the dead animals may be adult males but the population present (*i.e.* at risk) may have only contained adult males and hence the disease is not *necessarily* related to age or sex.

How to record data

It is important to record as much relevant information as possible as soon as events unfold.

Photographs and video footage can quickly convey specific information such as land use, landscape, environmental conditions, gross lesions and the appearance of clinical signs in sick animals. Sources of information may include local people, landowners and agencies working in the area preceding or during an outbreak. Information should be passed to the diagnosticians as soon as possible, updating them as appropriate.

Which data to collect

Checklist 3-3 provides a summary of the information to collect at a suspected outbreak.

CHECKLIST 3-3. Which data to collect at a suspected outbreak.

A broad range of data should be collected at a suspected outbreak, including:

- ☐ Population(s) at risk *i.e.* contextual information
- ☐ Species affected (including species unaffected *i.e.* population(s) at risk)
- ☐ Age
- ☐ Sex
- ☐ Number sick/dead
- ☐ Clinical signs
- ☐ Estimation of time of disease onset
- ☐ Location(s)
- ☐ Type of habitat/area and land use
- ☐ Environmental factors (*e.g.* weather conditions)
- ☐ Other contextual information
- ☐ Specific features of problem areas (*e.g*. population movements)

Population(s) at risk

Perhaps the most important contextual information is the species and numbers of individuals present and affected in the vicinity of a mortality event. A broad range of affected host species may suggest a storm, other sudden environmental event or toxic/poisoning incident, whereas a narrow host range, with other species present and at risk yet *unaffected*, may indicate a specific infectious agent.

The proportion of animals affected in the population provides information about the nature and seriousness of the problem. Statements such as '100 dead birds were found' are meaningless without an indication of what proportion of the population this constitutes.

Ensure that demographic data collected from affected animals are related to that of the wider population present. For example, if all the animals were juveniles yet this was the population present and at risk at the time, then this needs to be explicit to the diagnostician.

Species affected

It is important to note as much detail as possible regarding the species affected. See above point regarding species *not affected*.

An understanding of the ecology of the affected species will help to determine why some species might have been affected and others not. As an example, some species may have avoided exposure to an infectious source or poisoning event through differences in feeding behaviour.

Age

Where possible assess the age of the population at risk and the age of those individuals affected. Some diseases may only affect juveniles due to age-related immunity in adults. Other diseases affect all ages although those that are older or younger may be more susceptible due to other stresses. Diseases may also affect age groups differentially due to behavioural differences in feeding habits, for example.

Sex

Where possible assess the sex ratio of the population at risk and the sex of those animals affected. There may be inherent physiological or behavioural reasons for sex-related differences in susceptibility to disease.

Number sick/dead

The number of sick individuals compared with the number of deaths can help to determine the nature of the disease and the length of time it takes to become fatal. The longer it takes, the greater the proportion of sick compared with dead individuals and the less acute the disease process. This can also apply to the proportion of an area affected in marine environments. Again, relate numbers of affected animals to the population that was at risk.

Make an assessment of the number of sick or dead animals which may have been lost to predators and scavengers or that may have decomposed.

Clinical signs

As much detail as possible should be recorded about clinical signs observed in sick individuals, including changes in behaviour, physical features or temperament. Photographs and video footage can be extremely helpful in recording this information.

Estimation of time of disease onset

Establishing a timeline of events in an outbreak is crucial. When estimating the time of onset of a disease incident, aspects to be considered include:

- The earliest date when people would have been on site to observe individual animals showing signs of illness or mortalities.
- The date mortalities were first reported.
- The proportion of fresh carcases compared with those decomposed or scavenged.
- The number and type of scavengers should be assessed to determine how long carcases are likely to remain in view.
- Air, water and soil temperatures will affect rates of decomposition and should be taken into account when estimating how long individuals have been dead.
- Any change in coat or plumage (including stage of moult) between live and dead individuals as this can help pinpoint how long ago an individual died.
- Size of any dead young compared with known growth rates (and size of living young) to help assess how long ago the individual died.

Location

Record precisely (ideally GPS coordinates), in as much detail as possible, the location and spatial extent of the event and of carcases or sick individuals, so these data can be accurately mapped.

Type of habitat/area and land use

Identify the habitat type, including soil and vegetation present. Describe land use (by humans and animals) including any recent changes. This information together with topography can often be illustrated well using photography or video footage. Particular attention should be paid to areas where groups of dead individuals were found. Any differences in habitat in these areas should be noted.

Environmental factors

It is important to describe the habitat and land use *etc.* and also determine if any unusual event preceded or precipitated the suspected outbreak, *e.g.*:

- Stress can be caused by abrupt changes in environmental conditions (*e.g.* storms, temperature fluctuations or precipitation) and can precipitate outbreaks of disease.
- Food shortage or imbalance can also lead to loss of condition and disease outbreaks.
- Changes in water level may disperse or concentrate populations and change the availability of food and water and access to potential toxins (*e.g.* invertebrate die offs leading to outbreaks of avian botulism).
- Estimation of whether biting insect populations have increased can be important, as they may serve as disease vectors.
- Water quality may be important as poor water quality may contribute to disease and mortality (*e.g.* avian botulism). Primary contamination by toxic substances can also lead to morbidity and mortality (*e.g.* oil).
- Recent management practices (*e.g.* pesticide spraying) should be recorded, as should any previous disease issues in the area.

Other contextual information *e.g.* population movements

Other contextual information should be recorded, particularly if there have been changes in conditions or populations. Information on the condition and behaviour of animals prior to the outbreak should be recorded if possible, as should any changes in their abundance and distribution. Local people may be the best source for much of this information.

Specific features of problem areas

Other specific features not mentioned above should be noted and provided to the diagnostician.

Supplementary investigations

If further investigations are carried out these reports should be summarised and kept as a supplement to the original findings. These reports should be copied to the diagnostic laboratory where the specimens were sent. The date of investigations, type of searches carried out (*e.g.* air or ground), number of investigators, time spent on searches, weather conditions and time of day the search was carried out should all be reported.

Further information and sources

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3.4 Managing disease

Diseases are natural components of ecosystems and do not necessarily require any management intervention. Deciding whether a disease should be managed or not, rests largely on the extent to which it endangers human and animal health and welfare, economic systems, conservation aspirations, and the likelihood that intervention will achieve disease management objectives.

The appropriate approach will depend on the characteristics of the problem and, when dealing with an infectious disease, on the correct identification of reservoirs, hosts and vectors of infection. Prevention and control of a disease is usually more easily achieved than complete eradication [►**Section 3.4.8 Eradication, elimination, stamping out and lethal intervention**].

Appropriate disease management options will depend on whether one is dealing with endemic or epidemic disease, and whether the intention is to prevent or control disease spread. See ►**Section 2.6** for the principles of proactively and reactively managing animal disease and ►**Section 3.2** for techniques to both reduce risk of disease emergence and help limit potential spread.

Disease management options

Management measures may target the:

☐ **PATHOGEN**

1. Biosecurity ►**Section 3.2.4**
2. Disinfection and sanitation ►**Section 3.4.1**
3. Collection and disposal of carcases ►**Section 3.4.2**

☐ **VECTOR**

1. Control of vectors ►**Section 3.4.3**
2. Genetic manipulation ►**Section 3.4.5**

☐ **HOST**

1. Reducing stressors ►**Section 3.2.1**
2. Disease zoning, barriers and buffer zones ►**Section 3.2.2**
3. Standards for releasing and moving animals ►**Section 3.2.3**
4. Vaccination ►**Section 3.4.4**
5. Genetic manipulation ►**Section 3.4.5**
6. Movement restrictions ►**Section 3.4.7**
7. Eradication, elimination, stamping out and lethal intervention ►**Section 3.4.8**

☐ **ENVIRONMENT**

1. Habitat modification ►**Section 3.4.6**

☐ **HUMAN BEHAVIOUR AND ACTIVITIES**

1. Communication, education, participation and awareness ►**Section 3.5**

Ultimately, an integrated approach involving several methods is likely to be the most successful in managing diseases in a wetland.

Above all, management measures should be sustainable, based on accurate epidemiological and ecological information, and must balance the requirements for preserving biodiversity and protecting human health and economic well-being. This chapter describes several options for managing diseases in wetlands.

- *Disinfection and sanitation*
- *Collection and disposal of carcases*
- *Control of vectors*
- *Vaccination*
- *Genetic manipulation*
- *Habitat modification*
- *Movement restrictions*
- *Eradication, elimination, stamping out and lethal intervention*

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- **The appropriate approach to disease management will depend on the characteristics of the problem** and, when dealing with an infectious disease, on the correct identification of reservoirs, hosts and vectors of infection. **Management measures may target** the **pathogen**, **host, vector, environmental factors or human activities**. Ultimately, an **integrated approach** involving several complimentary measures is likely to be most **successful** in managing diseases in wetlands.
- **Disinfection and sanitation** procedures target pathogens and can be **very effective at controlling spread of infection** but must be **used with caution in wetland situations** to avoid negative impacts on biodiversity.
- **Animal carcases** represent a significant **potential source of infection** and **require rapid and appropriate collection and disposal**. Disposal options are varied and again need to be used with caution in wetland situations to reduce risks of pollution of water courses or further spread of infection.
- **Targeting vectors in integrated disease control strategies can be effective** and usually take the form of environmental management, biological controls and/or chemical controls, or actions to reduce the contact between susceptible hosts and vectors. To reduce negative impacts on biodiversity **caution must be used when using these measures within wetlands**.
- **Vaccination programmes**, often supplemented by other disease control measures, **can help control** and even eliminate diseases affecting livestock. Vaccination of wildlife is feasible but it is often complex - other management strategies may be of greater value.
- **Habitat modification in wetlands can eliminate or reduce the risk** of disease, by reducing the prevalence of disease-causing agents, vectors and/or hosts and their contact with one another, through the manipulation of wetland hydrology, vegetation and topography and alterations in host distribution and density.
- **Movement restrictions of animals and people**, usually imposed by government authorities, can be an **effective tool** in preventing and controlling disease transmission through avoiding contact between infected and susceptible animals.
- **Complete eradication of a disease requires a thorough understanding of its epidemiology, sufficient political and stakeholder support** and thorough resourcing and is thus rarely achieved! Elimination of disease from an area is a more likely outcome although this depends on measures to prevent re-emergence being taken. ‘Stamping out’ (involving designation of infected zones, quarantine, slaughter of susceptible species, safe disposal of carcases and cleaning and disinfection) is a management practice used for rapidly reducing the prevalence of a disease during an outbreak situation.

3.4.1 Disinfection and sanitation

Disinfection and sanitation

The spread of, and exposure to, *infectious* diseases can be significantly reduced through using effective sanitation and disinfection processes. Sanitation measures involve preventing animal contact with physical, microbiological, biological or chemical agents of disease, which are often found in wastes, and maintaining clean, hygienic conditions. Inadequate sanitation is a major cause of disease worldwide and simple measures for improving sanitation are known to have significant beneficial impacts on public and animal health.

Disinfection prevents the mechanical transmission of disease agents from one location to another by animals and inanimate objects, by eliminating many or all pathogenic microorganisms (except bacterial spores) on inanimate objects so that they will no longer serve as a source of infection. Disinfection measures can be used to help maintain good sanitation and hygiene.

Figure 3-15. Disinfection following fieldwork prevents transfer of infection on fomites such as boots and clothing.

Measures taken to prevent a disease outbreak

For public health and biosecurity reasons, people working in wetlands should maintain high standards of sanitation and hygiene, and avoid direct contact with human and animal faeces, solid wastes, domestic, industrial and agricultural wastes [►**Section 3.2.4 Biosecurity**]. Effective sanitation and hygiene can be achieved through engineering solutions (*e.g.* sewerage and wastewater treatment including treatment wetlands [►**Section 3.2.4 Biosecurity**]), safe storage structures (*e.g.* water and septic tanks), and by hygiene practices (*e.g.* disinfecting equipment and washing hands with soap). Any items that have been in contact with waste materials (*e.g.* clothes, equipment and hands) should be thoroughly cleaned and disinfected after use. Livestock housing should be regularly cleaned and disinfected and waste and clean water should be separated and safely stored. Waste materials from captive animals should be properly processed and disposed of.

Cleaning is a necessary first step that allows the subsequent disinfecting agent to come into direct contact with pathogens on the surfaces of an object. Cleaning is important as many disinfectants are inactivated by organic debris.

Some viruses, bacteria and other infectious agents can persist in the environment for protracted periods. Disinfection is only practical for circumstances in which the pathogen or disease transmission occurs in a very limited area. The appropriateness of disinfectants will be informed by information on the presence of non-target species and other potential environmental impacts, particularly any adverse effects on wetland ecosystem function. Disinfection for wildlife disease situations is often difficult and likely to be most effective where wild animals are concentrated, such as at artificial feeding or watering sites.

Measures taken during a disease outbreak

During a disease outbreak, it may be necessary (if practical) to disinfect the local environment to prevent recurrence. Procedures are generally similar, however, the nature and infectivity of the pathogen will affect the protocols employed. For example, chytrid fungus and foot and mouth disease virus will require very different procedures for decontamination. As a consequence, disinfection of a disease outbreak site should always be conducted under the guidance of disease control specialists.

From the above, the following should be done, as appropriate: during disinfection activities, easily cleaned protective clothes such as waterproof coveralls and rubber boots and gloves should be worn, and all clothes should be thoroughly washed after use and before leaving the outbreak area. If possible, personnel should wash their hair before leaving the area, and always before going to other wetland areas. Personnel handling potentially infectious agents should not work with similar species or those susceptible to disease for at least seven days after participating in disease control activities.

Disinfection processes require a suitable disinfectant, containers for the solution once it has been diluted to the appropriate strength and a suitable method for its application. Vehicles and boats with pumps and tanks can be used to store and dispense disinfectant. All vehicles should be cleaned and disinfected on entering and leaving an outbreak area. Brushes, buckets, and containers that can be used to clean and disinfect boots and pressure sprayers that can be used to dispense the disinfectant are also required.

Disease control specialists should advise on the most appropriate type of disinfectant and its application in wetland settings.

The effectiveness of a disinfectant in eliminating or reducing pathogenic microorganisms depends on the:

- **Number and location of microorganisms:** generally the larger the number of microbes, the more time required to destroy all of them.
- **Resistance of microorganisms to certain chemicals.**
- **Concentration and potency of disinfectants.**
- **Physical and chemical factors:** temperature, pH, relative humidity, and water hardness (*e.g.* the activity of most disinfectants increases as the temperature increases).
- **Organic and inorganic matter:** serum, blood, pus, faeces or other organic materials can interfere with the effectiveness of disinfectants.
- **Duration of exposure:** items must be exposed to the chemical for the appropriate contact time.

Commercial disinfectants are available from appropriate stores and sources. Disease control contingency plans should identify readily available sources of supplies and equipment needed for disinfection activities in case of an outbreak. Wetland managers, particularly those caring for housed livestock, should consider keeping a supply of disinfectant for general use. [►**Section 3.2.4 Biosecurity** for further information on biosecurity measures used in disease control].

Health and safety risks of using chemicals

Disinfectants may be toxic to humans as well as animals and plants, and therefore all chemicals should be used in accordance with the relevant safety precautions. Key factors that help to assess the human health risk of chemical exposure include the duration, intensity (*i.e.* how much chemical is involved) and the route (*e.g.* inhalation, skin) of exposure. Acute toxicity could be caused by an accidental chemical spill. Wetland managers may be responsible for informing workers about the chemical hazards involved and implementing disinfection control measures. Where required, wetland managers should be able to readily provide workers with appropriate personal protective equipment and Material Safety Data Sheets (usually available on the internet) for each chemical or mixture of chemicals that may be in use.

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Wetlands International, (2010). **Wetlands & Water, Sanitation and Hygiene (WASH) - understanding the linkages.** Wetlands International, Ede, The Netherlands. http://www.wetlands.org/WatchRead/Currentpublications/tabid/56/mod/1570/articleType/downloadinfo/articleId/2467/Default.aspx [Accessed March 2012].

3.4.2 Collection and disposal of carcases

When considering collection and disposal of carcases it is important to remember that approved methods vary according to animal species, disease, scale of mortality and country. Animal health authorities should be contacted to advise on appropriate measures remembering that the health and safety of the personnel involved in any disposal operation are paramount.

Rapid and effectively planned carcase collection and disposal is essential to prevent spread of infectious disease and to reduce potential secondary poisoning in the case of toxic diseases. Presented below is a broad overview of the most commonly used methods for animal carcase collection and disposal, each has strengths and weaknesses which should be considered in the context of each specific situation.

Collection of carcases

Ideally carcases can be dealt with *in situ* to reduce chances of spread of infectious agents. However, in most circumstances where an outbreak has occurred and there are a number of carcases, they will need to be gathered to a central location for disposal. To help prevent potentially contaminated body fluids leaking during collection and transport to the central location, wherever possible (depending on size of dead animal), the carcases should be *double* bagged in plastic leak-proof bags (noting that claws, beaks *etc.* may accidentally pierce bags). Wooden containers are difficult to decontaminate as fluids soak into wood so, wherever possible, plastic or metal bins/barrows *etc.* should be used for transporting bagged carcases.

If carcases are being transported off-site to disposal facilities this must be done in leak-proof vehicles. Advice should be sought from animal health authorities regarding transportation of potentially infectious carcasses.

Burial of carcases

This is the often a preferred method of disposal as it is relatively easy to organise, quick, inexpensive, has potentially fewer immediate environmental hazards and it is a convenient means of disposing of large numbers of carcases. However, the suitability of this method needs to be considered carefully in or around wetlands as pits must not contaminate ground water nor be susceptible to inundation. Also care must be taken to avoid later exposure of carcases to people or other animals. Open pits were historically used for this purpose but potential problems include exposure to scavengers and the threat to groundwater quality. If carcases do not decompose sufficiently then contaminants may leach from the pit.

Closed pits are now generally favoured with at least a metre of topsoil laid over carcases. This restricts the carcases rising in the pit due to gas entrapment, helps prevents access to scavengers, absorbs decomposition fluids and facilitates odour filtration. Potential scavengers can be further dissuaded by the addition of lime or fuel oil to the carcases, or use of thorny plants such as acacia spread across the pit.

Specialist animal health advice should be sought on pit site selection as a poor choice can have serious adverse effects on the environment and public and animal health in the vicinity. Factors to consider include:

- height of water table
- distance from watercourses or wells
- access to site
- facilities available
- equipment required
- safety to personnel
- acceptability to landowner
- protection from public view
- distance from residences/roads
- surface slope
- cultural/historical considerations
- biosecurity considerations.

Incineration (burning) of carcases

Incineration of carcases is advantageous due to the generally pathogen-free solid waste by-product. However, factors to consider prior to burning carcases include:

- location of site
- prevailing wind direction
- access to site
- type of animal carcase involved
- fuel availability
- number of carcases to burn
- environmental considerations.

Common methods of incineration include open air burning, fixed facility incineration and air curtain incineration.

To achieve the high temperatures required to completely consume carcases in open air burning additional combustible materials (*e.g.* timber or fuel oil) must be used. Carcases can be either put on a platform above a fire at ground level or within a pit. Soaking or sprinkling carcases with fuel oil and allowing approximately 15 minutes for absorption results in a high burn temperature to be achieved which aids complete incineration. However, structures such as burning platforms must be capable of withstanding this heat without collapsing. It is worth noting that animals with higher fat content will burn faster than those with a lower body fat.

When burning either above ground or in a pit, it is important to burn carcases one layer at a time as piling them up may result in incomplete incineration of those in the middle. It is important to note that if the burn is incomplete then foul odours, particulates and pathogens can be released into the wider environment after the fire has been extinguished.

Important factors for the location of open air burning include direction of prevailing winds, surrounding habitat or land use and visibility/access by the public. Surrounding vegetation should be cleared to reduce the risk of fire spreading and, in dry situations, pit burning is advised to reduce this risk further.

Fixed facility incinerators are available in different sizes from small on-farm units to large specialist municipal incinerators. All produce controlled high temperature burning and many are fitted with afterburners to ensure complete reduction of carcases to ash. Portable controlled burning units may be available and can be brought on site in some situations.

Air curtain incinerators involve a powered fan blasting air over a burning pit with the resultant high air pressure and temperature obliterating carcases and restricting the escape of particulates. Such devices can incinerate other contaminated organic materials alongside carcases and, as such, are useful in large scale infectious disease outbreak situations.

Composting of carcases

Composting of carcases involves controlled decomposition during which heat and microorganisms consume the organic materials. The process is relatively lengthy involving an initial phase of up to several months of high temperature, a similar period of lower-temperature ‘curing’ or stabilisation, resulting in the production of carbon dioxide, water vapour, heat and compost.

Within a contained unit (a bin or even a building) with an impermeable base and lid/roof for controlling water vapour, alternate layers of carcases and litter (or straw) are built up on top of a base layer of litter. It is important to ensure the right carbon to nitrogen ratio to achieve good composting conditions.

For some situations in hot countries it may be possible to rapidly compost carcases by placing them in sealed heavy duty black plastic and exposing to the sun for an appropriate period of time until decomposition has occurred.

Advantages of composting include relatively low cost, low levels of pollution and a fertiliser as an end product. However, this approach may be inappropriate for use in many infectious disease situations, as the causative organism may not be destroyed. Additionally the length of time the composting process takes (which requires monitoring) may limit its usefulness.

Rendering

Rendering involves cooking carcases as a means to separate animal fats and proteins with the resultant products sometimes used as animal foodstuffs and for other industrial processes. Rendering is often not appropriate for infectious disease situations, due to the risks of spread of infection (including risk of transmissable spongiform encephalopathies as was seen with the outbreak of bovine spongiform encephalopathy in the UK in the 1980s). This is a specialist disposal technique and animal health advise should be sought regarding its suitability.

Further information and sources

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3.4.3 Control of vectors

Certain biological vectors of disease are closely associated with wetlands and play a role in transmitting disease pathogens amongst hosts. Arthropods are particularly important disease vectors of wetlands. Biting flies such as mosquitoes, midges, horse flies, tsetse flies and sand flies, can transmit viruses, bacteria, protozoa and nematodes. Non-biting house flies, blow flies, and flesh flies mechanically transfer disease pathogens on their legs and other body parts, or by dropping infected faeces or even vomit. These organisms may fly, or be carried, large distances from wetland habitats by wind or vehicles. Other important disease-carrying vectors associated with wetlands include snails, crustacea and ticks.

Vector control strategies aim to reduce transmission by reducing or eliminating the vectors and by reducing contact between them and potential hosts. Measures vary depending on the disease and vector species, but may be broadly categorised as environmental management, biological control and chemical control.

Environmental management

Environmental management measures may involve altering hydrology, topography or vegetation to reduce the capacity of the local habitat to maintain populations of disease vectors and to provide suitable habitat for vector predators. This can be conducted through environmental modification where there is a temporary, long-lasting or permanent physical transformation of vector habitats (*e.g.* through irrigation schemes that increase water flows and reduce mosquito oviposition sites), and environmental manipulation where there are temporary changes to a vector habitat (*e.g.* by removing specific types of vegetation that provide breeding sites for vectors, or altering water levels at key vector breeding times to reduce their productivity).

Modification or manipulation of human habitation or behaviour can reduce contact between disease carrying vectors and animals and humans. This may involve improved water storage (*e.g.* with covers) and solid waste management and personal protective measures such as mosquito nets and effective hand hygiene.

Biological control

Biological control measures use living organisms such as larvivorous fish or bacteria, to manipulate pathogens, parasites, predators, competitors, alternate hosts and other symbionts of target organisms. Introduction of sterile vectors can also help reduce the vector population and hence disease transmission. Advantages of such measures include specificity against target organisms and no chemical contamination of the environment. However, there are a number of potential disadvantages: the efficacy of reducing disease transmission through biological control measures is unknown for many vector species; there are various (often significant) ecological considerations; rearing organisms may be expensive; there may be difficulty in their application and production; and their use will be limited to aquatic sites where temperature, pH and organic pollution meet the requirements of the agent.

Chemical control

Chemicals can be quickly applied and may rapidly kill vectors at a relatively low financial cost. However, chemicals may cause damage to wetland environments and their wildlife and prolonged use may lead to the development of resistance in some vector populations. Using pesticides for the control of vectors may not be considered 'wise use' of a wetland site particularly if they affect non-target species. The efficacy of chemicals in reducing vector populations depends on the appropriateness of formulations, local conditions and the vector species itself.

Appropriateness of vector control measures should be based on the vector species, life stages involved, type and extent of habitats to be treated, the presence of non-target species of special concern, in addition to other environmental impacts, such as any likely adverse effects on wetland ecosystem function.

Vector control programmes

Integrated vector management strategies (to also be integrated into the wetland management plan ►**Section 3.1.3**) use a combination of vector control measures and are often most effective in reducing disease transmission. When designing a vector control programme, an assessment should be made of vector ecology (species, habitat, population, distribution and breeding cycle), the immune status of the host populations at risk, and the nature and prevalence of the parasite. This assessment will inform what may be achievable from a strategy.

Common objectives for a strategy include the prevention and control of outbreaks, stopping preventable deaths and minimising illness. Advice on the most appropriate vector control measures and the availability of control resources should be sought from the appropriate national and international authorities.

FURTHER INFORMATION: Tick control

***Environmental (habitat) management*:**

Success in reducing the density of ticks is largely dependent on regular removal of ground cover, especially the mulch that shelters all tick stages, by mechanical means, herbicides or by fire.

- Remove low-growing vegetation and brush to reduce the structural support required by ticks to contact hosts.
- Remove leaf litter and underbrush to eliminate habitats for ticks and their small mammal hosts.
- Controlled burning of habitats favoured by ticks can reduce tick abundance from six months to one year.
- Larger host mammals such as deer, may be contained within certain areas separating them from areas inhabited by people (*e.g.* public walk ways) by physical barriers.
- The environmental impact of suggested control measures should be evaluated and appropriate approvals should be granted before they are undertaken.

Environmental management – adapting behaviour of people and animals

People – personal protection:

- Wear light coloured clothing to enable ticks to be observed easily.
- Apply insect/tick repellent containing DEET.
- Wear clothing to cover arms, legs, and feet whenever outdoors, tucking trousers into socks or wearing gaiters helps prevent tick access to legs.
- Walk in the centre of trails to avoid contact with overgrown grass and brush.
- Check yourself, others and companion animals thoroughly for ticks and manually remove any ticks found (►Tick removal).

People – tick removal:

- Use blunt curved tweezers or a thread.
- Grasp the tick as close to the skin surface as possible and pull upwards with a steady, even pressure.
- Pull firmly enough to lift up the skin, holding this tension for 3-4 minutes and the tick should back out.
- Do not twist the tick as this may cause the mouth to detach and remain in the skin.
- Do not squeeze or crush the tick as its fluids may contain bacteria.
- Dispose of the tick immediately. If you have any additional disease concerns, put the tick in a plastic bag and freeze it for taking to a medical professional.
- Immediately wash your hands and the affected area with soap and water.

Animals:

- Manually remove ticks from animals if practical to do so (►Tick removal).
- If tick infestation occurs, livestock can be dipped in recommended acaricides or pesticides.
- Consider use of resistant breeds of cattle.
- Rotational grazing regimes can also control infestations.
- There are vaccines available for some tick-borne diseases and even against some species of ticks themselves.
- Strict quarantine measures are important for domestic animal movements, particularly when importing into tick-borne disease-free areas.
- Companion animals should be closely monitored for ticks on a daily basis. Commercial products are available for controlling fleas and ticks on pets.

Integrated tick control

An integrated approach which uses personal protection methods, tick monitoring, habitat modification and acaricide application may be most effective in controlling ticks. Tick control measures should be tailored to the biology and seasonality of particular species. When choosing control measures, the type of habitat, density and activity of the human population, incidence of infection in the vector species, extent to which tick control is necessary, and degree of environmental modification that is acceptable should be carefully considered.

►Chapter 4: Tick-borne diseases factsheet

FURTHER INFORMATION: Mosquito control

Environmental (habitat) management

Encourage mosquito predators and their access to mosquito breeding habitats:

- Connect shallow water habitat (mosquito breeding areas) with deep-water habitat > 0.6 m (favoured by larvivorous fish) and steep sides, through meandering channel connections, deep ditches and tidal creeks.
- Include at least some permanent or semi-permanent open water.
- Construct artificial homes or manage for mosquito predators such as bird, bat and fish species.
- Do **not** introduce non-native species of fish or other predators into the wetland for mosquito control.

Reduce mosquito breeding habitat:

- Reduce the number of isolated, stagnant, shallow (5-7 cm deep) areas.
- Cover or empty artificial containers which collect water.
- Manage stormwater retention facilities.
- Strategically manipulate of vegetation.
- Vary water levels temporally.
- Construct a vegetation buffer between the adjacent land and the wetland to filter nutrients and sediments.
- Install fences to keep livestock from entering the wetland to reduce nutrient-loading and sedimentation problems.

In ornamental/managed ponds:

- Add a waterfall, or install an aerating pump, to keep water moving and reduce mosquito larvae. Natural ponds usually have sufficient surface water movement.
- Keep the surface of the water clear of free-floating vegetation and debris during times of peak mosquito activity.

Chemical control

It may be necessary to use alternative mosquito control measures if the above are not possible or ineffective. The environmental impact of vector control measures should be evaluated and appropriate approvals should be granted before undertaken.

- Use larvicides in standing water to target mosquitoes during their aquatic stage. This method is deemed least damaging to non-target wildlife and should be used before adulticides.
- Use adulticides to spray adult mosquitoes.
- During periods of flooding, the number and extent of breeding sites is usually too high for larvicidal measures to be feasible.

Open marsh water management

Control mosquitoes by introducing their natural predators to areas of tidal marsh using a system of pools connected by radial ditches. Fish feed on mosquitoes during high tide, then retreat to sumps or reservoirs at low tide.

Environmental management – adapting behaviour of people and animals

People:

- Wear light coloured clothing which covers arms and legs.
- Use impregnated mosquito netting when sleeping outdoors or in an open unscreened structure.
- Avoid mosquito-infested areas or stay indoors when mosquitoes are most active.
- Avoid physical exertion, and use colognes and perfumes sparingly as these may attract mosquitoes.
- Use mosquito repellent when outdoors. Note that some repellents cause harm to wildlife species, particularly amphibians. Wash hands before handling amphibians.
- Use citronella candles and mosquito coils in well ventilated indoor areas.
- Use mesh screens on all doors and windows.

Animals:

- Use insect repellent. Note that this method should not be solely relied upon.
- Use screened housing with measures to eliminate mosquitoes from inside structures.
- Use fans to reduce the ability of mosquitoes to feed on animals.

FURTHER INFORMATION: Snail control

Environmental (habitat) management

Reduce snail populations

Strategies should be implemented with specific knowledge of the ecology of the causative snail. Water impoundments of all shapes and sizes (*e.g.* irrigation systems, lakes and dams) provide fertile breeding grounds and good habitat for freshwater snails and encourage close and frequent contact between people and infected water. The following habitat alterations may help reduce snail populations.

Alter flow rate and water levels to disturb snail habitats and their food sources:

- Include 'v' shaped banks in irrigation channels.
- Remove vegetation/silt in channels to avoid a drop in velocity which may lead to further vegetation growth and good habitat for snails. Note that personnel involved in the manual removal of vegetation are increasing their exposure to snails. Frequent removal may be needed.
- Flow rate should only be addressed with knowledge of the ecology of the snail in question *e.g.* for *Biomphalaria* and *Bulinus* flows greater than 0.3 m/s would suffice but most snails can withstand flows up to 0.5 m/s.
- Borrow-pits, small pools and ponds serving no special purpose (for humans, wildlife or livestock) may be drained to eliminate breeding sites.

Expose snail habitat:

- Remove littoral vegetation from the sides of canals feeding irrigation projects to expose snail habitat. Heavy rain can also cause removal.
- Thought should be given to downstream conditions and the potential for the liberated snails to recolonise new habitat.
- Where possible dry out littoral zones to strand snail populations, however take into account the specific ecology and the resilience of the target species.

Chemical control

- Use of molluscicides may cause environmental damage and should be avoided. Use should be targeted rather than wide-spread. Applications are usually restricted to places frequently used by people for swimming, bathing *etc.*

Environmental management – adapting behaviour of people and animals

People:

- *Where possible*, avoid new human settlements near infested wetlands.
- Avoid contact with snail-infested waters. Use water supplied from covered pipes or pit-wells.
- Avoid swimming, wading, washing or bathing in water suspected of infestation. It is safest to consider all freshwater bodies in endemic areas as potential transmission sites if sites otherwise not identified.
- For agricultural workers at constant risk of infection, periodic examination and treatment may be the most feasible approach to disease control.
- Ensure good sanitary practices. A clean water supply and improved sanitation (including on board boats) must be provided to stop human excrement entering wetlands.

Animals:

- Prevent contaminated faeces from livestock entering wetland habitats. This is especially important for species that parasitise animal, livestock and human hosts.

CASE STUDY 3-6. Snail fever integrated control and prevention project in Tongxing Village of Wucheng Township, Yongxiu County of Jiangxi Province, P.R. China, 2007.

Summary

Disease issue or problem:	Snail fever / Schistosomiasis
Action taken:	▪ interruption of cattle-parasite-cycle by means of permanent stabling of cattle (long-term); ▪ awareness raising campaign by carrying out publicity and education activities; ▪ assessment of snail host spatial distribution; ▪ cattle examination and medical treatment.
Outcomes:	Newly reconstructed stables hosting approximately 100 head of cattle. The spread of snail fever in the Tongxing Village controlled and prevented effectively, which saves about 30.000 Yuan each year for human and cattle medical treatment.
What went well:	Participatory approach – stable reconstruction according to local farmers' needs.
Organisations involved:	Promotion Association for Mountain-River-Lake Regional Sustainable Development of Jiangxi Province (MRLSD) and the International Development Research Centre, Canada. Project funded by German Embassy Small Grant and additional contributions from local government on different levels.

Background

The project area is situated in the Tongxing Village of Wucheng Township, located in the Yongxiu County of Jiangxi Province, China. The Wucheng Township lies at the lakeshore of Poyang Lake, covering a total area of 368 km^2, with 47 km^2 consisting of grasslands infested with snail fever. The highest rates of infection with snail fever in the Wucheng population occurred in 1998 with more than 15% of the total population being infected; 10% of these suffered from terminal-stage snail fever. The infection rate among cattle was also elevated, with 71.8% being infected. The highly endemic situation for snail fever is explained by the extensive cattle raising on infested wetlands, maintaining a permanent snail-fever cycle among livestock. As snail fever can equally infect cattle and human beings, the ecological conditions for human infection with snail fever are, therefore, particularly hazardous throughout the entire township.

Tongxing Village is the largest administration village of Wucheng Township. The village-area consists of seasonally flooded wetlands and permanent water. Based on the abundant, grass-covered wetlands, cattle-breeding has become a major activity for local livelihoods. Therefore, the level of infection with snail fever remained alarmingly elevated, seriously hampering local economic development. Indeed, snail-fever was conceived as being a major cause for persisting poverty in the village.

The recent governmental programme of integrated control of snail fever, carried out in the Yongxiu County, prioritises preventative and sanitary measures combined with the development of secure livelihoods and the provisioning of preventive medical and veterinary services. It is well understood by villagers that the cattle-parasite cycle has to be interrupted in order to sustainably reduce the environmental risks of infection with snail fever. One principal intervention strategy is modifying the ongoing practice of extensive livestock breeding aiming at maintaining cattle outside snail fever infected areas (*i.e.* stabling or fencing).

Before the project started, 40 stables for the seasonal stabling of water-buffalos *Bubalus bubalis* already existed in the Paitou sub-village of Tongxing, but most of the stables had collapsed or were damaged. Therefore, they were not suitable for permanently keeping cattle outside the wetlands. The villagers wanted to reconstruct the stables and adapt them for permanent stabling.

In February 2007, the Promotion Association for Mountain-River-Lake Regional Sustainable Development of Jiangxi Province (MRLSD) submitted the Snail Fever Integrated Control and Prevention project in the Tongxing Village to the German Embassy in Beijing. The application was approved in April. The project started in April and was completed by beginning of October.

Activities

Reconstructing existing stables:

- the court, the access-ways, and the interior of the stables cleaned up and stabilised;
- 12 collapsed stables and 28 damaged stables repaired, including their roofs and cracked walls. All stables received painting to protect the outer facade;
- a wall was constructed around the main court covering an area of 1,200 m^2;
- the entrance was stabilised with cement covering an area of 160 m^2;
- sewage disposal ditches were constructed over a total distance of 300 m;
- two concrete ponds for dung disposal constructed with a total volume of 10 m × 5 m × 2 m;
- one sentinel house was newly constructed.

Carrying out publicity and education activities:

The Yongxiu County Snail Fever Control and Prevention Station, the Yongxiu County Agricultural Bureau and the Wucheng Township Government carried out a series of publicity and education activities on snail fever integrated control and prevention in Tongxing Village, by means of training, dissemination of educational materials and posting of pictures. A total of 2,000 people received the education/information.

Conducting investigations of snail hosts:

The Yongxiu County Snail Fever Control and Prevention Station conducted an investigation of snail hosts around the areas of Dahu Lake, Zhushihu Lake, Changhu Lake and Yanzihu Lake, which are frequently visited by the cattle. As result of the investigations, the spatial distribution of densities of the snail hosts was surveyed. The average density of snail hosts in Dahu Lake and Zhushihu Lake is 0.004/m^2, and 0.0097/m^2 in Changhu Lake and Yanzihu Lake.

Conducting cattle examination and medical treatment:

The Yongxiu County Agricultural Bureau provided examination of the cattle in Tongxing Village and free veterinary treatment. A total of 1,060 head of cattle were examined in the village, from which 9.8% were found to be infected. Of the infected cattle, 80% received treatment.

Use of funds

The total budget of the project was RMB 199,680 Yuan, among which 80,000 Yuan was contributed by the German Embassy in Beijing. Additional costs were met by local government.

Conclusions

After the implementation of the project, the newly reconstructed stables were able to house approximately 100 head of cattle. The spread of snail fever in the Tongxing village has been effectively controlled, saving approximately 30,000 Yuan each year in human and cattle medical treatment.

Case study from Mechthild H. Adameit & Martin Wiese,
Promotion Association for Mountain River Lake Regional Sustainable Development (MRLSD)

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3.4.4 Vaccination

Vaccination is used to protect against disease by inducing a level of protection from infection or the progression of disease in otherwise susceptible hosts. Vaccination programmes, supplemented by other disease control measures, can help control and even eliminate epidemic diseases affecting animals and humans.

The role and efficacy of vaccination as a means of control varies amongst diseases. Most commercially available vaccines are targeted against microparasites such as viruses and bacteria. Vaccination against macroparasites such as helminths (where immune responses are often more varied and less effective, and extent of disease is related to parasite burden), are generally less effective. Some vaccines may be highly effective in preventing clinical signs of the disease, preventing infection and reducing growth and shedding of the disease-causing agent.

Other vaccines may prevent clinical disease but not prevent infection or transmission. In some cases, vaccination may not be effective, may only reduce the severity of the disease or may offer different levels of protection depending on host age. Therefore, when deciding whether or not to include vaccination as part of a disease control strategy, a thorough knowledge of the characteristics of the disease agent and its epidemiology, and the suitability of available vaccines, is required. It is worth considering that outcomes of vaccination programmes are not always certain and success in one target population may not necessarily translate to elsewhere under different ecological, genetic or environmental conditions.

Humans at risk of exposure to zoonotic diseases, should seek advice about vaccination options from the appropriate health authorities.

The following issues should be evaluated before selecting an animal vaccination strategy:

Vaccine type

There are different types of vaccines and the advantages and disadvantages of each should be evaluated. Live attenuated vaccines often provide a longer lasting immunity. Some live vaccines can be administered using methods that involve little or no handling of animals, *e.g.* by using oral-baited vaccines, although more than one dose is often required. Inactivated (killed) vaccines should be safe in all circumstances.

To achieve adequate levels of immunity for epidemic livestock diseases where the disease-causing agent exhibits antigenic variation (*i.e.* exists as different types or is prone to change and/or mutation), it is important to select the correct antigenic type and subtype vaccine. Isolates of the agent should be regularly collected from a wide geographical spread, and submitted for reference laboratory analysis so that the appropriate vaccine strain can be selected.

All vaccines must be thoroughly tested on the target animal species to establish safety. Appropriate licences for target animal species may be required which can be particularly challenging and time-consuming to achieve for wildlife species.

Vaccine quality

Vaccines should be sourced from manufacturers who follow internationally accepted quality assurance procedures and codes of good manufacturing practice. The manufacturers should be subject to approval and quality control verification by independent national or international biological control authorities. Appropriate storage is essential for maintaining vaccine quality.

Vaccine protection

Not all vaccines protect animals from infection, although the prevention of disease progression may be sufficient to reduce transmission and hence aid in controlling disease spread. The pathogen will continue to circulate amongst unvaccinated individuals, but the purpose of a vaccination programme is to deliver the vaccine to a sufficient proportion of the population to enable an overall reduction in levels of transmission. Consequently an effective vaccination campaign will confer benefits even to the unvaccinated proportion of the population (often referred to as 'herd immunity').

The effectiveness of a vaccine in a given population is a function of the efficacy of the vaccine (*i.e.* the likelihood that it benefits an individual) and the proportion of the population to which it can be delivered. The level of vaccination coverage required to achieve disease control benefits will vary between host and pathogen populations. Sustained effort will be required in order to maintain the benefits of vaccination in the face of sources of re-infection (*e.g.* unvaccinated parts of the same population, other host species or environmental contamination).

Ongoing surveillance is, therefore, an important tool for monitoring the progress of vaccination programmes. Not all vaccines deliver life-long immunity and in some cases periodic re-administration may be required to deliver disease control benefits.

Vaccination and disease surveillance

Vaccination programmes may interfere with disease surveillance. For example, clinical surveillance may be more difficult in populations with a mixture of vaccinated and unvaccinated animals, as the disease may be unevenly distributed. Many serological tests cannot distinguish between antibodies that have been derived from vaccination or from natural infection, although some differential diagnostic tests do exist or may be developed. Interpretation of serology results can be greatly assisted by marking vaccinated animals, so that it is at least known whether samples have been taken from vaccinated or non-vaccinated animals. This may also be important to avoid the adverse welfare and financial implications of over-dosing individuals.

Vaccination storage and application

Vaccines should be stored at the correct refrigeration temperatures at all times and must be used before expiry dates. Vaccination teams must include personnel trained in administering vaccines.

Checklist 3-4 provides a summary of the principal considerations when selecting a vaccination programme.

CHECKLIST 3-4. Selecting a vaccination programme
When selecting a vaccination programme, the following should be considered:
☐ The programme should have a clear purpose and objective
☐ Once the target animal population and area have been defined, vaccination should be carried out as comprehensively as possible
☐ Separate vaccination personnel should be used for herds and flocks thought to have infection to minimise the spread of the disease between them
☐ Individual herds and flocks should be gathered separately to minimise the spread of disease
☐ Vaccinated animals should be permanently marked for future identification
☐ Vaccination programmes should be accompanied by other measures such as disease surveillance, livestock movement controls and quarantine (where possible and appropriate)
☐ Vaccination programmes should be accompanied by public awareness campaigns

Examples of vaccination programmes:

1. **Blanket vaccination** is the comprehensive vaccination of ‘all’ susceptible animals over a large area. This may be favoured when the disease has become well established, when there are many sources of infection, or when other disease control measures are impractical and/or ineffective. Areas with known and suspected infection and areas thought to be at high risk of disease should be covered. Several rounds of vaccination over several years may be required.
2. **Ring vaccination** is the rapid creation of a belt of vaccinated animals around an infected area. This can be implemented to contain a fast spreading disease outbreak, in situations where the effectiveness of other methods is unlikely to succeed, or in areas which are too inaccessible for blanket vaccination or other disease control measures. Epidemiological factors and resource availability should be assessed to determine the width of the vaccination zone. Vaccination should be completed within a short period of time, *e.g*. a week, and start at the outer edges of the ring, moving inwards towards infected animals. A second outer ring can be created if necessary [►**3.2.2 Disease zoning, barriers and buffer zones**].

Specific considerations for vaccination of wildlife

Vaccination of domestic livestock has been widely used and may often present a practical disease control option where an effective vaccine exists. Vaccination of wildlife is more challenging owing to many technological and logistical barriers including difficulties in delivering it to a sufficiently large proportion of the target population. Also, only few vaccines have been tested sufficiently to demonstrate their safety and efficacy and achieve a licence for their use in wild hosts. Even domestic animal vaccines against the same pathogen, may need to undergo significant testing to determine their safety and efficacy in wild hosts. These factors may rule vaccination out in many circumstances, although the approach has met with some success in controlling rabies and classical swine fever in wildlife populations in recent decades.

The aim of any wildlife vaccination programme needs to be clear from the outset, for example, does the vaccination programme aim to reduce mortality, reduce suffering, reduce the risk of spread to livestock or humans, or to ensure the viability of the population?

There may be risks associated with the vaccine itself, either in target or non-target populations. Live vaccines have the greatest potential for problems following release into the environment. Also, the ecological consequences of vaccination should be considered, including the possibility of altering demographic processes (*e.g.* survival rates, population growth rates).

Delivery of the vaccine to the target population may be logistically difficult or prohibitively expensive. Methods of vaccine delivery include the injection of captured animals and the deployment of palatable baits containing vaccine. Capture and injection options are likely to be relatively expensive and could have adverse welfare implications. Deployment of edible baits is often a more attractive option, but the development of a suitable bait which is compatible with the vaccine and sufficiently stable in the environment can be technically challenging.

Some well-resourced wildlife vaccination programmes such as rabies vaccination for red foxes *Vulpes vulpes* in Europe have proved successful. Other successful projects have involved vaccination of endangered wild populations against domestic animal diseases for which vaccines already exist, where populations were relatively restricted in range and well studied, and the aims of the project have been clear.

Vaccination of wildlife can be successful and may seem like an appealing option, however, other management techniques, particularly where naturally acquired immunity is developed, may be just as effective and in many ways preferable.

CASE STUDY 3-7. Buffalo treatment campaign in Iraq

Breeding marsh buffalo *Bubalus bubalis* is important in different parts of Iraq, particularly in its southern regions and wetlands such as the Central marsh due to the abundance of appropriate food, water and pasture land. Unfortunately, many by-products of modern technology and poor water management policies have damaged the natural environment of these areas.

This in turn necessitates the existence of veterinary centres to provide the proper treatment and vaccines needed for healthy buffalo populations. Due to an apparent lack of training and proper supplies, there is the potential for these centres to spread and worsen some diseases that afflict buffalo and cattle, such as septic blood haemorrhages and other diseases. These diseases lead to substantial losses in livestock, so consequently the authorities have instituted serious measures with the close support of Nature Iraq, an Iraqi environmental organisation, to contain these diseases through a campaign for fast and effective treatment of haemorrhagic blood septicaemia and other diseases.

Main diseases that afflict buffalo:

- Haemorrhagic septicaemia
- Symptomatic anthrax

The focus of this report is the prevention of haemorrhagic septicaemia. The following are the vaccines used in the prevention of this disease:

- Haemorrhagic Septicaemia Vaccine (H.S.V.)
- Blackleg Disease Vaccine (B.L.V.)

To make the combined vaccine, the two vaccines (H.S.V. with B.L.V.) must be mixed together and then it is ready for injection into the buffalo.

Haemorrhagic septicaemia

This is among the most common diseases infecting buffaloes throughout Iraq as well as in other African and Asian countries. After 13 years of two epidemiological studies in India, this disease was determined to be the more deadly than diseases such as cow plague, foot and mouth disease and symptomatic anthrax.

This disease appears along with seasonal increases in wind and heat. It is caused by the bacterium *Pasteurella multocida* and it is pathogenic in cows and deadly for buffaloes.

Infection

Cows and buffaloes which carry the disease are considered the main source of the disease, which can exist inside the mouth of other nearby animals that can infect them directly or indirectly.

The infection can be transmitted by breath or swallowing the germ. The high rate of infection is closely tied to the animals' wetland habitat and the close quarters the herds experience at night inside their enclosures.

Clinical signs

The infected buffaloes can be recognised by sluggishness, lack of movement, salivation, increased temperature, difficulty breathing, breathing through their mouth, nose excretions, and throat or neck lesions sometimes extending to the chest, as well as fluid in the throat and lungs.

Vaccination

Allergy testing is necessary, as are vaccine-resistant strains of the disease. The vaccination should also vary according to local conditions in various countries but it is essential that the vaccination must begin early, as soon as the disease is detected.

There are methods to help buffaloes survive the disease by making a slot in the trachea of the animals to give more time for the vaccine to work. It is possible to inject the animals intravenously whilst executing this minor surgical procedure at the same time by using anaesthetic.

In this project, work continued for a period of forty-eight days during which time 18,331 buffalo and 1,229 cows were treated in several regions of Thi Qar province, as shown in the following table.

District & sub-district	Number of vaccinated buffaloes	Number of breeders	Number of vaccinated cows
Suk Ash-Shuyook	6448	412	-
Al-Taar	1488	62	-
Al-Aslah	1479	51	846
Al-Cidaynoweya	617	28	-
Al-Fuhood	2232	81	-
Al-Chibayish	3783	252	60
Al-Hammar	1290	44	85
Karamatt Bani Saeyid	994	81	238

Results

The following results were obtained from the vaccination campaign:

- Improved conditions and help in controlling haemorrhagic septicaemia in the visited villages;
- Increased health awareness of Iraqi buffalo breeders;
- Creation of a trusting relationship between the citizens and Nature Iraq;
- Motivated the veterinary centres in Thi-Qar to contribute to increasing veterinary awareness for the people;
- Stopped the disease's migration from an infected area, and entrusted stewardship of the environment to the local people.

Case study from Al-Asadi A & Talib M. 2010. Buffalo Treatment Campaign. Nature Iraq Report, Pub. No: NI-0610-01. Thi-Qar Veterinary Hospital & Nature Iraq Chibayish, Iraq.

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3.4.5 Genetic manipulation

Altering the genetics of a host, by whatever means, can provide a way to reduce susceptibility to disease. For both wildlife and indigenous breeds of livestock, natural selection for genetic resistance to pathogens occurs over time, and, generally where the relationship between host and pathogen is well established, a balance is acquired.

Selective breeding has traditionally been achieved by cross-breeding two individuals, each possessing a favourable trait, to obtain one offspring with both. This is normally a lengthy process requiring many breeding cycles to eliminate undesirable traits that may also be inherited. Creating genetic resistance in hosts or using genetic techniques for other forms of disease control can be achieved more quickly using genetic manipulation/modification (GM) techniques.

GM is a biotechnology that involves the modification of the genes of an organism to produce a desirable trait, the altered organism is termed a genetically modified organism (GMO). Genetic manipulation allows the desired genes (and therefore traits) to be spliced directly into organisms and removes the randomness of sexual genetic recombination making the breeding process more targeted and efficient.

In the majority of cases, both old and new, methods of genetic manipulation are an impractical option for wetland diseases, nonetheless, both selective breeding and genetic modification (GM) are having an impact on some important diseases. These techniques have the potential to provide powerful and sustainable solutions to global health problems because they are effective, specific and can reduce pesticide use, which in turn reduces resistance and environmental damage. GM is, however, controversial as it raises concerns about risks of unintended and widespread impacts on the environment, *e.g.* transgenes moving between species or new insects or diseases filling niches once occupied by the eradicated/suppressed insect, potentially causing greater problems. Nonetheless, the huge potential for increased productivity and improved health benefits has resulted in continued development in the use of GM for disease management.

Genetic manipulation of hosts and habitats

Where disease resistance is known to have a genetic basis, traditional selective breeding can be used to generate resistant individuals and much research to date has focussed on breeding host individuals with immunity to a disease.

For domestic animals, in general, indigenous breeds of livestock will have greater resistance to endemic diseases and may, thus, provide a sound way to control diseases and maintain productivity. As an example, indigenous, trypanotolerant ruminant livestock (*e.g.* N’Dama and West African Shorthorns, as well as Djallonke sheep and goats) are useful in the control of African animal trypanosomiasis. Naturally resistant breeds are able to maintain productivity even in the face of disease risks and do not require expensive veterinary treatments, nor the costs (financial and environmental) of using chemicals for control of vectors.

For wildlife, genetic manipulation or selective breeding, is a subject for debates in environmental ethics, however, in the face of a particular threat from a pathogen causing serious impacts it provides a potentially practical solution. Selected individuals may then be used to repopulate a habitat that has already been adversely affected by a pathogen. For threatened species a successful *selective* captive breeding programme would offer a ‘safety net’ immune population that could be used for re-introductions should the species succumb to the pathogen (*e.g.* amphibians under threat from chytridiomycosis).

Genetic modification of plants can both increase their own disease resistance and bring broader health benefits. Wetland grasses and other monocotyledons are important natural remediators of pollutants, and through genetic modification researchers have demonstrated an ability to enhance performance in the metabolism of trichloroethylene and the removal of a range of other toxic volatile organic pollutants, including vinyl chloride, carbon tetrachloride, chloroform and benzene.

CASE STUDY 3-8. Dieback in Western Australia

Phytophthora cinnamomi (responsible for the disease dieback) is a destructive and widespread soil-borne pathogen that infects the roots of woody plant hosts. In Western Australia (WA) the disease has had devastating effects on native ecosystems: 40% of the 5710 known native species of WA are considered susceptible. Naturally occurring, genetic-based resistance to *Phytophthora cinnamomi* has been demonstrated and researchers are selectively breeding for resistant individuals. Resistant jarrah plants have been micro-propagated by tissue culture and clonal lines are being used for field trials and to repopulate dieback-decimated forests.

Genetic manipulation of vectors

For vector-borne disease management it is often favourable to target vector populations to break the life cycle between host and pathogen. The sterile insect technique (SIT) is a method which introduces sterile males into a vector population to compete with wild males reducing the population of the next generation. Historically, radiation had been used to sterilise males, which led, for example, to the successful eradication of the screwworm fly *Cochliomyia hominivorax* on the island of Curacao in the 1950s. A disadvantage of irradiation is that females often will not mate with the irradiated males. Modern GM techniques have offered improved options for SIT because GM insects have been shown to compete effectively with wild counterparts.

As vectors for globally important human diseases such as dengue fever and malaria, mosquitoes have been the target of a substantial body of research [►**Case study 3-9. The genetic manipulation of mosquitoes**]. There has also been development on GM-based control of the tsetse fly, the vectors of African human and animal trypanosomiasis. Research is demonstrating the potential to produce tsetse fly populations resistant to the trypanosome parasite by genetically modifying the symbiotic bacteria, which are passed down by the mothers and reside in the gut of the fly, to inhibit the trypanosome parasites.

CASE STUDY 3-9. The genetic manipulation of mosquitoes

The genetic modification of mosquitoes to produce sterile males was trialled in the Cayman Islands in 2009 where the *Aedes aegypti* mosquito is a vector for the human viral disease dengue fever. The modification allows GM males to mate with normal female mosquitoes, however, any offspring of GM males will only live up to the pupae stage. Following the release of batches of GM male mosquitoes, the GM insects made up 16% of the study population and went on to father 10% of the wild larvae and hence cause a 10% reduction in the progeny. The work demonstrated the potential for this technique to bring about vector population reductions even if GM insects are not equally as competitive as wild mosquitoes.

Other research projects are tackling the problem in different ways: one group has engineered *Anopheles* mosquitoes to be immune to the malaria parasite they normally carry; another has manipulated male *Anopheles* to produce no sperm; whilst others have modified the insect to produce flightless female progeny.

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3.4.6 Habitat modification

The Ramsar Convention Secretariat provides clear guidance on wetland management and what constitutes 'wise use'. Whilst operating within this framework, habitat modification in wetlands can eliminate or reduce the risk of disease, by reducing the prevalence of disease-causing agents, vectors and/or hosts and their contact with one another, through the manipulation of wetland hydrology, vegetation and topography.

Modifications to habitat features can help reduce the capacity of the local habitat to maintain populations of disease-carrying vectors through reducing vector breeding sites and encouraging vector predators [►**Section 3.4.3 Control of vectors**]. Such measures are often preferable to more environmentally damaging biological and chemical control methods. Habitat modification can also reduce the likelihood of exposure of disease-causing agents such as species of bacteria and toxic algae and other contaminants although this technique is more often directed at hosts and disease vectors than at the causative agents. Measures can alter or reduce host distribution and density and may be used to disperse and encourage hosts away from outbreak areas.

Maintaining 'healthy' naturally functioning wetlands is generally important for reducing the risk of disease. Damaged or degraded wetlands can result in poor water quality, reduced water flows and vegetation growth, features which provide ideal habitat for some disease-carrying vectors and may act as stressors for hosts. However, some characteristics associated with naturally functioning wetlands, such as good water quality and flow, may also directly encourage vector and host populations. It is therefore important to assess both the potential risks and benefits of wetland modification in reducing the risk of disease in light of the specific habitat requirements of the pathogen, vector and host. For invertebrate disease vectors and hosts, for example, measures will often depend on the specific environmental requirements of the aquatic life stage of the species.

Effective management of wetland habitats requires a thorough understanding of wetland ecosystem functions of the inter-connected hydrological, geomorphological, biochemical and ecological components, as changing one parameter can have implications for another. Important processes include flow regimes, water level changes and flood inundation, and their effects on vegetation and sediment and the requirements of wetland fauna. The effects of habitat changes on predator populations should always be considered when determining habitat modification measures. As long as undertaken in the context of the wetland management plan, the following alterations to wetland hydrology and vegetation (often through changes to topography) can be used to reduce the risk of disease spread in wetlands.

Altering wetland hydrology

Altering the extent of inundated and saturated areas

Wetland systems can be modified to alter the extent of an inundated and saturated area and hence available habitat for disease agents, vectors and hosts. A reduction in the extent of an inundated and saturated area will lead to a decrease in the abundance of some vectors and hosts (*e.g.* certain mosquito and snail species), particularly if other environmental parameters such as water flow and quality are favourable. However, this is accompanied by an inevitable loss of valuable wetland services and therefore any adverse impacts on wetland ecosystem function should be carefully examined before such actions are taken. Measures used for decreasing an inundated and saturated area include draining and infilling, and water control structures such as pumps, which must be carefully sited to minimise disturbance to wildlife.

Changes in habitat characteristics may benefit one host population, whilst disadvantaging another. For example, certain obligate freshwater snail hosts may decrease in number after the reduction of an inundated and saturated area, whilst some mosquito species favour smaller isolated pools, created after infilling or draining.

Altering water flow patterns
Altering the water flow may change the retention time of water within the wetland and affect several key characteristics such as water quality, retention of flood-flows and vegetation, in turn affecting the habitat's suitability for hosts and vectors. Alteration of water depth, for example, may change the extent of emergent macrophyte beds, manipulation of which can be used to minimise certain vector and host species. Reduced water depth and flow rates may cause decreased turbidity, and increased water temperatures in warmer weather, but can decrease temperatures in colder weather, influencing the distribution of some aquatic vector and host species, such as snails. Measures to alter water flow include changing the dimensions, gradient and features of water channels.

Altering water quality
Water quality may affect disease agents, hosts and vectors, primarily through changes to vegetation and water flows [►sections above and below]. Land-use in and around a wetland substantially influences water quality. Activities that generate high inputs of organic matter and pollutants to a wetland, such as intensive farming and industry, can be reduced to improve water quality, and piped inflows from potentially polluted sources can be routed away from the wetland system.

Altering wetland vegetation

The type and biomass of vegetation can be modified to reduce suitability for vectors and pathogens and availability of contaminants either through direct action, such as planting, or through the secondary effects of altering other wetland features such as hydrology. Emergent vegetation is known to have a deleterious effect on important disease vectors such as the tsetse fly *Glossina spp.*, and some mosquito populations by obstructing oviposition and supporting a greater diversity of aquatic predators. Vegetation can also provide protection for the larvae of other vectors from predators, causing an increase in their populations and enhancing disease risks. Vegetation may be used to improve water quality and reduce sediment load through filtering organic outflows. Ecological buffer zones can be created for these purposes [**►Section 3.2.2 Disease zoning, barriers and buffer zones** and **►Section 3.2.4 Biosecurity**].

Fire may be used to burn areas where certain disease agents occur, such as the burning of anthrax outbreak areas to destroy the bacterium and burning selected trees to reduce certain species of tick.

Altering topography

The shape, height, depth and profile of the land surface can be physically modified to reduce attractiveness to vectors and exposure to disease agents. This can be achieved through modifications to vegetation and hydrology [►sections above] and by using other mechanical methods such as removing the top layer of contaminated soil to reduce exposure of a disease agent or reducing the number of isolated, stagnant, shallow water areas to deter disease vectors such as mosquitoes from laying eggs.

Figure 3-16. Replacing topsoil on an island used by high densities of birds in the winter helps to reduce environmental contamination and can be useful for small areas of land.

Altering host distribution and density

Habitat modification by the methods outlined above, may also be employed to disperse host animals away from known disease sites and encourage them to use areas of lower risk. For example, waterbirds can be redistributed to lower risk areas by lowering the water level of contaminated areas whilst creating or enhancing other habitats. Outbreak/contaminated areas may be fenced and other measures such as fire and scare devices may be used to deter animals from those areas and separate livestock from wildlife disease reservoirs and *vice versa*.

The provision of more favourable habitat at a distance from an outbreak/contaminated area may encourage animals away from those areas and thus reduce risks of further disease spread. Habitats can be modified to prevent large host die-offs, whose carcases could become substrates for the growth of disease-causing agents. For example, raising water levels in warm, dry weather may prevent the death of bacteria-harbouring fish and aquatic invertebrates.

Compensatory habitat restoration

Although measures taken to modify habitats should not, in so far as possible, impact adversely on the wetland ecosystem as a whole, this may be unavoidable in certain circumstances where options are limited and the potential impact of disease is severe. Under these circumstances compensatory habitat restoration should, wherever possible, be undertaken. This may involve habitat restoration, creation or enhancement with the aim of compensating for lost habitat.

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3.4.7 Movement restrictions

The movements of wildlife and domestic animals may facilitate the spread of disease through the introduction of pathogens to formerly disease-free areas. Restrictions on the movements of domestic animals and people, usually imposed by government authorities, can therefore be an effective tool for preventing and controlling disease transmission by reducing contact between infected and susceptible animals.

Such measures are particularly useful in wetland sites with substantial human activity, such as human residencies, intensive livestock production, large numbers of visitors or hunters, captive breeding and/or translocation programmes.

Movement restrictions to prevent an outbreak

Preventative measures may be taken as a response to periods of elevated risk of an outbreak affecting a wetland. In the event of a disease outbreak near to a wetland or at a national level, implementation of animal movement restrictions may be considered a prudent measure. Where a disease outbreak is considered serious, national stock 'standstills' may be imposed which restrict all animal movement. It is also important to note that movements of people may also be restricted to and from a wetland. Trade in animals and derived products may also be prohibited locally, nationally or internationally.

Movement restrictions to control an outbreak

Rapid notification of the presence of disease by wetland managers is vital for the timely mobilisation of control activities. The overall cost of a disease management strategy may be reduced if disease is prevented or controlled at an early stage during the outbreak, and economic impacts related to restricted animal trade will be minimised. If a notifiable disease is confirmed in domestic animals and/or wildlife at a wetland site, there are likely to be automatic movement restrictions placed on people and animals by government authorities to reduce the risk of further spread. During such an outbreak stock must not be moved within or external to the site until restrictions are lifted: contravention of statutory movement restrictions can result in criminal prosecution. The site contingency plan should be implemented and personnel guided through the process in the event of a disease outbreak [►**Section 3.1.4 Contingency planning**].

Controls may be implemented whereby movements of susceptible species are only permitted under strict, designated conditions, when it is deemed safe. There may be restrictions on gatherings of susceptible animals (*e.g.* at livestock markets) or the transport of animals directly to abattoirs for immediate slaughter for animals with diseases that are transmitted by meat or other animal products. When such activities are allowed to resume, they should be subject to surveillance and rigidly enforced codes of practice. If area restrictions have been imposed on a site, visits to other wetland sites or areas with livestock should only take place if they are essential and should be subject to strict biosecurity measures [►**Section 3.2.4 Biosecurity**].

Until a disease outbreak is brought under control, rights of way through the infected area should be closed and non-essential visits to infected sites should be suspended. To remove the source of infection and to help eradicate the pathogen, destroyed animal carcases and related products including contaminated fomites, should be disposed of in the appropriate manner [**►Section 3.4.2 Collection and disposal of carcases**] and generally should not be moved off, or within, the site.

Infected or potentially infected sites, animals and their products, personnel, potentially contaminated animal products and other materials may be placed under **quarantine**. Appropriate health restrictions can be placed on the movement of susceptible animals into, or out of, the quarantine area until the infection is considered to have been removed. This may be supported by disinfection and decontamination of personnel, vehicles, equipment and other materials leaving and entering the quarantine area [**►Section 3.4.1 Disinfection and sanitation**, and **►Section 3.2.4 Biosecurity**]. Quarantine guidelines vary depending on the case and factors involved (disease, terrain, local human and animal populations) but will generally cover at least a 3-5 km radius from the initial case.

Movement restrictions are often imposed over a wider area around the quarantined or infected site as part of a **zoning** strategy which seeks to identify disease infected, disease-free and buffer zone areas [**►Section 3.2.2 Disease zoning, barriers and buffer zones**]. The coverage of the outbreak area and surrounding areas of risk can be determined from surveillance activities and relies on an understanding of the epidemiology of the disease and host ecology [**►Section 3.3.1 Surveillance and monitoring**]. Animal movement tracking may help identify the source of disease [**►Section 3.2.3 Standards for releasing and moving domestic animals or translocating wildlife**].

A number of control zones may be set up around infected premises. Animal movement within identified zones is not permitted unless appropriate permits have been issued by the local authorities. Trade in certain animals and their products may be permitted under particular circumstances from disease-free zones but only where this has been authorised. Controlled area restrictions may apply whereby the movement of animals outside the protection and surveillance zones is controlled. This is advisable when there is a risk of the disease spreading more widely (*e.g.* if an infected animal has passed through a market).

Imposed movement restrictions and other disease control activities should be communicated promptly and clearly to relevant stakeholders and local communities by local authorities [**►Section 3.5 Communication, education, participation and awareness**]. An integrated disease management strategy, which includes a range of disease control activities such as movement restrictions, zoning, surveillance and vaccination, is often most effective. A disease management strategy for the site should incorporate how best to respond to and cope with movement restrictions. Consideration should be given to voluntary implementation at times of increased risk (*e.g.* suspension of hunting activities or site visits) if a disease has emerged within a region. It should be noted that long term restrictions will affect commercial enterprises and so consideration should be given to incorporation of a business continuity plan into the site contingency plan.

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3.4.8 Eradication, elimination, stamping out and lethal intervention

Global disease eradication

Global eradication is used to refer to the permanent reduction of the worldwide incidence of a disease to zero as a result of deliberate efforts, eliminating the need for further disease control measures. This has been achieved for smallpox in 1979, and, more recently, rinderpest in 2011 [**►Case study 2-1. Rinderpest – eradication of a disease affecting all sectors**]. Successful eradication programmes produce sustainable improvements in health and many other benefits but depend on significant levels of global co-operation in the sustained and co-ordinated control of infection, usually requiring a combination of approaches.

An eradication programme will not succeed in the absence of a sound scientific basis, availability of sufficient resources and public and political will. International coordination and collaboration with regional and national governmental, and non-governmental organisations is essential for the control and eradication of transboundary animal diseases.

Disease elimination

Elimination of a disease usually refers to the reduction to zero of incidence in a defined geographical area as a result of deliberate efforts. Examples include the successful elimination of polio in the Americas and of neonatal tetanus in 19 countries between 1999 and 2010. Importantly, unless the disease can be globally eradicated, continued disease control intervention measures are needed to prevent re-emergence.

Disease elimination in wetlands poses a number of problems particularly in relation to wildlife diseases and water-borne infectious agents. The following measures can aid disease elimination and their merits should be considered within any disease control strategy:

- Identification of infected zones through intensive disease surveillance [**►Section 3.3.1 Surveillance and monitoring**].
- Designation of infected zones [**►Section 3.2.2 Disease zoning, barriers and buffer Zones**].
- Imposition of quarantine and livestock movement restrictions [**►Section 3.4.7 Movement restrictions**].
- Possible slaughter of infected or susceptible animals using a range of methods [**►Stamping out and lethal intervention**].
- Vaccination of susceptible animals [**►Section 3.4.4 Vaccination**].
- Safe disposal of carcases and other potentially infectious materials [**►Section 3.4.2 Collection and disposal of carcases**].
- Disinfection and cleaning of infected premises [**►Section 3.4.1 Disinfection and sanitation**].
- Ensuring that the infected area is free of susceptible animals for an appropriate period of time.

Stamping out and lethal intervention

'Stamping out' is a term often used to describe the localised destruction of susceptible or infected animals. The most appropriate use of this approach at a wetland site would be for the rapid elimination of a disease in livestock. Lethal methods include dispatch by firearm or captive-bolt, the use of gaseous, biological or injectable agents.

Stamping out may often be a cost-effective approach to disease control in livestock in an emergency situation, as in appropriate circumstances (*e.g.* in the absence of an external source of infection) it can have rapid results. This allows restrictions on trade and other animal movements, *e.g*. restocking, to resume more quickly.

As with all disease strategies, the scientific feasibility, and health, ethical, social and economic costs and benefits of stamping out and lethal intervention should be carefully evaluated before it is selected as a disease control strategy. The likely success of alternative strategies should also be considered. Lethal intervention has been used for disease control in wildlife, but in wetland sites this may not be consistent with conservation objectives. Hence, the potential costs and benefits of lethal interventions need to be considered carefully. This requires some knowledge of the likely behavioural and demographic responses of host populations to lethal control as these can result in complex outcomes in terms of disease control. Selective culling may be an appropriate approach in some circumstances. However, implementation at the level of individual hosts requires the availability of adequate diagnostic tools, and at the population level it is important to be able to accurately identify the target population.

Lethal interventions of invasive alien species or pests is likely to be consistent with conservation objectives but, nevertheless, a sound understanding of the response of the target population is required prior to intervention to help predict impacts.

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3.5 Communication, education, participation and awareness (CEPA)

Well planned, targeted and resourced CEPA programmes for wetland stakeholders are essential for raising awareness of wetland diseases and the measures that can be taken to successfully prevent, detect, control and mitigate disease outbreaks, in particular the basic principles of 'healthy habitat' management. These programmes form some of the most fundamental aspects of managing diseases in wetlands and should be included in all wetland disease management strategies.

Successful communication relies upon establishing a regular dialogue between wetland stakeholders and disease control authorities. A 'culture' of disease management can only be developed if: a broad range of wetland stakeholders (*e.g.* local wetland users, local government agencies, community leaders, hunters and NGOs) participate in these programmes; information between and within stakeholder groups flows regularly; and communication, awareness raising and educational activities are on-going or at least 'refreshed' periodically.

This section contains further information on the following topics:

- *Communication and public awareness*
- *Building capacity by education and training*

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- **Well planned, targeted and resourced CEPA programmes** for wetland stakeholders are **essential for raising awareness and appreciation of wetland diseases** and the measures that can be taken to successfully prevent, detect, control and mitigate disease outbreaks. Such programmes should be integrated into all wetland disease management strategies.
- **Programmes should aim to inform** wetland stakeholders of **the basic principles of healthy habitat management,** thus reducing the risk of a disease outbreak.
- **A 'culture' of proactive disease management** can only be developed if a broad range of **wetland stakeholders participate in CEPA programmes**.
- **Communication strategies should aim to make stakeholders aware of the nature and potential consequence** of animal disease and of the benefits gained from prevention and control measures. They should **ultimately encourage people to take the recommended courses of action** in preventing and controlling a disease outbreak. Awareness raising campaigns should emphasise the importance of early warning systems and of notifying and seeking help from the nearest government animal and/or human health official as soon as an unusual disease outbreak is suspected.
- **Selection of the appropriate message, the messenger and the method of delivery is critical for successful communication.**
- **A strategy**, written in 'peacetime' f**or dealing with the media can increase likelihood of successful outcomes** from this relationship maximising potential benefits and minimising potential negative impacts.
- **Simulation exercises and testing of contingency plans are a valuable method for training**.

3.5.1 Communication and public awareness

Communication programmes and activities can raise awareness of diseases, the risks they pose, and the measures that can be taken to prevent, detect, control and mitigate outbreaks [**►Case study 3-8. Snail fever integrated control and prevention project in Tongxing Village of Wucheng Township, Yongxiu County of Jiangxi Province, P.R. China**]. Such programmes are one of the most critical aspects of managing disease in wetlands, and engender a 'bottom-up' approach. A 'culture' of disease management can only be developed if a broad range of wetland stakeholders participate in these programmes.

Target audience

Communications and awareness raising materials should be targeted at those likely to affect potential for disease emergence, those likely to be affected by disease or come into contact with it, and to those whose activities may influence its prevalence and spread, such as local authorities, people living in 'high risk' areas, farmers and livestock owners and traders. Each different group is a specific audience and communications need to be tailored appropriately.

The message

It is important to consider the intended audience for your message when writing/determining its content. Be mindful of the key purpose of the message whilst considering your target audience's education, socio-economic status, current knowledge and experience of the issue/disease, age, language, culture and geographic location. The audience will determine the length, content and style of the message. Given the multiple benefits of disease control, there can be an element of rallying the community to a common cause, ideally involving local community groups, key land users and farming organisations where appropriate.

Messages need to be communicated clearly and simply and with credibility, accuracy, consistency and speed.

An effective message should be:

- repeated
- come from a trusted, credible and legitimate source
- be specific to the event being experienced, and
- offer a positive course of action.

Materials and services

Messages can be communicated using various materials and services. These include signs, printed materials, the internet, media coverage, public service announcements, national campaigns, audio conferences, seminars and workshops. Ways should be considered for the audience to submit information or ask questions too, for instance by giving a phone number or email address: they may be your eyes and ears on the ground.

Partnerships with agencies and organisations that have relationships with the target audience, or are otherwise recognised as community leaders (*e.g*. religious leaders, service groups), should be fostered.

It is valuable to find out how a target audience likes to receive information, such as, online, on the radio, on a notice board, in the newspaper. Communicating through sources trusted by the target audience can heighten the credibility of, and attention to, messages.

Figure 3-17. Engaging the public in disease control: the public information sign on a trail in Victoria, Australia, outlines the problem, provides a positive course of action and provides contact information for further communication.

The media

The media, such as television, radio, newspapers and online news sources, can help get a message to a large number of people quickly and easily. When working with the media, be aware that control of the message can be lost. There is a greater chance that it may be edited or misinterpreted. Developing partnerships or good relationships with local or national media can reduce potential for misunderstanding.

Ideally, selected personnel should receive media training and be designated spokespersons on behalf of an organisation involved with managing disease, to effectively convey information before, during and after an outbreak or other problem. ►**Disease outbreaks and the media**

Community outreach and mobilisation

Community outreach involves presenting messages in person, in addition to media and educational materials, and cultivates community ownership for disease prevention activities. A community task force that includes health, environmental, civic and business concerns can be valuable in reaching various segments of society and in developing a common message. Community outreach activities should encourage community mobilisation whereby groups take part in actions to prevent and control an outbreak, *e.g.* community efforts to improve water sanitation and reduce pollution risks.

Target audience research

Knowing different audiences is critical to putting communication plans into practice. Attitudes to disease management measures may vary considerably by region or section of society. Previous experience with disease prevention and control measures will affect the acceptability of future efforts. Target audience research can identify local attitudes, motivations, barriers to 'change', and opportunities to promote desired behaviours. Surveys assessing knowledge, understanding, attitude and practice levels can be of particular value - ideally combining qualitative and quantitative methods. Evaluations, including lessons learned, should be conducted, whenever possible, to measure the efficacy of communications in achieving their aims, and adjustments made accordingly.

Emergency communications for a disease outbreak

Emergency communications are inevitably focused on managing for the worst case scenario. This is where planning will be invaluable. Above all, a communication plan is a resource of information for those that need it and should be integrated into the overall wetland disease management strategy. All relevant wetland stakeholders, disease control authorities, spokespersons and communications professionals should be involved (*e.g*. wetland users, animal and human health agencies and governmental authorities), key messages should be clear and understood by all, and resources should be shared.

Overall a communication plan can be broken down into:

1. **Crisis Communication:** this is used when there is an unexpected disease outbreak and there is a need to quickly communicate about that crisis to wetland stakeholders and the wider public.
2. **Issues Management Communication:** this is used with the knowledge of an impending crisis and, therefore, the opportunity to choose the timing of the communication to the wetland stakeholders and the wider public.
3. **Risk Communication:** this is used to prepare people for the possibility of a disease outbreak and to provide appropriate steps to prevent an outbreak and mitigate for its impacts.

It is important to understand the stages of an unfolding disease outbreak in order to help communicators anticipate problems, anticipate the information needs of disease control authorities, wetland stakeholders, the media and the general public, and therefore respond effectively. There will be stages to every outbreak and communication must also evolve with each stage. The following cycle demonstrates the likely stages of an outbreak:

Precrisis	Initial	Maintenance	Resolution	Evaluation
▪ Be prepared. ▪ Foster alliances. ▪ Develop consensus recommendations. ▪ Test messages.	▪ Acknowledge the event with empathy. ▪ Explain and inform the public, in simplest forms, about the risk ▪ Establish agency/spokes-person credibility. ▪ Provide emergency courses of action (including how/where to get more information). ▪ Commit to stakeholders and public to continued communication.	▪ Help public more accurately understand its own risks. ▪ Provide background and encompassing information to those who need it. ▪ Gain understanding and support for response and recovery plans. ▪ Listen to stakeholder and audience feedback, and correct misinformation. ▪ Explain emergency recommendations. ▪ Empower risk/benefit decision-making.	▪ Improve appropriate public response in future similar emergencies through education. ▪ Honestly examine problems and mishaps, and then reinforce what worked in the recovery and response efforts. ▪ Persuade the public to support public policy and resource allocation to the problem. ▪ Promote the activities and capabilities of the agency (corporate identity reinforced- internally, too).	▪ Evaluate communication plan performance ▪ Document lessons learned. ▪ Determine specific actions to improve crisis systems or the crisis plan.

Figure 3-18. Crisis communication lifecycle (*from CDC, 2002*).

Disease outbreaks and the media

In the case of a significant disease outbreak, it is likely that the media will want information. Tactics for dealing with the media should be covered within a communications plan. Strategies for dealing with the media will vary depending on desired outcomes, for example, the media may be an effective way to communicate with wetland stakeholders.

There are, however, risks. Control of the messages is ceded by adding an additional level between you and your target audience. Sometimes the media can negatively affect a situation either by inaccurate reporting or taking up too much time and resources during a crisis.

By being prepared and planning for this scenario, it can be ensured that the attention of the media works to help the situation. The communications plan should cover, for example, whether:
a) nominated people within an organisation are a spokesperson and/or field enquiries, or
b) enquiries are passed on to other organisations with greater relevant communications resources and experience.

When dealing with the media over disease risks, there are a number of guidelines which may be helpful and should be borne in mind. ►**Checklist 3-5** summarises these.

CHECKLIST 3-5: Guidelines for dealing with the media over disease risks

- ☐ Create a very detailed communications plan with specific actions for dealing with the media (not just a top line strategy) that covers all media aspects and has clearly assigned roles and responsibilities for individuals.
- ☐ Write the plan in ‘peacetime’, before a disease problem, when it is easy to take time and plan calmly.
- ☐ Work out clear simple messages.
- ☐ Agree an organisational line but be adaptable as the situation changes.
- ☐ Clearly assign roles and responsibilities, including a single organisational contact point for media inquiries and spokespeople.
- ☐ In peacetime, train a small number of key spokespeople (exercises can be very useful).
- ☐ Foster good relationships with the media in ‘peacetime’ by briefing them on wetland issues. This will help them see you as an authority to be trusted during a crisis.
- ☐ Do not let allow the media to control you or the situation. If dealing with the media does not bring benefits, then do not be afraid to say no to journalists - you will not offend them or ruin your relationship, they are used to hearing no, they respect it and often expect it. It will help to determine scenarios when you will proactively use the media and when you will only react to enquiries.
- ☐ If you are responding to an inquiry, ask beforehand what is the nature and angle of the media story so you have opportunity to prepare and do some background research.
- ☐ Ensure that what you say is evidence-based (qualify the certainty of your statements if necessary), avoid speculation and stick to your area of expertise.
- ☐ When deciding whether to answer media enquiries, keep asking yourself 'what are the potential risks to the situation of doing this and what are the potential benefits?' This may sounds simple but it helps to ensure you are maximising your resources during a time of crisis.
- ☐ Try to harmonise your communications with other stakeholders. Working together provides a stronger voice. As an example, joint statements can be powerful.
- ☐ Interviews tend to be very short so messages must be clear and brief. In general, for crisis situations ‘CARE’ offers a simple three line framework:

  C – concern. Share the concern about the situation

  A – action. Say what action is being taken

  RE – reassure. Where appropriate, provide reassurance.
- ☐ It is key is to get people to stick to the plan and not panic – this is sometimes hard!

CASE STUDY 3-10. The media and highly pathogenic avian influenza H5N1

The autumn of 2005 and spring of 2006 saw a significant westward spread of Eurasian lineage HPAI H5N1. Long before a case had been diagnosed in western Europe the media had, by its own admission, 'gone to town' on the story and its potential threats to human health in particular. The stories invariably discussed the bird infection, wild bird migration and a human pandemic together as if all were closely linked, and the latter was inevitable and possibly imminent. In general, the coverage was misleading and led to public misunderstanding of the threat from birds and thus was detrimental to conservation as measured by various means such as significantly reduced visitation to nature reserves.

Figure 3-19. Sensationalist media coverage: photo montage of ducks over London in national newspaper.

This case study documents some lessons learned from dealing with this unusual and very challenging time:

When an outbreak occurs it is easy to get completely overwhelmed by journalists, media and the general public demanding information and/or organisational statements. Because of this, it is important and extremely useful to have a dedicated person and/or team to deal exclusively with AI to agree policy line and the method of response to an outbreak. The extent to which this is possible depends on organisational capacity.

It is helpful to have:

- One or two people to be spokespeople with all media queries directed to them.
- Someone to keep up-to-date with a rapidly changing situation, accumulating news and disseminating it to the organisation and interested parties.
- Making sure that all staff are well informed of any new developments (they may be approached by journalists too) using:
    i. Emails
    ii. Intra/internet updates
- Easy access to information for journalists and the general public.
    i. Web based for quick and easy updating of information.

Proactive messages/strategies

- What is the message that you are trying to convey? Agree on the message but be ready to adapt it constantly as new facts emerge.
- Get across a balanced message with verified facts. Use trusted sources of information.
- With all the negative coverage of wild birds in the media, it is sometimes easy to overstate the lack of evidence for the spread of AI by wild birds. Stick to the facts.
- Use sympathetic journalists/media to get across your views to specific/targeted audiences. Actively seek out and develop relationships with journalists.

Reactive strategies

Forward planning. Much of the background information and accompanying text can be prepared in advance of a case of H5N1. Different scenarios can be envisaged and the appropriate information for each prepared. This can be crucial in saving time.

Checklist of things to remember

- Every situation/outbreak is different.
- Keep an open mind as to the source of infection.
- If feasible, have someone at or near the site to talk to media.
- Be easily available to the media, whether in person or by phone and email.
- Keep those commenting on developing situations to as few as possible.
- Approach the media yourself.
- Stick to your area of expertise. It is very easy to stray into and comment on other topics to reinforce your point. Try to avoid this.
- Taking the scientific approach of waiting for evidence before commenting on likely routes of infection may be seen as ‘sitting on the fence’, especially when media will want immediate answers. It is vital that one does not make comments based on ‘gut feelings’. Reiterate the importance of an evidence base before making comments.
- Check your facts regularly and make sure they are as up-to-date as possible.

Case study from RSPB/Birdlife International 2007

3.5.2 Building capacity by education and training

Systematic multidisciplinary education and training programmes should be established for all those who, in their professional capacity, are involved in any stage of managing and controlling disease. This includes personnel managing a site, assessing the risk of an outbreak, reducing the risk of disease emergence, involved in the diagnosis and surveillance of a disease, and controlling an outbreak.

Training is particularly important for front-line personnel, who are likely to come into contact with an incursion or outbreak of disease first, such as, wetland managers and members of disease diagnostic teams. All appropriate stakeholders should be thoroughly trained in their roles and responsibilities in a disease emergency. More intense and specialised training is needed for personnel/professionals holding key positions, such as members of specialist diagnostic and surveillance teams, forecasting experts and animal and human health professionals. Selected staff should also receive training in disease reporting procedures.

Given the complexity of multi-use wetlands (*i.e.* those supporting people, agriculture and wildlife), training programmes can be most useful for trainees when they are multidisciplinary. Moreover, training programmes should be comprehensive and regular, to accommodate the possibility that a disease may occur in any part of a country, and to allow for staff turnover. Training must extend to staff in remote areas, as well as to selected officials, such as local authorities. Back up staff for each position should also be trained, in the eventuality of absent front-line staff.

It will not always be possible, or practical, to train all personnel to a high level of expertise in the diseases themselves. Knowledge of basic clinical, pathological and epidemiological features of diseases known to be important, or potentially important, to a site, together with an understanding of actions to be taken when the presence of disease is suspected, may suffice in many circumstances. Importantly, the principles and practicalities of investigating a disease outbreak with an open mind should be the subject of training [►**Section 3.3.5 What data to collect at a suspected outbreak**].

The following training possibilities may be selected, as appropriate:

- **National emergency disease training workshops**: coordinated workshops should form the focus of training and should target those involved in each stage of managing an outbreak. These workshops should be organised by trained personnel and ideally include representatives from, for example, neighbouring counties or regions, or those countries or regions with experience of dealing with the specific disease in question.
- **Exchange of personnel**: key staff should be sent to other disease control centres which are proficient in dealing with the relevant disease, particularly those in the process of controlling an outbreak, to gain first-hand experience of steps taken to manage an outbreak. Other opportunities for staff to gain knowledge and understanding of managing outbreaks, such as attending workshops, should also be utilised.
- **Linkages with international disease control centres and reference laboratories** should be fostered to share knowledge about, and 'lessons learned' from, managing outbreaks.
- **Training and field manuals** may be useful for reference but ideally, should not be solely relied upon for training.

Simulation exercises

Simulation exercises are valuable for testing and refining contingency plans in advance of any disease emergency [**►Section 3.1.4 Contingency planning**] and are an effective way of building teams for emergency disease responses and training staff. Realistic disease outbreak scenarios should be created, using real data where possible. A scenario may cover several phases of an outbreak, with a range of possible outcomes, but should not be overly complicated or long. It is useful to test one system at a time (*e.g.* communication network or operation of a local animal disease control centre). Simulation exercises can be desk-based, involve mock activities or combine both approaches. There should be a review after completion of each simulation exercise to identify further training needs and any areas of the contingency plan in need of modification.

A full-scale disease outbreak simulation exercise should be attempted after individual components of the disease control response have been tested. Care must be taken to ensure that the simulation exercises are not confused with actual outbreaks in the minds of the media and the public (*e.g.* simply ensuring all electronic files and paperwork have the word 'Exercise' printed across it).

Figure 3-20. Desk top or practical simulation exercises to test contingency plans are highly valuable, particularly when bringing together a range of stakeholders including disease control agencies.

CASE STUDY 3-11. Training for live wild bird avian influenza surveillance in the Dagona Wetlands of Northern Nigeria

Prompted by the clear need for building capacity for national wild bird avian influenza surveillance programmes, particularly in an African context, the Food and Agriculture Organization of the United Nations (FAO) and the European Commission's New FluBird project funded a three week advanced waterbird capture and avian influenza surveillance training programme at the Dagona Base Camp in the Chad Basin National Park, Yobe State, northern Nigeria, in October/November 2009.

The aim of the course was to develop skills amongst ornithological practitioners and infrastructure to allow long term wild bird avian influenza surveillance to be established in this region of Nigeria and provide skilled personnel for surveillance in the countries of the other African participants. The course trained 31 participants from five mainly Chad Basin countries (Nigeria (23), Niger (2), Chad (2) and also Sudan (2) and Kenya (2)). The course proved to be very successful and was deemed by participants to have fully achieved its objectives and their personal objectives also.

The course was run by trainers from the Wildfowl & Wetlands Trust (WWT) and Wetlands International (WI). A variety of capture techniques were taught with the main focus on the advanced technique of cannon netting. Cannon netting has the potential to allow the capture of large numbers of ducks (the main target for avian influenza surveillance) and is of particular use in areas where other trapping methods cannot be used.


Figure 3-21. Course participants constructing a duck trap and a set trap in a wetland.

Duplicate sets of avian influenza cloacal and oropharyngeal swab samples were taken from trapped waterbirds, one set for in-country analysis[1] at the National Veterinary Research Institute, Vom, Nigeria, and one set for the New FluBird partner University of Kalmar, Sweden.

Cannon netting is a technical, complex and potentially hazardous trapping technique and successful cannon netters and cannon netting teams require certain key attributes. Many of the already experienced participants proved themselves to be very technically adept and capable bird trappers and with a little extra training within existing experienced cannon netting teams should be competent at being part of a regional cannon netting team capable of both national and international wild bird surveillance programmes.

Given the experience of the Nigerian authorities and institutions in dealing with outbreaks of highly pathogenic avian influenza (HPAI) H5N1, the existing ornithological skills in Nigeria (primarily at the A.P. Leventis Ornithological Research Institute, APLORI) and this, and previous, Nigerian capacity building and

FIGURE 3-22. Sampling a Northern Pintail *Anas acuta* as part of avian influenza surveillance.

surveillance work, it is proposed that Nigeria becomes a regional platform for future wild bird avian influenza activities.

[1] *When conducting surveillance for notifiable disease it is likely that the country of sample origin will wish to conduct their own analysis of samples in addition to any samples being analysed out of country.*

Case study from the Wildfowl & Wetlands Trust, November 2009.

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Chapter 4
Animal Diseases Currently Causing Concern in Wetlands

In this chapter you will find:

A summary of the animal diseases currently causing concern in wetlands.

Key questions to ask when a disease is detected: geographic range, wetland characteristics, host range, seasonality, transmission, field signs and potential impacts.

Factsheets on a selection of diseases currently impacting wetlands providing a brief description of the disease and the methods used for prevention and control.

Chapter contents

4.1 Animal diseases currently causing concern in wetlands

What is a wetland disease?

For the purposes of this Manual a wetland disease is considered to be one that either occurs in wetlands or is caused by agents that depend on wetlands. Diseases with water-borne pathogens and/or aquatic hosts such as amphibian chytridiomycosis, crayfish plague and epizootic ulcerative syndrome are obvious diseases of wetlands. There are numerous other diseases (such as bovine tuberculosis and some of the tick-borne diseases) which, at first consideration, would seem to be unrelated directly to water and wetlands. Yet these habitats are involved in the dynamics of the disease. This may, for example, relate to seasonal rainfalls, heralding temporary wetlands, flushes of vegetation attracting high densities of waterbirds or grazing ungulates and conditions for hatch-off of large numbers of invertebrate vectors. These seasonal triggers, thus, result in 'seasonal' disease – related to water and wetlands. Considering wetlands, temporary or permanent, as 'meeting places' where wildlife and humans, with their associated livestock, are attracted due to the provision of food and water, allows us to appreciate how density and variety of hosts at wetlands result in diseases being related to these wetland settings.

Figure 4-1. The diversity and density of both wetland and terrestrial hosts at wetlands allows us to understand how these 'meeting places' allow disease emergence and transmission.

One of the greatest central causes of disease problems in wetlands is the issue of faecal contamination in wastewaters from both humans and livestock. The problem is particularly great where there are intensive animal rearing facilities or high densities of people with poor or little sanitation and sewage treatment. The shared nature of so many infectious diseases across the sectors of humans, livestock and wildlife [►**Figure 2-3**] illustrates how inadequate or breakdowns in water management, hygiene and sanitation, can lead to wider infection in hosts of other sectors which can then perpetuate infection cycles and spillback into the original sector.

What is an important or priority wetland disease?

Both from the original Ramsar COP 10 request for guidance and the user needs survey, it was apparent that the provision of *practical* guidance for wetland managers was of importance. Specific guidance for every disease, of every wetland, in every location, would be both complex and beyond the capability of a small team of authors operating over only one COP period *i.e*. a triennium. Instead, the Manual focuses on principles and practices of disease management with specific information on only a sub-set of priority animal diseases of wetlands.

An ability to prioritise diseases of importance within particular wetlands would allow wetland mangers, policy makers and professional health services to allocate resources accordingly for surveillance, management, research, awareness raising, and other prevention and control activities. Prioritisation of important diseases is not as easy as it sounds as ‘importance’ may depend on personal, cultural or organisational perspectives. Taking an ecosystem approach to health helps ensure that diseases are seen, and dealt with, from a broader perspective with an understanding and appreciation of the interconnectivities.

An experts workshop was held in 2010 to perform a disease prioritisation exercise and identify which diseases were of greatest importance, for which specific factsheets would be produced. The aim of the workshop was to identify approximately 30 of these priority animal diseases of wetlands which also impact humans, ensuring that this subset contained at least some diseases of each animal taxa, and for all regions of the world, to help maximise the utility of the Manual.

The first task of the workshop drew up a long list of animal diseases associated with wetlands. Each disease’s relevance to wetlands was scored, priority being given to those diseases where either the host, pathogen/toxin or vector was entirely dependent on wetlands.

Diseases were then scored according to their impact on:

- Wildlife health (*data were often lacking so expert judgements were made*);
- Livestock health;
- Human health; and
- Livelihoods.

A number of diseases, such as tick-borne diseases were grouped together as many of the practical approaches to managing them were similar.

The scoring was then summed, using a weighting towards relevance to wetlands and impacts on wildlife. This decision was made given the focus of the Manual and the available information already in existence regarding livestock diseases. This prioritisation provided a relative ranking rather than an absolute cut-off beyond which diseases were not considered important [►**Appendix VI**].

Ultimately, the factsheets that were produced and presented within this chapter, cover a broad range of priority animal diseases in wetlands, and together cover at least some diseases of all taxa, in various geographical regions. Further disease factsheets will be developed in later additions of this Manual.

Points for consideration

The reader must appreciate that the factsheets presented within this chapter represent information on only a sub-set of diseases and thus must not constraint thinking with respect to trying to diagnose a disease. Animal health expertise should always be sought when making decisions on priority diseases of particular wetlands. It is also worth understanding that many disease problems are multifactorial and a single disease may not be responsible.

Figure 4-2. The aftermath of a lesser flamingos *Phoeniconaias minor* die off. The causes of lesser flamingo mortality events appear to be multifactorial and not due to one specific disease. The thinking of the wetland manager must not be constrained by the limited number of disease factsheets presented herein (*Ruth Cromie*).

4.2 Key questions to ask when a disease is detected

Given the diversity of hosts and diseases on the planet, it is difficult to provide specific disease guidance for every situation. For a wetland manager faced with a disease problem in need of a rapid diagnosis, expert animal disease expertise should be sought from local or national authorities. This section merely provides some guidance to the key questions to help the wetland manager to begin to 'eliminate' some disease possibilities and to assist the dialogue with disease professionals conducting an epidemiological investigation. Further relevant concepts regarding epidemiological information are provided in ►**Section 3.3.5 What data to collect at a suspected outbreak**.

Which diseases are found in this geographical range?

Many abiotic diseases, such as anthropogenic toxic diseases, may have a broad geographical range. Conversely, most biotic diseases have a defined geographical range determined by the range of the pathogen, host or vector. The nature of trade (legal and illegal) and other anthropogenic movements can allow the introduction of disease into new areas and so this should be borne in mind – *novel disease is a possibility*.

Which diseases are found in wetlands with these particular characteristics?

The character of the wetland greatly affects the nature, prevalence and incidence of associated diseases. As an example, deep lakes or fast flowing rivers are much less likely to be sources of schistosomiais or Rift Valley fever as the vectors of these diseases (freshwater snails and mosquitoes, respectively) will be less abundant. A wetland manager should familiarise themselves with the diseases associated with the type of wetland for which they are responsible.

Which diseases are found in this host range?

The species affected by a particular disease are a key part of an epidemiological investigation and will help guide a wetland manager and animal health professional into considering possibilities of a cause. As an example, within a biodiverse wetland, an outbreak of avian botulism may kill many waterbirds and leave other taxa unaffected, whereas, a harmful algal bloom may affect almost all animal taxa present.

Which diseases are prevalent in this season?
Many diseases are seasonal (*e.g*. duck virus enteritis), related to temperature (*e.g*. avian botulism), rainfall (*e.g.* tick-borne diseases), or human activities (*e.g*. lead poisoning during, and at the end of, a hunting season). A wetland manager should become familiar with how seasons trigger health events within a particular wetland.

Which diseases might be transmitted by a certain route?
A wetland manager should be familiar with how diseases are transmitted, which then allows a better ability to assess risk and potential cause of disease. A strong likelihood of water-borne pathogens associated with faecal contamination having entered waterways provides a pointer for a wetland manager to start contemplating the range of associated diseases that might be at play, *e.g.* salmonellosis, campylobacteriosis, *E. coli* poisoning, and so on. As another example, a relative absence of invertebrate vectors such as mosquitoes may make an outbreak of Rift Valley fever unlikely.

Which diseases are associated with which specific field signs?
A wetland manager should know what represents 'normal' behaviour and ecology in livestock and wildlife in the wetlands they manage. Deviations from this normal state, whether behavioural or otherwise, may then provide a good indication of the disease processes at play. The field signs of *e.g*. crayfish plague or *e.g*. avian botulism are not necessarily specific to those diseases but they are indicative.

What are the potential impacts?
Determining the potential impacts of a disease will be impossible without a diagnosis from animal health experts, however, the wetland manager will be able to contribute to the impact assessment given their knowledge of human, livestock and wildlife activities within a wetland site.

►**Appendix VII** provides a summary of the factsheets within this chapter as a matrix allowing a wetland manager to search by wildlife taxon or geographical area for particular disease information.

Figure 4-3. Wetland characteristic and geographical range: a mesotrophic lake in Iceland and a eutrophic lake in Nepal, choked with invasive alien water cabbage *Pistia spp*. Regardless of susceptible hosts present in these wetlands, the geochemical, hydrological, climatological and biological attributes of these wetlands ensure a different diversity of potential diseases and invertebrate vectors (*Ruth Cromie, Sally Mackenzie*).

4.3 Disease factsheets

The following section contains the factsheets for the selection of priority wetland diseases.

The factsheets are designed for wetland managers focusing on the aspects most relevant to disease management in wetlands, such as prevention and control measures. The factsheets are not intended as diagnostic guides, but as primers describing the disease, listing available management strategies, and directing the reader to sources where further technical guidance can be obtained.

Factsheet sections

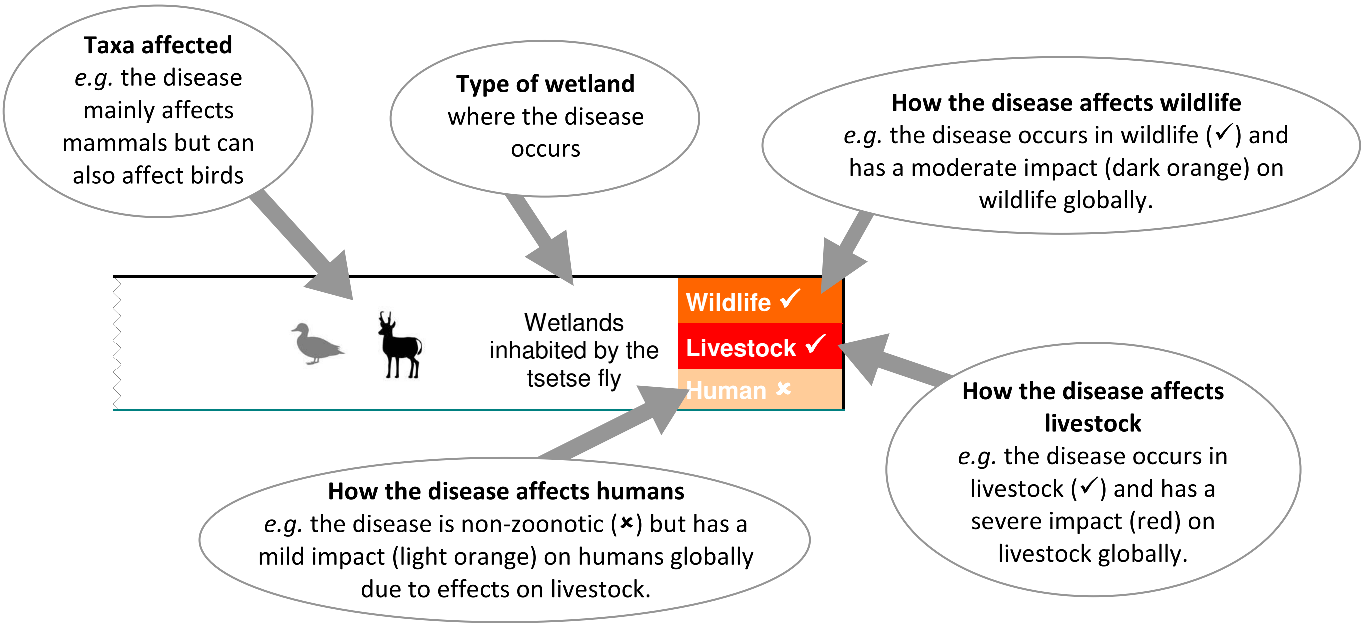
The factsheets are divided into eight sections:

Header	At-a-glance summary of taxa affected, relevant wetland type and levels of impact.
Synonyms	Alternative names by which the disease may be known.
Key facts	Brief description of the disease, the causal agent, the species affected, the geographic distribution and the environment in which the disease usually occurs.
Transmission and spread	How the disease is transmitted and spread, including (when relevant) vectors*, transmission between individuals, spread between geographic areas and how/if the disease is transmitted to humans.
Identification and response	Identifying and responding to a disease problem, including field signs, recommended action if the disease is suspected and information about how a diagnosis may be made.
Prevention and control in wetlands	Prevention and control measures in the environment, livestock, wildlife and humans.
Importance	Global importance in terms of effects on wildlife, livestock and humans, and economic importance.
Further information	Useful publications, websites and contacts.

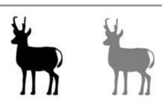
**Vector usually refers to a biological carrier which transfers an infectious agent from one host to another. For the sake of these practically-focussed factsheets they refer to various means by which infection can be transferred.*

Factsheet header explained

The factsheet header contains a quick summary of the disease, including the most widely known names of the disease, symbols to indicate which taxa are affected, a brief description of the wetland types in which the disease might be found, and three boxes indicating whether or not the disease can occur in wildlife, livestock and humans, plus the level of impact the disease has on each of these groups.



The taxa categories are invertebrates, fish, amphibians & reptiles, birds and mammals. The taxa symbols appear in the factsheet headers in two colours: **black** indicates the taxa that are usually affected, and **grey** indicates the taxa that can also be affected (see example above).

Taxa symbols	
	**Invertebrates** Animals without backbones – all animals except fish, amphibians, reptiles, birds and mammals. Includes corals, molluscs, insects, crustacea *etc*.
	**Fish** Unlike groupings such as birds or mammals, ‘fish’ (not a meaningful term for a biological grouping in itself) are not a single clade or class but a group of taxa, including hagfish, lampreys, sharks and rays, ray-finned fish, bony fish, coelacanths and lungfish - any non-tetrapod craniate with gills throughout life and limbs (if present) in the form of fins.
	**Amphibians and reptiles (together known as herpetafauna)** Animals from the classes Amphibia (such as frogs, salamanders and caecilians) and Reptilia (such as crocodiles, lizards and turtles).
	**Birds** Animals from the class Aves.
	**Mammals** Animals from the class Mammalia. Includes humans.

The impact categories are severe, moderate, mild and none. These categories are assigned based on impacts at the *global scale* rather than impacts on an individual or a population.

Impact colours			
	Severe impact		**Mild impact**
	Moderate impact		**No impact**

The ✓ and ✖ symbols indicate whether or not a disease can occur in the group specified, so for example if the humans box is ticked (✓), the disease is zoonotic (can be transmitted to humans and cause disease); if the box is crossed (✖), the disease does not occur in humans.

Notifiable diseases

Ⓝ Diseases notifiable to the World Organisation for Animal Health (OIE) are shown with this symbol. The majority of the world's countries are members of the OIE [►**Appendix IV**] and, as such, they are obliged to report these diseases *via* the country's Chief Veterinary Officer to the OIE. It should be noted that this symbol may refer to the disease in only some situations, *i.e.* disease in specific taxa.

►**Appendix V** provides a full list of notifiable diseases, correct at time of publishing (these diseases and host criteria change and the reader should check with the OIE website http://www.oie.int/ for the latest information). Some disease factsheets represent a collection of diseases *e.g.* tick-borne diseases, some of which are notifiable and some of which are not.

On suspicion of a notifiable disease, local or national animal health authorities must be contacted immediately, and these authorities will confirm or reject the diagnosis by OIE approved standards and notify them accordingly. Notifiable diseases bring trade restrictions and a range of necessary disease control measures.

African animal trypanosomiasis

Wetlands inhabited by the tsetse fly and susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: Trypanosomosis, nagana, nagana pest, tsetse disease, tsetse fly disease, souma or soumaya (in Sudan), baleri (in Sudan), surra, dourine, cachexial fevers, Gambian horse sickness (in central Africa), kaodzera (Rhodesian trypanosomiasis), tahaga (a disease of camels in Algeria), galziekte or galzietzke (bilious fever of cattle), gall sickness (in South Africa), mal de caderas and peste boba (South America).

KEY FACTS

What is African animal trypanosomiasis?

A disease caused by protozoa primarily transmitted by tsetse flies *Glossina spp*. that can affect almost all domestic mammals and infect a wide range of wild mammal species but these are mostly trypanotolerant. Trypanosomiasis is considered the most important disease of livestock in Africa where it causes severe economic losses. The disease has the greatest impact on domestic cattle but can also cause serious losses in domestic swine, camels, goats and sheep. Infection of susceptible cattle results in acute or chronic disease which is characterised by intermittent fever, anaemia, occasional diarrhoea and rapid loss of condition and often terminates in death.

Although most trypanosomes that cause African animal trypanosomiasis are not known to be zoonotic, some are of zoonotic concern, *e.g. Trypanosoma brucei rhodesiensi* and other closely related trypanosomes do infect humans. Non-zoonotic trypanosomes might cause disease in people with certain genetic defects.

Causal agent

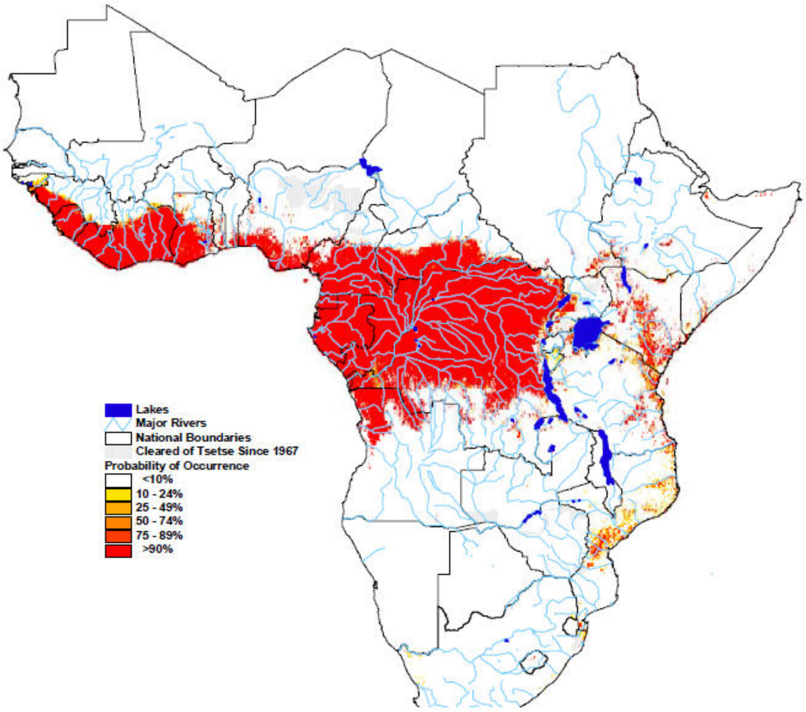
Trypanosomes, protozoan parasites of the genus *Trypanosoma* that live in the blood, lymph and various tissues of vertebrate hosts. The most important species for this disease are *Trypanosoma congolense, T. vivax* and *T. brucei subsp. brucei* and *rhodesiensi.*

Species affected

Many species of domestic and wild animals including cattle, swine, camels, goats and sheep. Cattle are prefered by the tsetse fly and this preference can shield other animals from the effects of trypanosomiasis. Wild animals known to be infected but which are trypanotolerant include greater kudu *Tragelaphus strepsiceros*, warthog *Phacochoerus africanus*, bushbuck *Tragelaphus scriptus*, bush pig *Potamochoerus porcus*, African buffalo *Syncerus caffer*, African elephant *Loxodonta africana*, black rhinoceros *Diceros bicornis*, lion *Panthero leo* and leopard *Panthera pardus*. Several species of wild animal appear not to be trypanotolerant, *e.g.* the southern white rhinoceros *Ceratotherium simum simum* can die from infection.

Geographic distribution

Endemic in Africa, primarily occurring in areas inhabited by the tsetse fly. In Africa this falls between latitude 14° N and 29° S - that is from the southern edge of the Sahara desert to Zimbabwe, Angola and Mozambique ('the tsetse fly belt') an area of 10 million square miles affecting nearly 40 countries. Some trypanosomes, particularly *T. vivax*, have spread beyond the 'tsetse fly belt', to the Americas for example, by transmission through 'mechanical vectors' (mechanical vectors transmit pathogens from one host to another but, unlike in 'biological vectors', the pathogen does not require the vector to complete its life cycle). Despite a century or more of effort to eradicate the tsetse fly, the trypanosomes have persisted across their range except in areas where all vegetation has been removed.



Probabilities of tsetse distributions in Africa (FAO, February 2000).

Environment

Any environment inhabited by the tsetse fly. The three main species of tsetse flies responsible for transmission are *Glossina morsitans*, which favours open woodland on savanna; *G. palpalis*, which prefers shaded habitat immediately adjacent to rivers and lakes; and *G. fusca*, which favours high, dense forest areas. Fly densities fluctuate seasonally which often impacts on grazing patterns.

TRANSMISSION AND SPREAD

Vector(s)

Tsetse fly, genus *Glossina* and various mechanical vectors, including biting flies particularly those of the genus *Tabanus*, but also *Haematopota, Liperosia, Stomoxys,* and *Chrysops* flies. Fomites (inanimate objects such as footwear, nets and other equipment) can also mechanically transmit trypanosomes. The vector for *T. vivax* in the Americas remains unknown, but several species of haematophagous ('blood eating'; especially tabanid and hippoboscid) flies are suspected. Trypanosomes may also be mechanically transmitted – see below.

How is the disease transmitted to animals?

Trypanosomes must first develop within tsetse fly vectors for one to a few weeks. They are then transmitted through tsetse fly saliva - when flies feed on an animal they inject saliva before sucking blood. Tsetse flies will remain infected for life. Trypanosomes can also be mechanically transmitted by biting flies when these flies transfer blood from one animal to another. In South America *T. vivax* can be mechanically transmitted and does not require the tsetse fly to develop. One trypanosome, *T. equiperdum*, is thought to be transmitted during coitus and does not have a vector. Transplacental transmission can also occur.

How does the disease spread between groups of animals?

Tsetse flies or mechanical vectors carrying trypanosomes from one group of animals to another. Animals never completely clear their parasites and thus may have inapparent (subclinical) infections. Stress can reactivate the disease in these 'carriers'.

How is the disease transmitted to humans?	Same transmission routes as for animals. Whilst African animal trypanosomes generally do not cause disease in humans, the closely related *T. brucei gambiense* and *T. b. rhodesiense* cause significant human disease ('sleeping sickness' and 'Chagas disease').

IDENTIFICATION AND RESPONSE

Field signs

Trypanosomiasis should be suspected when livestock in an endemic area are anaemic and in poor condition. Animals imported from endemic areas can be subclinical carriers and may become ill with the disease when stressed.

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals immediately if there is any illness in livestock. Tsetse-transmitted trypanosomiasis is a notifiable disease and suspected cases must be reported to local and national authorities and the OIE.

Diagnosis

The disease should be confirmed by health professionals identifying pathogenic trypanosomes in blood or lymph node smears. Anticoagulated fresh blood, dried thin and/or thick blood smears, and smears of needle lymph node biopsies can be submitted from live animals. Trypanosomes are most likely to be found in the blood by direct examination during the early stages of infection. They are less likely to be detected in chronically ill animals, and are almost never seen in healthy carriers. Xenodiagnosis (looking for the parasite in a previously uninfected vector which is exposed to the host, rather than the host itself) is also a useful technique when attempting to isolate from wildlife.

Laboratory tests should follow the methods and diagnostic thresholds described in the OIE's Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 2008, Chapter 2.4.3 (Identification of the agent).

Before collecting or sending any samples from animals with a suspected animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorised laboratories to prevent the spread of the disease. Although the trypanosomes that cause African animal trypanosomiasis are not known to be zoonotic, precautions are recommended when handling blood, tissues and infected animals.

PREVENTION AND CONTROL IN WETLANDS

Environment

Control of tsetse-transmitted trypanosomiasis relies on the control of the vector, the parasite or a combination of both. Various environmental measures can be used to control the vector:

- **Buffer zones:** if tsetse fly wetlands occur near villages, a buffer zone, *i.e.* an area around the village in which cultivation is restricted to dryland crops, functions as an obstacle for the movement of tsetse flies between the village and the wet areas.
- **Habitat modification/removal:** tsetse flies need shady and relatively humid conditions. The distribution and ecology of the different species of tsetse fly are closely linked with vegetation. Any modification in vegetation cover may affect the dynamic behaviour of the tsetse fly populations and the transmission of trypanosomiasis. In extreme circumstances, it may be necessary to remove the tsetse fly habitat however bush clearing can lead to soil erosion and other ecological disruption.
  (Note: If habitat is already unfavourable for tsetse flies, trypanosomiasis would not be expected to increase through more intensive swamp farming and water management).

Livestock

Vector control

Primary control methods should focus on reducing or eliminating tsetse fly populations *e.g.* using spray-on livestock insecticides, pheromone-baited traps, sterile insect techniques and other methods. Persistent chemicals are no longer used for environmental reasons and other non-persistent forms of spray are applicable in certain, mostly open, habitats *e.g.* Okavango swamps

►**Section 3.4.3. Control of Vectors**

Secondary control methods should employ veterinary interventions and reduce the spread of the parasite by using preventative treatments, treating infected animals and monitoring the number of animals that carry the disease.

Vaccination

There is currently no vaccine against human or animal trypanosomiasis.

Livestock management

- Good husbandry can reduce tsetse fly-livestock contact.
- Some African cattle and small ruminant breeds have some tolerance to trypanosomiasis. Introduction and development of these breeds may be effective in lessening the impact of trypanosomiasis. However it should be noted that:
  - Immunity may only be local and therefore ineffective against trypanosomes from a different region.
  - Compared with other breeds, trypanotolerant cattle are smaller in size, have lower fecundity and produce lower milk yields.
  - Immune cattle may remain carriers of trypanosomes.
  - Translocation of livestock carries the risk of spreading diseases into new areas and should be accompanied by strict sanitary controls.
- Switching from cattle to poultry farming, for example, can allow animal protein production without losses to trypanosomiasis.
- In mixed wildlife-livestock systems, tsetse can preferentially feed on wildlife species and this has a dilution effect on livestock attack.

If an outbreak is detected early, the parasite might be eradicated by:

- Movement controls and quarantine periods
- Euthanasia of infected animals - trypanosomes cannot survive for long periods outside the host and disappear quickly from the carcase after death.
- Controlling arthropod vectors to prevent new infections.
- Administration of curative drugs (*e.g.* diminazene aceturate and quinapyramine methylsulfate).
- Good nutrition and rest will allow an animal to recover more rapidly.

Wildlife

Wild animals carry trypanosomes and are an important food source for the tsetse fly. Each type of fly derives nourishment from a narrow range of animal species, however, tsetse flies have been shown to be adaptable and will utilise novel hosts in the absence of a favoured host. For this reason, and because of the obvious detriment to the local wildlife, eradication of game hosts is no longer an acceptable method of control. Prevention should be directed towards controlling vector populations or preventing human and livestock access to tsetse habitat and dedicating the land to alternative land use and income generation.

Humans Although most trypanosomes that cause African animal trypanosomiasis are not known to be zoonotic, trypanosomes related to *T. brucei brucei* and *T.brucei rhodesiense* can infect humans, and non-zoonotic trypanosomes might cause disease in people with certain genetic defects.

IMPORTANCE

Effect on wildlife Wild animals rarely show clinical signs of trypanosomiasis but wildlife hosts are a reservoir of trypanosomes. Some species such as southern white rhinoceros, which prefers open grassland, can suffer mortality from the disease.

Effect on livestock Trypanosomiasis has the greatest impact on domestic cattle but can also cause serious losses in domestic swine, camels, goats and sheep. The cattle of African nomadic communities are at particular risk as they are increasingly driven to utilise higher risk habitats due to agriculture reducing their available range. The presence of the disease can reduce livestock holdings by 10-50%. Although acute cases can be caused by less pathogenic types, in general the disease has a high morbidity rate and is often chronic in susceptible animals. The mortality rate can reach 50-100% within months of exposure, particularly if the animal is exposed to poor nutrition and other stressors. The majority of untreated animals infected with *T. congolense, T. vivax* and *T. brucei brucei* will die of the disease.

In Africa, tsetse fly transmitted trypanosomiasis is a persistent endemic disease. In South America trypanosomiasis is mechanically transmitted and epizootic outbreaks occur cyclically every few years.

Effect on humans African animal trypanosomes are not known to be zoonotic so health impacts are negligible but they are of concern in wildlife tourism areas where rare cases in wildlife can occur. This can have significant negative economic knock-on effects where illness deters visitors. The greatest impact to humans is felt through direct and indirect losses to livestock production.

► **Effect on livestock**
► **Economic importance**

Economic importance Trypanosomiasis is the most important livestock disease in Africa. Economic impacts will vary considerably depending on a number of variables such as the affected livestock species, type, productivity, susceptibility or the extent of challenge by the fly.

Direct economic impacts are felt by livestock owners without trypanotolerant breeds who suffer significant constraints on production through morbidity, mortality and impaired fertility. Indirectly, the disease affects crop producers who rely on livestock (draught oxen) to pull farm machinery and produce manure. Farmers are also hindered by perceived risks of the disease, for example, on tsetse fly-infected ground they may reduce their numbers of livestock or exclude livestock from infested regions all together. In Africa, 7 million hectares of suitable grazing land are left ungrazed due to trypanosomiasis. However, the benefits for wildlife balance this economically where tourism and other forms of wildlife utilisation exist. In some countries the wildlife contribution to GDP is far bigger than from the agricultural sector.

Implementing prevention and control measures using trypanocidal drugs represents an additional expense.

FURTHER INFORMATION

Useful publications and websites

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Amphibian chytridiomycosis

Wetlands inhabited by amphibians

Wildlife ✓
Livestock ✗
Human ✗

Synonyms: Chytrid, chytrid fungus, chytrid disease, B.d, *Batrachochytrium dendrobatidis*

KEY FACTS

What is amphibian chytridiomycosis?

A disease of amphibians caused by the fungus *Batrachochytrium dendrobatidis*. The fungus affects the keratinised tissues of amphibians *i.e.* the skin of adult amphibians and the mouthparts of tadpoles of most species of anuran amphibians (frogs and toads). The disease has become a major cause of amphibian mortality and morbidity worldwide over the last decade, leading to catastrophic declines in populations in North America, South America, Central America, Europe, Australia and the Caribbean. The disease does not affect livestock or humans, their only role being as carriers of the fungus on *e.g.* feet, equipment or clothing.

Causal agent

The fungus *B. dendrobatidis*.

Species affected

Most species of amphibian, although its severity can range from no clinical signs to acute mortality, depending on the amphibian species, the infectious dose, the strain of fungus and the environmental conditions. The disease has been described in a wide variety of anurans (frogs and toads) and caudates (salamanders and newts), but not yet in caecilians.

Geographic distribution

The disease occurs in every continent where there are amphibians *i.e.* all continents except Antarctica.

Environment

Any environment inhabited by amphibians. This disease has occurred at varying altitudes and degrees of humidity in areas of standing water. It affects aquatic, terrestrial and arboreal amphibians. It has also occurred in more arid areas inhabited by salamanders *e.g.* in Europe.

TRANSMISSION AND SPREAD

Vector(s)

Although the fungus is not vector-borne, it may be spread mechanically by movement of infected amphibians, contaminated water or mud, or *via* fomites (inanimate objects such as footwear, nets and other equipment).

How is the disease transmitted to animals?

The fungus has two life stages, an intra-cellular sporangium and a free-swimming zoospore. Zoospores are released from the skin (or mouthparts) of an infected animal and move through the water, or remain in a damp environment, until they come into contact with another (or the same) amphibian, which they then infect.

How does the disease spread between groups of animals?

Movement of amphibians or spread of contaminated material (including water, mud or fomites) between groups.

How is the disease transmitted to humans?

The disease is not transmitted to humans.

IDENTIFICATION AND RESPONSE

Field signs

Field signs can vary: there may be numerous dead amphibians visible in and surrounding water bodies, or no dead amphibians visible (especially in areas where they are swiftly scavenged). The causative fungus has different impacts in different amphibian species (*e.g.* infected American bullfrogs *Lithobates catesbeianus* have been shown to not display clinical signs in most cases), therefore, an absence of diseased/dead amphibians does not mean that a population is uninfected. Some of the most common signs in individuals are reddened or otherwise discoloured skin, excessive shedding of skin, abnormal postures, such as a preference for keeping the skin of the belly away from the ground, unnatural behaviours such as a nocturnal species that suddenly becomes active during the day, or seizures. Many of these signs are said to be "non-specific" and many different amphibian diseases have signs similar to those of chytridiomycosis.

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals. *B. dendrobatidis* infection is a notifiable disease and suspected cases must be reported to local and national authorities and the OIE.

Diagnosis

Diagnosis is carried out by taking samples using swabs: swabbing the skin of the back legs, drink patch (*i.e.* ventral pelvic skin) and tail (in caudates) of adults and of the mouthparts of larvae in live amphibians. These are then analysed for the presence of *B. dendrobatidis* using real-time PCR. The skin of dead amphibians can be similarly swabbed and freshly-dead specimens can be submitted for *post mortem* examination, including histology, in specialist laboratories.

Before collecting or sending any samples from animals with a suspected disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorised or suitably qualified laboratories to prevent the spread of the disease. Although the fungus that causes amphibian chytridiomycosis is not known to be zoonotic, routine hygiene precautions are recommended when handling animals. Also, suitable precautions must be taken to avoid cross-contamination of samples or cross-infection of animals.

(Left) Trapping newts for chytrid fungus surveillance: high standards of biosecurity must be observed, *e.g.* using site-specific equipment and thoroughly disinfecting and drying all equipment after use. (Right) Swabbing the drink patch (ventral pelvic skin) of a smooth newt *Lissotriton vulgaris* for chytrid surveillance. Note the use of clean gloves when handling each animal to reduce the chances of transfer of infection (*WWT*).

PREVENTION AND CONTROL IN WETLANDS

Environment

Ensure that the site is regularly scanned for dead amphibians or signs of non-native species. If either are found, they should be sampled for *B. dendrobatidis* infection. Ideally, population monitoring and *B. dendrobatidis* infection surveillance should be conducted at any site containing a reasonable population of amphibians, especially if endangered species are present.

Livestock

The disease does not affect livestock, however, ensure that livestock moving between sites (especially those travelling from known infected sites) do not mechanically spread infection by carrying infected material on their feet or coats. Ensure that feet are clean and dry before transport. Use foot baths and leave animals in a dry area after the bath for their feet to fully dry before transport.

Wildlife

Do not allow the introduction of non-native amphibian species to the site. Ideally avoid amphibian re-introductions unless as part of well managed re-introduction programmes with rigorous biosecurity and infection screening protocols.

Adopt a biosecure approach to managing your wetland:
►Section 3.2.4. Biosecurity

People coming into contact with water or amphibians should ensure where possible that their equipment and footwear/clothing has been cleaned and fully dried before use if it has previously been used at another site.

To properly clean footwear and equipment:

- First use a brush to clean off organic material *e.g.* mud and grass.
- Rinse with clean water.
- Soak in fungicidal disinfectant for one minute.
- Rinse with clean water and allow to dry. Drying thoroughly is important and will act to kill any chytrid present.

If any clothing is particularly soiled during activities, then wash it at 40^{o}C with detergent to remove any contamination with chytrid.
Ideally use different sets of footwear for different sites.
► Case study 3-4. Managing chytridiomycosis in wetlands (Section 3.2.4)

Humans

The disease is not transmitted to humans.

IMPORTANCE

Effect on wildlife

Only amphibians are affected. Significance varies greatly from no obvious signs to extremely severe effects leading to extinction of affected populations or species. This is the most important disease for amphibians.

Effect on livestock

None

Effect on humans

None

Economic importance

Of economic importance due to its impact on the commercial amphibian trade, particularly the pet and scientific trades, and on the harvesting of wild amphibians for the food trade in some areas. The likely declines and extinctions of multiple species will have long-term ecological impacts and as yet unknown economic ramifications.

FURTHER INFORMATION

Useful publications and websites

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Contacts

Diagnostic laboratories (contact before sample submission).

- **Histology**: any specialised laboratories
- **qPCR**: Institute of Zoology: Zoological Society of London, Regent's Park, London NW1 4RY, UK. matthew.perkins@ioz.ac.uk
- **PCR**: Exomed, Erich-Kurz-Str. 7, 10319 Berlin, Germany. mutschmann@exomed.de
- **PCR**: Tobias Eisenberg, Landesbetrieb Hessisches Landeslabor, Schubert Str. 60 - Haus 13, 35392, Giessen, Germany.
- Pisces Molecular**,** 2200 Central Avenue, Suite F, Boulder, CO 80301,USA. jwood@pisces-molecular.com.
- School of Biological Sciences, Center for Integrated Biotechnology,Washington State University, Pullman, WA 99164-4236,USA. astorfer@wsu.edu.
- Wildlife Disease Laboratories, Institute for Conservation Research San Diego Zoo. apessier@sandiegozoo.org
- Center for Wildlife Disease, University of South Dakota, **Biology** Department, 414 E. Clark Street, Vermillion, SD 57069, USA. Jacob.Kerby@usd.edu

Anthrax

Wetlands in primarily arid regions supporting *Bacillus anthracis*

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: *Bacillus anthracis*, charbon, inhalation anthrax, Ragsorter's disease, Woolsorter's disease, Woolsorter's pneumonia

KEY FACTS

What is anthrax?

A highly infectious disease caused by the aerobic spore-forming bacterium *Bacillus anthracis*. Spores may remain dormant and viable for decades, surviving adverse environmental conditions then germinating during favourable conditions. An acute infectious disease, anthrax can affect almost all species of mammal, including humans.

Animal anthrax primarily affects herbivores which most likely consume the bacteria whilst grazing or browsing, the disease usually results in sudden death.

Causal agent

Bacillus anthracis, a bacterium that forms spores in the presence of air.

Species affected

A wide range of mammal species, including humans. A disease of domestic herbivorous mammals such as cattle, sheep, goats, horses, donkeys but also pigs and dogs.

Susceptible wild animals include rhinoceros, zebra, elephants, antelope, wild bovids (*e.g.* Bison *Bison bison*), cervids, carnivores and omnivores (*e.g.* primates). Although cases have been recorded in ostriches *Struthio camelus* and vultures, birds are considered to be relatively resistant to anthrax.

Anthrax rarely infects humans in the developed world but is a threat to those who work with affected animals and their by-products. Some forms of the disease (*e.g.* cutaneous) are relatively common in some pastoral livestock communities in the developing world.

Geographic distribution

Occurs worldwide and is endemic in southern Europe, parts of Africa, Australia, Asia and North and South America. It persists in arid deserts of the Middle East, Asia, Africa, Australia and South America with most cases reported from Iran, Turkey, Pakistan and Sudan.

Environment

Alkaline or neutral calcareous soils provide favourable conditions in which spores can persist and the bacteria can multiply. Outbreaks occur primarily in warmer seasons, or in drier seasons following previous wet seasons of unusually high rainfall.

TRANSMISSION AND SPREAD

Vector(s)

The bacterium is not vector-borne but may be spread mechanically *via* insects, carnivorous and scavenging animals. In Africa, blowflies are an important means of transferring infection to browsing herbivores.

How is the disease transmitted to animals?

The principal mode of transmission is ingestion of infective bacteria from the environment.

How does the disease spread between groups of animals?

Following the death of an infected animal the carcase decays and bacteria are exposed to oxygen. The vegetative form of the bacteria then turns back into the spores that contaminate the soil. Grazing animals spread the bacteria by eating/picking up contaminated dirt or food sources. Spores have also been found in the guts of insects, although the importance of their role is not yet known. During droughts, when animals graze closer to the ground, more dirt is consumed and the incidence of anthrax appears to increase.

Outbreaks have been reported in some domestic animals (mainly pigs) after consuming feeds containing meat and bone meal originating from carcases contaminated with anthrax bacterial spores.

Wild carnivores and scavengers become infected through the consumption of infected meat.

After feeding on an infected carcase, non-biting blowflies may contaminate vegetation by depositing vomit droplets and subsequently animals feeding on such vegetation then become infected. Although a minor mode of transmission, biting flies may transmit the disease from one animal to another during severe outbreaks.

How is the disease transmitted to humans?

Humans can become infected with anthrax by breathing in anthrax spores from infected animal products (*e.g.*wool) or cutaneous anthrax may be aquired through contact with broken skin following handling of hides, hair, fur, bone, meat or wool from infected animals. Consumption of undercooked meat from infected animals may cause gastrointestinal anthrax.

Anthrax is not known to spread from one person to another.

IDENTIFICATION AND RESPONSE

Field signs

Animals in apparently good condition die suddenly. Acute cases in cattle, sheep and wild herbivores are characterised by fever, depression, difficulty in breathing and convulsions, and, if untreated, animals may die within two or three days. In pigs, anthrax is characterised by swelling of the throat, causing difficulties in breathing and similar characteristics are seen in dogs, cats and wild carnivores. The incubation period of anthrax is typically 3 to 7 days (ranging from 1 to 14 days).

Anthrax in animals can take three forms: apoplectic, acute/subacute, and chronic.

- **Apoplectic** – occurs most frequently at the beginning of an outbreak, where animals (mostly cattle, sheep, goats and wild herbivores) show signs of loss of conciousness and sudden death.
- **Acute** and **subacute** – common in cattle, horses, sheep and wild herbivores. Signs include fever, ruminal stasis, excitement followed by depression, difficulty in breathing, uncoordinated movements, convulsions and death. Unclotted blood issuing from body orifices, rapid decomposition of the carcase and incomplete *rigor mortis* are often observed.
- **Chronic** anthrax – can be seen in cattle, horses and dogs but occurs mainly in less susceptible species such as pigs and wild carnivores. Characterised by swelling of the throat and tongue and a foamy discharge from the mouth.

Sporadic wildlife cases occur in high risk locations associated with spore accumulation from historic infections and die-offs.

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals. Anthrax is a notifiable disease and suspected cases must be reported immediately to local and national authorities and the OIE.

Diagnosis

In animals, anthrax is diagnosed using samples taken from superficial blood vessels or natural openings of dead animals and by examining blood smears on a microscope slide. Artificial media can be used to grow the micro-organism from a dead animal, hides, skin, wool or soil. For rapid diagnosis of anthrax, polymerase chain reaction (PCR) is used.

In humans, anthrax is diagnosed by isolating *B. anthracis* from respiratory secretions, the blood, skin lesions, or in persons with suspected cases, measuring specific antibodies in the blood.

PREVENTION AND CONTROL IN WETLANDS

Environment

There is no easy method of disinfecting the environment and therefore anthrax is difficult to eliminate due to long-lived spores in soil. Burning of low vegetation can help to decontaminate an area.

Livestock

In areas prone to anthrax a preventive strategy should be adopted involving thorough surveillance and annual vaccination of susceptible animals (usually cattle, sheep and goats).

Vaccination is normally carried out 2-4 weeks before the onset of the known period of outbreaks. Following vaccination, a ten day quarantine ensues for the herd and premises in countries following OIE recommendations. Any animals showing signs of anthrax must be treated and not used for food until several months after the completion of treatment. The live Sterne vaccine is effective but there is some concern over its ecological effect and possible pathogenicity in some species. Antibiotic treatment (penicillin or tetracycline) can be an option if animals show clinical signs of anthrax but often it is not a practical or feasible method of control.

Culling of infected animals and removal of diseased carcases reduces contamination sources. Burn all anthrax-infected carcases or bury in deep lime pits. When this is not possible, place the unopened carcases in heavy duty black plastic bags which are sealed and leave in the heat. This destroys the vegetative bacteria and prevents spore contamination. After several hours the carcase is effectively sterilised under these conditions. Carcases infected with anthrax should not be moved, instead they should be disposed of using appropriate methods on site to prevent further environmental contamination.

Other control measures include **autoclaving** (*i.e.* high heat and high pressure) animal products (hides, bristles, hair) to destroy spores, prompt disposal of bedding and contaminated materials, control of scavengers, and observation of general hygiene by people who have come in contact with diseased or dead animals.

Wildlife

Prevention involves recognising the risk factors associated with anthrax which include:

- History of previous outbreaks in the region.
- Topography, in particular alkaline and calcium-rich soil.
- Rain and drought patterns associated with outbreaks *e.g.* long dry periods following previous heavy rainfall.
- High densities or overabundance of susceptible species *e.g.* near and around watering holes.
- Drainage areas where spores accumulate.
- Contemporaneous outbreaks in livestock.
- Changes in vaccination programmes in livestock.

Above all, be alert, vigilant and maintain surveillance particularly during high risk times. Anthrax is a seasonal disease which may reoccur the following year and being prepared for potential outbreaks is vital. This includes early carcase detection along with minimising environmental contamination through proper carcase disposal and decontamination

Wildlife species should be monitored for any interaction with livestock (*e.g.* at water sources and grazing areas).

Control measures include:

- Rapid diagnosis of the disease.
- Rapid disposal of carcases by *e.g.* burning on site.
- Scavengers should be kept away from carcases by reducing access to carcases *e.g.* by covering them, or providing decoy uncontaminated meat elsewhere.
- Controlling blowflies in the area.
- Burning surrounding areas of bush to kill spores and disperse unaffected wildlife.
- Ring vaccination of susceptible hosts.

Trained personnel and advisory information are required to effectively manage the control of an outbreak and attempts should be made to identify the source and mode of transmission in order to inform the response team.

Zebra *Equus quagga* in an arid area surrounding a wetland. Prevention of anthrax in wildlife depends on recognising risk factors such as seasonality, density of susceptible hosts, rainfall patterns, history, soil type and so on (*Sally MacKenzie*).

Humans

Protection measures:

- Vaccination is available for humans who are at particular risk (veterinarians, animal handlers, persons working with animal carcases or products, *etc.*).
- Use personal protective equipment (PPE) when handling infected animals and their by-products.
- Wash hands with soap and water to remove the vast majority of spores and keep fingers away from the mouth and nose.
- Treat wounds or scratches as soon as possible to reduce cutaneous infection by spore contamination.
- In the presence of acute respiratory infections or other debilitation, be on alert for "flu-like" symptoms as pulmonary infections are most likely.
- In the unlikely event of contracting anthrax, treatment is highly effective with simple penicillin, erythromycin G, tetracycline and a variety of other antibiotics.

IMPORTANCE

Effect on wildlife

- Recurring outbreaks have occurred in some regions and the disease is considered endemic and 'normal' in some large wildlife areas.
- The impacts can be greater where protected areas are smaller and where losses are proportionally greater.
- Outbreaks can put endangered species at risk of mass die-offs and rapid population decline.
- A number of significant, high mortality anthrax epidemics in wildlife have occurred in Africa over the last decades. It is suggestive of re-emergence but the cause of this is not always clear. These have included: thousands of hippopotamuses on the Zambesi; in Queen Elizabeth National Park, Uganda; and affecting a variety of species in Zimbabwe, Ethiopia, Tanzania; and endangered Grevy's zebra *Equus grevyi* in Kenya.
- Some protected areas and other environments have recurrent infection where the epidemiology is now well understood, *e.g.* in Kruger and Etosha National Parks in South Africa and Botswana. Some of these outbreaks are a result of spillover of infection from livestock epidemics especially where there is a breakdown in livestock vaccination.
- Other disease control measures such as foot and mouth disease fences have had an impact on the incidence of anthrax, keeping population densities high in some susceptible regions allowing the disease to become endemic and causing regular outbreaks.

Effect on livestock

Livestock anthrax is declining in many regions of the world due to good prevention and control measures. That said, the disease can still cause heavy losses and will remain a particular problem where the disease is present in wildlife areas and there is contact between wild and domestic populations.

Effect on humans

A potentially fatal zoonotic infection and thus a risk to human health when dealing with infected animals or their products. Livestock losses impact food security and livelihoods particularly in regions where disease is endemic.

Economic importance

Economic losses may be significant as a result of anthrax outbreaks especially for livestock traders.

FURTHER INFORMATION

Useful publications and websites

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Avian botulism

Any wetland supporting *Clostridium botulinum* and susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Alkali poisoning, duck disease, limberneck, Western duck sickness

KEY FACTS

What is avian botulism?

A paralytic and often fatal disease of birds caused by ingestion of a toxin produced by the bacterium *Clostridium botulinum*. Bacterial spores are widely distributed in wetland sediments and can be found in the tissues of most wetland inhabitants, including aquatic insects, molluscs and crustacea and many vertebrates, including healthy birds. Spores may survive for years but only give rise to the bacteria that produce the toxins under certain environmental conditions. These conditions include lack of oxygen, high temperature (noting that the disease may still occur in cold winters), and an organic nutrient source. These ecological factors largely control botulism outbreaks in birds. Illness in humans is rare and associated only with specific toxins.

Causal agent

Toxins produced by the bacterium *Clostridium botulinum.* There are seven types of toxin; A, B, C, D, E, F and G. Types C, D and E cause botulism in mammals, birds and fish. Types A, B, E and rarely F, cause illness in humans. Humans are reported as being resistant to the other toxins but this may be relative resistance and dose related.

Species affected

Many species of birds, particularly waterfowl, pheasants and poultry, and some mammals, including cattle, mink, sheep and horses. Illness in humans is rare.

Geographic distribution

Occurs worldwide.

Environment

Any environment supporting *Clostridium botulinum* and its animal hosts. Conditions needed for toxin production include lack of oxygen, high temperature, and an organic nutrient source, often in the form of dead invertebrates or vertebrates and decomposing vegetation, plus the presence of a bacteriophage - a bacteria-targeted virus. These conditions are produced during, for example, hot weather when water levels drop and create a layer of dead and decaying matter at the edges of water bodies. Salinity (up to 3 parts per thousand) can increase the likelihood of toxin production.

TRANSMISSION AND SPREAD

Vector(s)

Spread by infected invertebrates (*e.g.* maggots) and birds (see below for details of carcase/maggot cycle) and by transfer of infected carcases by predators/scavengers.

How is the disease transmitted to animals?

Through direct ingestion of the toxin or through ingestion of contaminated food and water. Birds commonly acquire bacteria through feeding on infected invertebrates. A cycle develops where the presence of dead animals and high ambient temperatures attract flies which lay eggs and produce maggots. Maggots feeding on a bird that has died of botulism concentrate the toxin and birds eating these maggots may die. This carcase/maggot cycle may then amplify the disease. Birds can develop botulism after consuming only a few larvae. Cattle may ingest toxin through chewing infected bones and carrion in phosphorous-deficient areas, and ingesting rotting organic matter and other contaminated food.

How does the disease spread between groups of animals?

Spreads from one animal group to another through the methods detailed above. Transfer of infected carcases by predators may also indirectly spread the bacteria. Avian botulism is not directly transmissible or communicable by casual contact but, in some cases, tissues from dead animals can be toxic if ingested by other animals.

How is the disease transmitted to humans?

Most commonly transmitted through ingesting contaminated food, particularly fish, wildfowl, marine mammals and processed animal products. It can also be transmitted through wound infections or intestinal infection in infants. Occasionally, humans can be exposed to the toxin by an aerosol. Person to person transmission of botulism does not occur.

IDENTIFICATION AND RESPONSE

Field signs

Appearance of lines of bird carcases coinciding with receding water levels may signal an outbreak. Healthy, sick and dead birds are often found together during an outbreak with carcases in various stages of decay. Affected birds may be unable to use their wings and legs normally or unable to control the third eyelid (may not be visible), neck muscles and other muscles and may therefore be seen propelling themselves using weak wings across water and mudflats. Birds with paralysed neck and leg muscles cannot hold their heads up and may therefore drown. Death is frequently caused by respiratory failure caused by the toxin paralysing muscles used for breathing. A fish die-off may also indicate an outbreak, particularly with botulism E toxin.

Affected cattle and horses tend to have a stiff gait and are often found recumbent with laboured breathing. Saliva **may** drool from their mouth.

In humans, symptoms include blurred vision, dry mouth, difficulty in swallowing or speaking, general weakness, and shortness of breath. The illness may progress to complete paralysis and respiratory failure, but, if treated, rarely death.

The disease often affects the same wetlands, and the same spots within a wetland, each year.

Recommended action if suspected

Contact and seek assistance from animal and human health professionals immediately if there is any illness in birds and/or people. Report suspected cases to local or national authorities.

Diagnosis

Avian botulism can be tentatively diagnosed by the clinical signs and the exclusion of other neurological diseases. Detection of the toxin by health professionals is needed for a definitive diagnosis. Diagnosis in animals relies on identifying the toxin in faeces, blood, vomit, gastric aspirates, respiratory secretions or food samples. Serum is required for diagnosis in sick birds and tissue samples such as clotted heart blood, stomach contents, or liver are required for diagnosis in dead birds. Laboratory diagnostic tests have poor sensitivity and specificity. In wild birds clinical diagnosis is most frequently made - flaccid paralysis being very characteristic.

Food and water samples associated with suspect cases should be obtained immediately, stored in sealed containers, and sent to reference laboratories for diagnosis.

PREVENTION AND CONTROL IN WETLANDS

Overall

It is not currently feasible to eliminate botulism spores from wetlands as they are so widespread and resilient. Some actions can be taken to mitigate environmental conditions that increase the likelihood of outbreaks.

Habitat management

- **Reduce organic inputs** (*e.g.* sewage, pollutants) into wetlands, particularly in warm weather. Inputs will introduce large amounts of decaying matter and may cause death of aquatic life (which forms a nutrient source for the bacteria).
- **Oxygenate water** if possible with pumps, or by improving water flow.
- **Keep water levels stable**, particularly in warm weather.
- In areas managed primarily for migratory waterbirds, avoid flooding land that has been dry for a long time and avoid lowering water levels when warm. Both could result in die-offs of fish and aquatic invertebrates whose carcases could then become substrates for bacterial growth.
- In areas managed for shorebirds, lowering water levels provides essential habitat. Avian botulism control must therefore focus on **quickly removing any carcases**.
- Waterfowl can be redistributed to lower risk areas by draining contaminated areas whilst creating/enhancing other habitats.
- *Take care to ensure these measures do not cause the dispersal of infected birds out of the area.*

Quick and careful collection of carcases and their disposal by burial or burning, especially during outbreaks, removes nutrient sources for bacteria.

- Immediately place carcases into two plastic bags to prevent leakage of fluids. Bags should always be securely closed before they are removed from the area.
- Submit carcases to disease diagnostic laboratories before being incinerated.
- Take care to avoid contaminating new areas whilst carcases are being transported to the laboratory and disposal site.
- Wear gloves and thoroughly wash exposed skin surfaces after any contact with contaminated birds.
- Disinfect field equipment used in infected areas.

Avoid locating power lines across marshes used by large concentrations of waterbirds. Carcases from collisions provide substrates for toxin production.

Sick waterfowl are easily caught and can recover if provided with freshwater and shade, or injected with antitoxin.

Monitoring and surveillance

Regular monitoring of live and dead birds, particularly in endemic areas and areas where migratory birds are concentrated, and during warm periods, can help identify early stages of an outbreak and allows disease control activities to be activated before any outbreaks develop further.

- Document environmental conditions, outbreak sites and dates of outbreak occurrence and cessation.

- Plan for, and implement, intensive surveillance and vertebrate carcase collection.
- Where possible, monitor and modify environmental conditions to prevent the pH and salinity of wetlands from reaching or being maintained within high hazard levels.

Livestock

- Vaccination
- Prevent stock from having access to animal carcases.
- Control vermin and pest animals to reduce the risk of spread of rotting material.
- Providing nutritional supplements of protein and phosphorus to reduce bone chewing among cattle.
- Take care with the harvesting and storage of feeds to reduce the possibility of small animals contaminating feeds.
- Check water sources for organic matter contamination.

Wildlife

►Section above: Prevention and control in wetlands – overall
►Case study 3-2. Managing avian botulism at wildlife reserves in the UK (Section 3.1.3).

Humans

- **Thoroughly cook fish or waterfowl** to an internal temperature of at least 180°F to destroy the toxin.
- Anglers and hunters should **never harvest fish or waterfowl** that appear sick or dying in areas where avian botulism is known to be present.
- **Refrigeration** temperatures combined with **salt** content and/or **acidic** conditions will prevent the growth of bacteria or the formation of toxin.
- **Good personal hygiene.** Wash hands thoroughly with soap and warm water, particularly before and after preparing food and after contact with animals.
- If exposure to the toxin *via* an aerosol is suspected, remove any clothing and store in plastic bags until it can be washed with soap and water. Shower thoroughly.
- **Antitoxin** may be used to treat the disease. Severe cases require supportive treatment, especially mechanical ventilation, which may be required for weeks or months. Antibiotics are not required (except in the case of wound botulism).
- There is no fully tested vaccine against botulism.

IMPORTANCE

Effect on wildlife

It causes *significant* mass mortality of birds, particularly waterfowl, where a million or more may die in a single outbreak. Waterbirds on fresh and salt (sea) water may be affected. Some affected birds may recover without treatment. Impacts vary between species. Impacts on wild bird populations are currently unknown. The disease can result in negative perception and therefore unnecessary destruction of wildlife.

Avian botulism is probably one of the most important diseases of migratory waterbirds worldwide, and without intervention, great numbers of birds can die over a short period of time.

Effect on livestock

Causes morbidity and mortality in chickens, cattle, sheep and horses. Relatively uncommon in domestic mammals although up to 65% of affected cattle herds may fall ill and up to 40% of affected chicken flocks may die. Livestock mortality associated with dead poultry and poultry waste can be a relatively frequent occurrence.

Effect on humans

Causes morbidity, and less frequently, mortality. The death rate is high if left untreated but vastly decreases with supportive care. Recovery may take several months or longer.

Economic importance

There is potential for economic losses to the livestock industry, due to illness and death of infected animals, with cattle and poultry particularly affected, and likely trade restrictions imposed during and after an outbreak.

Illness in humans can result in significant economic losses due to the time lost from normal activities.

FURTHER INFORMATION

Useful publications and websites

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Contacts

National Wildlife Health Center (USGS)
US enquiries: +1 608 270 2400
AskNWHC@usgs.gov

WHO Communicable Diseases Surveillance and Response (CSR)
zoonotic_alert@who.int, fmeslin@who.int and outbreak@who.int

FAO Animal Production and Health Division
www.fao.org/ag/againfo/home/en/who.htm [Accessed March 2012].

Avian cholera

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Fowl cholera, avian pasteurellosis, *Pasteurella multocida* infection, avian haemorrhagic septicaemia

KEY FACTS

What is avian cholera?

A highly infectious bacterial disease which can lead to mass mortality of birds, particularly waterfowl. Death occurs quickly after infection (in less than 24 hours) and the disease can spread rapidly through a wetland killing thousands of birds in a single outbreak. Mass mortality of poultry can cause significant economic impacts on the poultry industry. Outbreaks occur at all times of the year, but major mortality events are usually observed when waterfowl are concentrated in wintering areas or during spring migration. The disease often affects the same wetlands and bird populations each year and outbreaks tend to follow the migration routes of some birds.

Causal agent

The bacterium *Pasteurella multocida.*

Species affected

Domestic fowl and almost any species of bird can be infected: most commonly ducks, geese, swans, coots, shorebirds, gulls, and crows. The bacterium can also cause infections in domestic cattle, pigs, rabbits, cats and dogs. Infections in humans are most commonly as a result of an animal-related injury.

Geographic distribution

Frequent reports of affected waterfowl in North America but also occurs in South America, Antarctica, Africa, Europe, Asia and Australia.

Environment

Occurs in a range of habitats including freshwater wetlands, brackish marshes, and saltwater environments which support birds.

TRANSMISSION AND SPREAD

Vector(s)

Infected birds, biting arthropods (ticks, mites or flies) and contaminated objects - see below.

How is the disease transmitted to animals?

Direct contact with infected birds, contact with secretions or faeces of infected birds and ingestion of contaminated food (*e.g.* infected carcases) or water. Transmission may also occur through the inhalation of airborne water droplets when birds take flight and possibly through mechanical transfer by biting arthropods that feed on birds after having fed upon contaminated carcases or contaminated environments. Bacteria are released into the environment by dead and dying birds, by live birds carrying the disease or from contaminated objects (*e.g.* cages, equipment and clothing).

How does the disease spread between groups of animals?

Dense concentrations of waterfowl can enhance disease spread through bird to bird transmission in the ways described above.

How is the disease transmitted to humans?

Most human infections result from an animal bite or scratch, mainly from domestic dogs and cats. Infections can also arise through inhalation of bacteria which is most likely to happen in confined areas of air movement where a large amount of infected material is present (*e.g*. during disease control operations).

IDENTIFICATION AND RESPONSE

Field signs

The sudden appearance of large numbers of dead birds which are in good body condition with few sick birds observed may signal an outbreak. Birds often die quickly before showing any clinical signs of illness although the number of sick birds increases when a die-off is prolonged over several weeks. Sick birds appear lethargic and may die within minutes of capture. Other signs include:

- Convulsions, swimming in circles, throwing the head back between the wings, erratic flight, mucous discharge from the mouth, soiling or matting of the feathers around the vent, eyes, and bill, nasal discharge and fawn-coloured, yellow or blood-stained droppings.
- Wild ducks and geese are particularly affected.

In poultry, sudden die-offs can occur without obvious signs. Chronic conditions can occur with birds exhibiting depression, diarrhoea and anorexia. Birds may appear lame, weak, wheezing, with swollen wattles, and twisted necks. Avian cholera in poultry can be easily confused with other diseases.

Recommended action if suspected

Contact and seek assistance from animal and human health professionals immediately if there is illness in birds and/or people. Report suspected cases to local or national authorities.

Diagnosis

Isolation of the causative agent by health professionals is needed for a definitive diagnosis. A whole bird carcase is ideally required for laboratory diagnosis. When this is not possible, heart blood, liver tissue and bone marrow should be collected in a sterile manner. Remove whole organs and package at least half of each in separate bags. The samples must be refrigerated as soon as possible after collection and kept cool during shipment. Freeze tissues if transit time is expected to exceed 24 hours.

PREVENTION AND CONTROL IN WETLANDS

Environment

Avian cholera is highly infectious and can spread quickly and so prompt action is needed to prevent and minimise the spread of the disease.

- Healthy waterbirds (*i.e.* ahead of an outbreak or migratory birds not yet at an infected site) can be redistributed to lower risk areas by draining contaminated areas/discouraging wildlife whilst creating/enhancing other habitats. *Take care to ensure these measures do not cause the dispersal of infected birds out of the area.*
- The addition of large volumes of water to a contaminated area can help dilute the bacteria to less dangerous levels.

Livestock

The disease in livestock may be avoided by employing good sanitation and animal management practices.

- Prevent the introduction of infection through movement controls, testing and quarantine.
- Detect any infected animals in the population as early as possible through surveillance and thoroughly investigate all suspect cases.
- Vaccination with an approved vaccine can be effective.

Wildlife

Quick and careful collection of carcases will reduce the exposure of migratory and scavenger bird species to the bacteria and minimise its transmission.

- Pick up dead birds by the head, preferably by the bill, and immediately placed into two plastic bags to prevent leakage of fluids. Bags should always be securely closed before they are removed from the area.
- Submit carcases to disease diagnostic laboratories before being

incinerated.

- Remove carcases before there is a major arrival of scavengers which may spread the disease further. *Take care to ensure these measures do not cause the dispersal of infected birds out of the area.*
- Take care to avoid contaminating new areas whilst carcases are being transported to the laboratory and disposal site.
- Disinfect field equipment used in infected areas.
- Scavengers and predators can be attracted away from infected areas to other feeding sites using other food sources such as road killed carcases.
- These actions need careful evaluation of bird movement patterns and of the disease cycle to assess whether they are suitable. Moving infected or potentially infected birds from one geographical location to another is not advised.

Vaccination to protect captive or endangered waterbirds may be appropriate however efficacy and safety information are often lacking. There is no practical method for immunising large numbers of free-living migratory birds.

Monitoring and surveillance
Regular monitoring of live and dead birds, particularly in endemic areas and areas where migratory birds are concentrated, can help identify early stages of an outbreak and allows disease control activities to be activated before the outbreaks develop further.

Humans

- Wear gloves and thoroughly wash exposed skin surfaces after any contact with contaminated birds.
- Process infected birds outdoors or in a well ventilated area. When disposing of carcases by open burning, care should be taken to avoid direct exposure to smoke from the fire.

IMPORTANCE

Effect on wildlife

Causes significant mass mortality of birds, particularly when bird density is high. Large gatherings of wild waterfowl are particularly affected with mortality known to exceed more than 1,000 birds per day. There may be a significant impact on wild bird populations when breeding birds are affected and through reduced survival rates of disease-carrying waterfowl. Avian cholera is becoming an increasing threat to endangered avian species due to increasing numbers of outbreaks and the expanding geographic distribution of the disease. The disease can result in negative perception and therefore unnecessary control measures directed at wildlife.

Effect on livestock

Causes significant mass mortality of poultry and can affect future viability of poultry flocks.

Effect on humans

Not considered a high risk disease for humans although infections are not uncommon.

Economic importance

Potential for significant economic impacts on the poultry industry through mass mortality of birds.

FURTHER INFORMATION

Useful publications and websites

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- Samuel, M.D., Botzler, R. G. & Wobeser, G. A. (2007). **Avian cholera**, pp. 239-269. In: Infectious diseases of wild birds, Thomas, N. J., Hunter, D. B., & Atkinson, C. T., (eds.), Blackwell Publishing Ltd., Oxford, UK.
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Contacts

- **FAO Animal Production and Health Division.** www.fao.org/ag/againfo/home/en/who.htm [Accessed March 2012].

Avian influenza

Wetlands associated with poultry farming or used by high concentrations of waterbirds

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: AI, bird flu, fowl plague, highly pathogenic avian influenza, HPAI, low pathogenic avian influenza, LPAI, poultry plague

KEY FACTS

What is avian influenza?

Avian influenza is a highly contagious disease caused by influenza A viruses, affecting many species of birds. Avian influenza is classified, according to disease severity, into two recognised forms: low pathogenic avian influenza (LPAI) and highly pathogenic avian influenza (HPAI). LPAI viruses are generally of low virulence, whilst HPAI viruses are highly virulent in most poultry species, resulting in up to 100% mortality in fully susceptible infected domestic flocks.

The natural reservoir of LPAI viruses is wild waterbirds – most commonly ducks, geese, swans, waders/shorebirds and gulls. These hosts and their viruses have become well-adapted to each other over time and infection does not usually cause overt disease. That said, recent studies indicate that some behavioural changes may occur in response to infection *i.e*. birds may be less likely to feed and move any great distances during the short period of time it takes them to clear infection (~4-10 days).

These low pathogenic viruses replicate mainly in the intestinal tract (and also in the respiratory tract) of aquatic birds. Hence, LPAI viruses may be transmitted in faeces. Thus, transmission in aquatic birds is mainly by the faecal-oral route, *i.e.* wetland habitats provide the natural source of infection for other individuals.

Mammals – most commonly pigs but also humans – can be infected with influenza A viruses.

Eurasian lineage HPAI H5N1 viruses, which emerged in China in 1996, re-emerged in 2003 and have subsequently spread across large areas of Asia, the Middle East, Europe and parts of Africa, are unusual in respect of their significant impacts and broad host range *i.e*. affecting poultry, wild birds, various species of mammal and humans. Broader public health concerns relate to the potential for these, or other, avian influenza viruses to mutate or reassort to create a pandemic strain (*i.e*. readily transmissible between humans and causing widespread disease and socioeconomic problems).

Causal agent

Influenza A viruses. Influenza viruses have a high rate of natural mutation and reassortment. Viruses belonging to the H5 and H7 subtypes (in contrast to other virus subtypes), may become highly pathogenic. The most usual route for emergence of a highly pathogenic strain of virus is following circulation of LPAI viruses in poultry. With conditions that may include high population density, genetically homogenous stock, and different husbandry systems, mutations for pathogenicity may be selected for, and thus an HPAI virus may emerge, causing high morbidity and mortality in susceptible poultry populations.

Species affected

Poultry are very susceptible to avian influenza infection and the disease causes high mortality and/or loss of producitvty.

Most species of wild bird are susceptible to infection, but the majority of reports are from waterfowl and shorebirds. LPAI viruses are particularly associated with wild ducks and high prevalence may be found in juvenile ducks in particular. Eurasian lineage HPAI H5N1 viruses have also been found in a range of predatory and scavenging birds, and even mammals (both wild and captive), most likely as a result of feeding on infected birds or bird meat.

Humans are, in general, relatively resistant to avian influenza viruses, but in some individuals infection can be severe.

Geographic distribution	Avian influenza is reported globally, including in the Americas, Asia, Middle East, Europe and Africa. The high density duck, and other poultry, farming of eastern and south eastern Asia, including outdoor and backyard flocks, have made these regions prone to outbreaks with Eurasian HPAI H5N1 viruses in recent years leading to endemic status.
Environment	AI viruses have variable environmental survival properties that differ depending on the virus subtype and environmental characteristics including temperature, pH, humidity, salinity and the type of medium the virus is found in *e.g.* water, faeces, fomites *etc*.

TRANSMISSION AND SPREAD

Vector(s)	The disease is not vector-borne, but infected animals, fomites (inanimate objects) or people contaminated with faeces and other infectious secretions can spread infection. Mechanical transfer on the feet of pests *e.g*. rodents in poultry houses is also possible.
How is the disease transmitted to animals?	The viruses have evolved to be transmitted by the faeco-oral and/or respiratory routes *i.e*. in general viruses are passed out with the faeces and/or respiratory secretions and exposed birds then ingest or inhale viruses and, if susceptible, will become infected.
How does the disease spread between groups of animals?	For poultry, infection is primarily spread through the movement and trade of poultry and poultry products locally, nationally and internationally. Live and/or wet markets pose a particular risk. Movements of people, vehicles and fomites contaminated with AI viruses can also spread infection. Hence, good biosecurity and hygiene practices are essential to prevent introduction, and control spread of, AI virus infections. The practice of outdoor poultry production, including grazing domestic ducks in rice paddies, is considered to be one way in which disease can easily transfer between wild and domestic birds (in both directions). As has also been found for Eurasian lineage HPAI H5N1 viruses, infection can be spread through the pet bird trade, wild bird trade, the farming of wild birds, and wild bird movements. The relative importance of these routes is often difficult to determine (and will differ by situation, location and time period). Scavenging and predatory birds and mammals may acquire infection by ingesting infected birds.

The farming of wild birds which have frequent access to wetlands has been highlighted as a means by which AI viruses, including Eurasian lineage HPAI H5N1 virus infection, can be spread to wild bird populations (*Richard Hearn*).

How is the disease transmitted to humans?

Humans can become infected *via* close contact with infected birds or inhalation of aerosols containing virus. In general, humans are relatively resistant to avian influenza viruses. However, situations where there is exposure to high levels of virus, such as during disease control activities or butchering or preparation of infected birds, are of higher risk and appropriate hygiene precautions should always be taken, including use of personal protective equipment.

IDENTIFICATION AND RESPONSE

Field signs

For poultry, LPAI infection may be inapparent or mild. In layer hens a drop in egg production may be seen. HPAI infection is characterised by sudden mortality which can be extremely high, up to 100%.

For wild birds, LPAI infections typically cause no obvious clinical signs. Eurasian lineage HPAI H5N1 virus infections in wild birds can be characterised by neurological signs: trembling, falling over, swimming or walking in circles. For waterbirds, other conditions such as lead poisoning can also cause these signs although this is more likely to be a longer term illness *i.e*. birds tend to be in poorer condition, unlike HPAI H5N1 where infection is acute and birds may be in good condition.

In humans, the symptoms vary from mild to severe including mortality. Symptoms include conjunctivitis, 'flu-like symptoms (including fever), coughing and shortness of breath, diarrhoea, vomiting, and abdominal pain.

Recommended action if suspected

In poultry, both H5 and H7 LPAI and HPAI are notifiable to the OIE and local and national veterinary authorities should be contacted immediately on suspicion of AI infection. HPAI H5N1 is notifiable in wild birds and veterinary authorities should be informed of any unusual mortality event of susceptible species such as waterbirds. Public health authorities should be contacted if there is suspicion of human infection.

Diagnosis

Diagnosis in poultry can be made by either assessment of antibody levels in the blood indicating exposure to AI viruses or detection of the virus, or particles thereof, on swabs taken from the cloaca or throat of birds. Virus detection assays include growing the virus within inoculated fowls' eggs or use of molecular techniques including PCR to detect presence of virus, its type and its pathogenicity – all of which are important for epidemiological investigations and informing disease control responses.

PREVENTION AND CONTROL IN WETLANDS

Environment

Measures should be taken to reduce the exposure of wetlands to poultry manure or outflows from poultry establishments.

Livestock

Poor hygiene and biosecurity, overstocking, and mixing of different animals greatly increases the risk of both introduction and the spread of infection. Primary management efforts must be focused on limiting the opportunity for infection to be introduced. The main recommended courses of action following an outbreak of disease are culling of domestic poultry flocks, implementation of movement restrictions and cleansing and disinfection of affected premises.

Biosecurity

High standards of biosecurity will help prevent introduction of virus:

- Reduce/prevent contact with wild birds (for small scale poultry holders this may involve feeding birds under cover).
- Have disinfection facilities for hands, footwear, clothing, equipment and vehicles/trailers on entering or leaving areas with poultry and after contact with animals.
- Wear protective clothing and footwear, either disposable or if re-useable, easily disinfected (*e.g.* waterproof clothing, face shields, gloves and boots).
- Have separate clothing and equipment for each person using areas with livestock.
- Pest control – although not the most important mode of transmission, controlling rodents will help prevent/reduce mechanical transfer of infection between poultry holdings.
- Disease can be reduced by good hygiene and optimal animal husbandry and by minimising stressful events.
- Isolate newly acquired animals.
- Buy animals or eggs from AI-free sources.
- Ensure water from poultry holdings or untreated manure does not enter wetlands.
- Ensure untreated/unsanitised water is not used for poultry.

Vaccination is not considered an appropriate option as it can 'mask' disease. However, it has been suggested as a control measure in some areas of endemic Eurasian lineage HPAI H5N1 infection in South East Asia, as well as for collections of captive birds.

Monitoring and surveillance

National AI surveillance schemes may help in diagnosis of the disease, but poultry keepers should be vigilant for suspect clinical signs including loss of production or unusual mortality.

In the event of an outbreak

Confirmation of disease usually results in the implementation of sanitary measures comprising the slaughter of infected stock, movement restrictions, and cleansing and disinfection of affected premises.

Wildlife

Generally LPAI viruses do not require disease control responses for wildlife, but for HPAI H5N1 measures should be taken due to the potential for high mortality.

All practical measures to reduce contact between wild and domestic birds in wetlands should be taken:

- Poultry holdings should not be sited at wetlands.
- Ideally domestic ducks should not be reared in areas frequented by wild birds. It may be possible to reduce risks by seasonal use of the wetland *e.g*. removing domestic ducks at times of year when there are high densities of wild birds.
- A zoning approach to use of the wetlands may help although the viruses can be water-borne and thus this could be of limited value.
- The use of live decoy birds for hunting/trapping carries risks of introduction of infection and should be minimised.

At times of higher risk, *e.g.* when infection has been found within country or region, and/or during long periods of extreme weather conditions, stressors to wild bird populations (*e.g*. hunting and other disturbance) should be minimised.

If disease has been confirmed in a region:
- Extra care should be taken regarding potential for introducing infection on fomites such as footwear or vehicle tyres, using disinfection procedures, as appropriate.
- Access should be restricted during these times.
- Hunting, or other disturbing activities, should be suspended.
- Public education to raise awareness of HPAI H5N1, the risks it poses, and some simple precautions and response actions, should be given, including suspension of feeding of wild birds.

Monitoring and surveillance
Wetland managers and users should be aware of, and vigilant for, unusual mortality events of waterbirds and know how, and to whom, to report this. Early warning allows stakeholders to protect themselves and their livestock from any infection in wild birds.

Surveillance from live birds can also be conducted at wetland sites although prevalence to date has been found to be extremely low in wild birds.

Harnessing the eyes and actions of the public for early warning: a sign used at wetlands in Scotland, informing the public about surveillance activities, their role and how to report unusual mortalities (note a phone number is included).

Collecting an oropharyngeal swab from a whooper swan *Cygnus cygnus*. To date, active live wild bird surveillance to date has indicated an extremely low prevalence of HPAI H5N1 virus in healthy birds (*Taej Mundkur*).

Humans

Humans are relatively resistant to AI viruses but high standards of personal hygiene should be used when dealing with poultry or handling wild birds including hand washing and taking care to avoid rubbing eyes and touching the mouth, eating, drinking or smoking until hands are clean. Appropriate personal protective clothing should be worn.

Particular care should be taken for staff involved in disease control operations.

In areas where Eurasian lineage HPAI H5N1 is prevalent, people working in bird markets or preparing food should take particular precautions. All poultry meat and eggs should be thoroughly cooked.

In poor areas where it is typical to eat poultry even if a bird has become ill (to maximise protein availability), public education should be used to warn about the high risks associated with this practice and to minimise them.

IMPORTANCE

Effect on wildlife

LPAI viruses typically have little obvious effect on wildlife.

Eurasian lineage HPAI H5N1 viruses have caused a large number of incidents involving 100s or 1000s of wild bird deaths (mainly wildfowl and grebes). The initial confirmed outbreak in wild birds at Lake Qinghai, China, in 2005, killed 10% of the global population of bar-headed geese *Anser indicus*. The number of large scale incidents reported has declined in more recent times.

Conservation impacts have been varied and include direct mortality of birds, including threatened species. Indirect impacts, some in response to inaccurate representation of risk by media and others, include: killing wild birds as part of ill-advised disease control measures; negative perception and fearfulness of wild birds leading to some killing of wild birds and habitat destruction; the suspension of conservation projects; a reduction in garden bird feeding; a reduction of visitation at nature reserves; and the massive diversion of conservation organisations' resources from existing projects to tackling the various consequences of this disease.

Effect on livestock

The disease causes heavy losses for small scale poultry keepers as well as the poultry industry. Disease control operations involve slaughter and eradication of susceptible birds as well as infected individuals.

Effect on humans

Humans are relatively resistant to AI viruses. With respect to Eurasian lineage HPAI H5N1 viruses (although the total number of reported human cases is relatively low given the period of time it has been prevalent and the broad geographical range of the infection) the mortality rate is high (~60%).

Concerns remain about the potential for any avian influenza viruses providing the precursor for a human pandemic strain of influenza and the extreme social and economic consequences that can cause.

Economic importance

The disease has great impacts on local and national economies both in terms of costs of disease control operations but also lost revenue from trade restrictions. Costs of controlling HPAI H5N1 have run to billions of US$ since its re-emergence in 2003. Public health costs can also be prohibitive.

FURTHER INFORMATION

Useful publications and websites

- The Ramsar Convention on Wetlands. **Handbook no. 4: avian influenza and wetlands**. http://www.ramsar.org/pdf/lib/hbk4-04.pdf [Accessed March 2012].
- 10th meeting of the conference of the parties to the convention on wetlands (Ramsar, Iran, 1971). **Resolution X.21. Guidance on responding to the continued spread of highly pathogenic avian influenza**. http://www.ramsar.org/pdf/res/key_res_x_21_e.pdf [Accessed March 2012].
- 9th meeting of the conference of the parties to the convention on wetlands (Ramsar, Iran, 1971). **Resolution IX.23. Highly pathogenic avian influenza and its consequences for wetland and waterbird conservation and wise use**. http://www.ramsar.org/pdf/res/key_res_i_23_e.pdf [Accessed March 2012].
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- van Gils, J.A., Munster, V.J., Radersma, R., Liefhebber, D., Fouchier, R.A., and Klaasen, M. (2007). **Hampered foraging and migratory performance in swans infected with low-pathogenic avian influenza A virus**. *PLoS ONE,* 2(1): e184. doi:10.1371/journal.pone.0000184.
- Wildpro. **Avian influenza**. http://wildpro.twycrosszoo.org/S/00dis/Viral/Avian_Influenza.htm [Accessed March 2012].

Further information on disinfectants:

- FAO, Rome. **Manual on procedures and for disease eradication by stamping out**. (2001). www.fao.org/DOCREP/004/Y0660E/Y0660E03.htm [Accessed March 2012].

Avian tuberculosis

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Avian mycobacteriosis, avian TB, mycobacteriosis, *Mycobacterium avium* complex (MAC) disease, *M. avium intracellulare* (MAI) disease

KEY FACTS

What is avian tuberculosis?	Avian tuberculosis (avian TB) is an insidious, slowly developing, chronic bacterial disease of birds, usually affecting older individuals. The causative organism and its relatives are also capable of causing disease in a wide range of other non-avian taxa.
Causal agent	*Mycobacterium avium* complex (MAC) contains several subspecies including *Mycobacterium avium avium*, (often simply called *M. avium*), which is the principle cause of avian tuberculosis in wild, domestic and captive birds. However, a number of other species of mycobacteria may be involved such as the closely related *M. intracellulare,* or other species such as *M. genavense* (now realised to be relatively common in zoo and pet birds and clinically indistinguishable from *M. avium* infection). In general, *M. avium* should be seen as a slow growing, persistent, environmental organism with many related strains of which only some prove to be pathogenic.
Species affected	The disease has been found in a wide range of avian hosts but is most commonly reported in wild waterbirds, gregarious birds, raptors and scavengers, and those associated with agricultural premises. The disease can be relatively common in poultry where densities of birds are high, hygiene poor, and older stock are retained. The culling of poultry in commercial industrial flocks at a young age has all but eradicated the disease from these units. *M. avium* is also capable of infecting a wide range of mammals, both domestic and wild, including humans, pigs, sheep, mustelids, cervids and bovids. However, clinical disease is uncommon and may be associated with host immunocompromise. Exposure to mycobacteria in the *M. avium* complex is of importance in cattle, where sensitisation may affect tuberculin skin test results. Hence, in many regions where skin testing is used for bovine TB diagnosis, it is typical to use separate avian and bovine tuberculins to distinguish between infection with *M. bovis* and mere sensitisation to *M. avium* complex . Interestingly, *Mycobacterium avium* subspecies *paratuberculosis* (also known as MAP) is the causative agent of paratuberculosis or Johne's disease, a chronic enteritic disease of adult cattle, sheep and goats.
Geographic distribution	Reported from around the globe, and for practical purposes it can be considered to have a worldwide distribution.
Environment	The causative bacteria can live in the environment and tend to prefer damp areas with low pH. High levels of UV radiation will kill the bacteria and the majority of reports are from temperate zones rather than hot arid areas.

TRANSMISSION AND SPREAD

Vector(s)

Infected individuals provide the greatest single source of infection, however, the causative organisms are tenacious and can be carried in mud and faeces on fomites such as shoes, tyres *etc*.

How is the disease transmitted to animals?

The most common route of infection is ingestion and large numbers of bacilli may be shed in faeces from ulcerated intestinal lesions, thus contaminating the environment. High densities of animals lead to build up of faecal material providing ideal conditions for the transmission of infection.

Aerosol inhalation either from a contaminated environment, or directly from lesions in the respiratory tract of infected birds, has been suggested as the cause of pulmonary infections in domestic or captive birds, but this is relatively unusual.

Infection from an infected bird to young *via* the egg is also thought to be very unusual, and for practical purposes eggs can be seen as a good way to introduce avian TB-free birds.

How does the disease spread between groups of animals?

Close proximity of susceptible groups of animals such as pigs and poultry allows disease transfer and the feeding of poultry manure to domestic mammals provides a means for transmission of infection.

Raptors and scavenging birds may also be infected by consuming infected prey.

How is the disease transmitted to humans?

Humans are generally very resistant to *M. avium* infection, however, where there is an underlying chronic lung condition or immunocompromise, humans may be at risk. *M. avium* is a common infection in people with HIV/AIDS in the developed world however these infections are thought to be mainly environmental strains of *M. avium* rather than those of animal origin.

IDENTIFICATION AND RESPONSE

Field signs

In birds there are generally few specific signs of avian tuberculosis. Most typically there is chronic wasting with birds becoming emaciated often exhibiting a prominent keel. Birds are usually weak and lethargic, often with poor or ruffled plumage. In late stages of the disease, abdominal distension as a result of liver enlargement and a build up of ascitic fluid can give an emaciated bird an unusual ‘bottom heavy’ appearance. Lameness is relatively common if there is bone involvement. Diarrhoea is common whether chronic or intermittent. Ceres and other areas of exposed skin may become progressively paler as the disease progresses. Respiratory involvement is relatively unusual but this may result in wheezing. Alternatively birds may just be found dead or succumb to another cause of death before these clinical signs are apparent.

In cattle, *M. avium* complex infection is an uncommon cause of disease, but may cause localised abscesses or mastitis. Johne’s disease often presents as progressive weight loss and reduced milk production.

In deer, *M. avium* complex infection may cause progressive weight loss, emaciation and diarrhoea.

In pigs, there are generally no obvious signs of disease with evidence of infection being found at slaughter in either or both the lymph nodes around the neck or those draining the intestine.

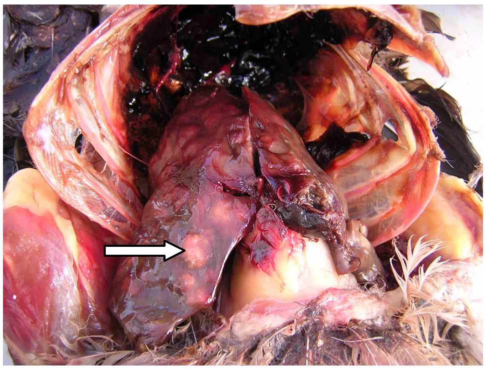
Recommended action if suspected

The disease is not notifiable to the OIE but prevention of establishment of the disease is highly desirable as control thereafter is complex and often unsuccessful. ► **Prevention and control in wetlands below.**

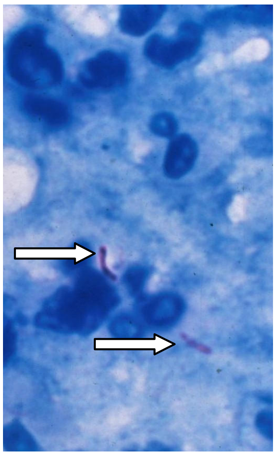
Diagnosis

In live birds the disease is difficult to diagnose, and diagnosis relies on a combination of laboratory tests such as abnormal blood cell counts and/or finding bacteria in the faeces. More often the diagnosis is reached at *post mortem* examination, based on the presence of acid-fast bacilli within tuberculous granulomatous lesions in affected tissues. Microscopy using a modified Ziehl Neelsen stain (see images below), or further laboratory tests (*e.g.* molecular probes), are necessary to confirm the presence of the causative bacilli. A whole bird carcase is ideally required for *post mortem* examination. When this is not possible, the liver, kidneys and intestines or any other obviously affected tissues should be submitted to the diagnostic laboratory.

Similarly, in mammals, diagnosis is often made at *post mortem* examination.



At *post mortem* examination a shot pink-footed goose *Anser brachyrhynchus* is found to have typical tuberculous lesions in its liver.



In a stained slide of a smear from the liver lesions, the causative bacteria show up as pink rods.

PREVENTION AND CONTROL IN WETLANDS

Environment

For domestic stock, maintaining **high standards of hygiene** by good cleansing and disinfection helps to minimise spread of *M. avium,* although it is important to note that the bacteria have a tough cell wall which makes them resistant to many disinfectants.

The bacteria prefer a low **pH** and increasing this may help reduce environmental contamination *e.g.* by the addition of lime (noting that changing pH will affect vegetation and associated invertebrate communities also). **Cutting back vegetation** and **turning soil** to expose it to UV radiation will help to reduce environmental contamination.

Livestock

Action should be directed firstly towards preventing the introduction of infection, as subsequent control can be very difficult. Good biosecurity practices will help to reduce risk of introducing *M. avium,* including purchasing animals/eggs from known avian TB-free stock. Good surveillance ensures any problems can be dealt with quickly before infection becomes established. Diagnosis of the disease in poultry ideally should prompt a policy of culling of the flock. In addition, cleansing and disinfection is important, as subclinically infected animals and environmental contamination may result in the disease becoming endemic.

For poultry, keeping the **age structure** young and slaughtering early provides a powerful means by which to control the disease.

The disease is often slow to progress and con-current infections or stress can allow activation or reactivation of subclinical infection, hence efforts should be made to reduce both of these contributory factors.

Wildlife

Contact with domestic/captive birds should be avoided. **High densities of wildlife** represent a risk factor for this disease and practices such as supplemental feeding of wildlife can contribute to this risk.

As for poultry, **stress** may play an important role in allowing a subclinical infection to develop into full-blown disease hence efforts should be made to mitigate against other stressors such as poor nutrition, pollution, con-current infections, disturbance *etc*.

Humans

General standards of **personal hygiene** are sufficient to reduce risk to most humans in and around wetlands and infected animals.

IMPORTANCE

Effect on wildlife

In most situations, the disease is likely to have relatively limited impact on wildlife other than as an occasional cause of death. However, it has been a problem for several threatened species such as the whooping crane *Grus americana* in North America, and the lesser flamingo *Phoeniconaias minor* in east Africa. It can be a problem where wild birds are attracted to wetlands where infected captive birds are maintained. Overall, efforts should be made to prevent infection becoming established in wild populations.

Effect on livestock

The greatest impact is on poultry flocks where control actions involve culling.

Effect on humans

Public health concerns are relatively limited although care should be taken if it is known that infection is present, to reduce potential for opportunist infections. High risk (*e.g.* immunocompromised) individuals should take extra precautions in such situations.

Economic importance

Where the disease is diagnosed in industrial units, and culling, cleansing and disinfection measures are required, economic losses can be significant. Within smaller flocks the loss of production and general unthriftiness of animals is of importance.

FURTHER INFORMATION

Useful publications and websites (Avian TB)

- Friend, M. & Franson, J.C. (2001). **Avian tuberculosis**. In: Field manual of wildlife diseases: general field procedures and diseases of birds. E. A. Ciganovich (ed.). pp 93-98. U.S. Department of the Interior and U.S. Geological Survey, Washington, DC. www.nwhc.usgs.gov/publications/field_manual/chapter_8.pdf [Accessed March 2012].
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- Merck & Co. Inc. **The Merck veterinary manual: tuberculosis**. www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/204400.htm&word=avian%2ctuberculosis [Accessed March 2012].
- Wildpro. **Avian tuberculosis**. http://wildpro.twycrosszoo.org/S/00dis/Bacterial/Avian_Tuberculosis.htm [Accessed March 2012].

Useful publications and websites (Johne's Disease, paratuberculosis)

- European Association of Zoo and Wildlife Veterinarians. **Johne's Disease**. http://www.eaza.net/activities/tdfactsheets/091%20Paratuberculosis%20Or%20Johne%20s%20Disease.doc.pdf [Accessed March 2012].
- Centre for Food Security and Public Health (CFSPH). **Johne's Disease Factsheet**. www.cfsph.iastate.edu/Factsheets/pdfs/paratuberculosis.pdf [Accessed March 2012].

Bovine tuberculosis

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: bovine TB

KEY FACTS

What is bovine TB?

Bovine tuberculosis (TB) is a significant zoonotic disease which affects cattle and other domestic and wild mammals and is transmissible to humans. Eradication programmes in most developed countries have reduced or eliminated bovine TB in cattle and subsequently human disease is rare, however, complete eradication is difficult as wildlife may act as reservoirs for the disease. In many less developed countries bovine TB is common and creates public health concerns, economic losses resulting from livestock deaths, persistent disease and trade restrictions. This disease is typically spread to humans by inhalation of aerosols, or ingestion of contaminated unpasteurised milk (relatively rare).

Causal agent

Mycobacterium bovis (*M. bovis*), a Gram positive, acid-fast bacterium in the *Mycobacterium tuberculosis* complex of the family Mycobacteriaceae.

Species affected

The primary hosts for *M. bovis* are cattle but a broad range of domesticated and wild mammals may also be infected. High profile and well studied apparent wildlife reservoirs of infection include badgers *Meles meles* in the UK and Ireland and brushtail possums *Trichosurus vulpecula* in New Zealand. The disease has a broad host range and numerous wildlife species have been affected to varying degrees including kudu and African buffalo *Syncerus caffer* in southern Africa and bison and elk *Cervus canadensis* in Canada. The disease has also been described in wild felids, deer, elephants, rhinoceroses, hares, raccoons, bears, warthogs, primates, opossums, foxes, coyotes, mink, otters, seals, sea lions, deer, elk and some rodent species. In general the wetland manager should consider all wild mammals to be potentially susceptible to infection.

Domestic species known to be susceptible include dogs, cats, pigs, ferrets, camelids, sheep, goats and horses.

Although generally thought to be resistant there is little known about the susceptibility of birds to *M. bovis*.

Geographic distribution

Once found worldwide but now 'kept at bay' in domesticated animals in many countries due to control programmes. Bovine TB remains widespread in Africa, parts of Asia and some Middle Eastern countries. Eradication programmes are underway in some countries of Central and South America, the United States, Mexico, New Zealand, Japan and Europe. It is important to periodically consult the OIE and wider literature as whilst a country may be currently classified as bovine TB free or under eradication, this may sporadically change if some herds become infected. Complications in eradication efforts occur particularly where wildlife are involved in the epidemiology of the disease such as infection in wild white-tailed deer in parts of the USA, badgers in the UK and Ireland, and brushtail possums in New Zealand.

Environment

Wetlands supporting groups of susceptible animals.

Survival of *M. bovis* in the environment is primarily affected by exposure to sunlight. In cold, dark and moist conditions it can survive for several months and at 12-24°C (54-75°F), depending on the exposure to sunlight, survival time varies from 18 to 332 days. Studies showed *M. bovis* remained viable for four to eight weeks in dry or moist soil samples in 80% shade [34°C (93°F)] and another showed it was destroyed within four days in either summer or winter on New Zealand pastures.

TRANSMISSION AND SPREAD

Vector(s)

As discussed above, maintenance and spillover hosts may both act as disease vectors. The bacterial agent may be carried on the clothing or shoes of personnel in contact with infected animals.

How is the disease transmitted to animals?

The main source of transmission is an infected animal. *M. bovis* may be transmitted by aerosol inhalation (organisms excreted in exhaled air), secretions and excretions, by ingestion (contaminated food/water) or by cutaneous infection (through wounds or abrasions). Genital and congenital infections occur but are rare. The chief mode of transmission is exchange of respiratory secretions between infected and uninfected animals and ingestion of infected milk for calves. Population densities and social structure can be key in *M. bovis* transmission which usually occurs when animals are in close contact.

Humans have been known to transmit *M. bovis* to cattle, which is linked to genitourinary TB, and most reported cases are associated with urination in cowsheds.

How does the disease spread between groups of animals?

Infection has been observed to spread in both directions between livestock and wildlife, when both share the same environment and food. Examples of such spread include infection in badgers in the UK and possums in New Zealand. Potential routes of transmission include by aerosol, when in close proximity, and by ingestion when feeding in contaminated environments. In pigs, ferrets and most likely deer, ingestion seems to be the primary route of transmission. Cats may be infected *via* ingestion or percutaneous transmission in bites and scratches or by the respiratory route. Non-human primates are typically infected by inhalation. Predatory and scavenging animals are infected from consumption of infected prey. In the case of badgers, aerosol transmission would appear to be the main route with biting being an additional possibility. *M. bovis* may be shed in the urine and the faeces of infected badgers with advanced disease.

How is the disease transmitted to humans?

M. bovis can be transmitted to humans in a number of ways, primarily through ingestion of unpasteurised milk and other dairy products, and inhalation of aerosols. Although rare, agricultural workers in contact with infected livestock are at risk of developing pulmonary bovine TB by inhaling aerosolised bacteria. Infection may also be caused by ingestion of raw or undercooked meat and through breaks in the skin.

Person-to-person transmission is possible, particularly in immunocompromised humans, alcoholics or HIV-infected individuals but evidence for extensive human-to human transmission is limited.

IDENTIFICATION AND RESPONSE

Field signs

In cattle, early infections are often asymptomatic, but in the late stages common symptoms include a low–grade fluctuating fever, weakness and inappetence, progressive emaciation and animals with pulmonary association typically have a moist cough. Animals may become acutely emaciated and develop severe respiratory distress in the terminal stages.

In cervids, infections may be subacute or cause chronic disease with variable rates of progression. Some animals may only show abscesses of unknown origin with additional symptoms developing years later and other cases may exhibit rapid dissemination with relatively quick onset of symptoms.

In general, any field signs seen depend on the host species. Often there may be no obvious clinical signs. The most likely presentation in wildlife such as wild ungulates and carnivores (*e.g.* lions) with advanced disease, is progressive wasting, emaciation and weakness, possibly with coughing in the former.

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals. Bovine TB is a notifiable disease and suspected cases must be reported immediately to local and national authorities and the OIE.

Diagnosis

Based on clinical signs alone, bovine TB can be very difficult to diagnose and there are numerous other conditions which display similar signs (including a broad range of bacterial and parasitic infections). In developed countries most infections in domestic livestock are diagnosed by routine testing or found at the slaughterhouse.

Bovine TB may be diagnosed in live cattle in the field with the tuberculin skin test. A strong skin-based immune response to bovine tuberculin is consistent with infection. In many instances this is performed in a comparative manner, using avian tuberculin in addition to bovine. The magnitude of the avian response is taken into consideration when determining positive or negative status. All skin tests are two step procedures involving tuberculin injection on day one, and a reading of the skin response 72 hours later. Presumptive testing may be carried out using histopathology and/or the microscopic demonstration of acid-fast bacilli, where direct smears from clinical samples or tissues (usually collected *post mortem*) may be stained with the Ziehl Neelsen stain, a fluorescent acid-fast stain or immunoperoxidase techniques. Confirmatory testing involves isolation of *M. bovis* on selective culture media, which are incubated for eight weeks. The organism can be confirmed with biochemical tests or polymerase chain reaction (PCR) assays (including spoligotyping which can both confirm and type bacteria).

Blood-based tests for immune responses to *M. bovis* include the lymphocyte proliferation and gamma-interferon assays and serological tests. For the diagnosis of infection in animals that are difficult to capture or handle (wildlife or zoo animals), blood based tests may be more useful than the skin tests as only one capture event is required.

In cervids, bovine TB should be considered as a differential diagnosis when abscesses of unknown cause are found.

PREVENTION AND CONTROL IN WETLANDS

Environment

In a wetland setting, disinfection is unlikely to be considered a viable control measure. In domestic animal housing, however, **disinfection and sanitisation** may help minimise spread of *M. bovis* within a herd. It is important to use an effective disinfectant, such as 5% phenol, iodine solutions with a high concentration of available iodine, or glutaraldehyde, as *M. bovis* is moderately resistant and long contact times are necessary for inactivation. On infected farms rodent control may be advisable given these species may become infected and may be able to transmit infection more widely.

Livestock

The insidious, chronic nature of this disease makes prevention or early detection and control imperative. The most effective method to eradicate bovine TB from domesticated animals is the **test-and-slaughter** technique. However, eradication efforts can be complicated by the occurrence of *M. bovis* in wildlife reservoir hosts.

Summary of some methods to decrease the risk of bovine TB in cattle:

- Where possible maintain a closed herd (a herd with animals all bred from within the same herd).
- Limit opportunities for contact with neighbouring herds.
- Isolate and test purchased stock.
- Isolate and test cattle re-entering the herd.
- Enforce biosecurity on premises to prevent contact with cattle of unknown bovine TB status.
- Develop and implement a herd health programme (record individual records).
- Keep stocking densities low.
- In collaboration with the authorities conduct routine diagnostic tests and report suspected cases and dead animals.
- If suspected cases confirmed then quarantine the animals and bovine TB test the rest of the herd and re-tested periodically.
- Develop a bovine TB testing policy for employees.
- Control of wildlife reservoirs or means by which to isolate livestock from the reservoir.

Carcases with confirmed bovine TB should not be used for human consumption and the herd of origin of the infected carcase should be bovine TB tested.

A badger entering a cattle shed, and a badger-proofed shed, which is a relatively straightforward means by which contact between livestock and wildlife can be reduced (*Fera*).

Wildlife

However desirable, there are many difficulties in controlling the disease in wildlife. Control can be achieved to some extent by using a combination of surveillance and management to monitor and control the spread and occurrence of the disease. Within *well managed* strategies, **culling** of infected wildlife may be considered but *ad hoc* or even well planned culling may not bring benefits and may even exacerbate the problem. Also, this measure is unlikely to constitute an 'ecosystem approach' to health.

Restricting access of wildlife to infected domestic herds helps to reduce risk. This might be achieved in various ways including use of physical barriers to restrict wildlife access to domestic animal housing.

In some wildlife populations **reducing population density** and/or **changing social behaviour** can help to reduce risk. This may be achieved in a number of ways including *not* providing supplementary food which can maintain animals above a carrying capacity for an area and not using feeding stations (for *e.g.* hunter or tourist interests) to reduce risk of transmission at these localised feeding sites.

Vaccination is a possibility for control of the disease in wildlife (primarily to reduce risk to livestock). However, the only TB vaccine currently licensed for use in wildlife is an injectable BCG vaccine for badgers in use in the UK.

Humans

Humans should protect themselves by wearing **protective clothing** (including gloves, masks) when dealing with infected animals as infections in humans are difficult to treat. **Cooking meat thoroughly** or **pasteurisation** of milk and other dairy products reduces risk of infection.

IMPORTANCE

Effect on wildlife

In some situations *M. bovis* may be a serious threat to wildlife in particular where disease becomes endemic and present in a wide range of hosts (*e.g.* some southern African protected areas). It can affect common and threatened species alike and in some species (*e.g.* lions) has been found to negatively affect social structures and ultimately populations. In Spain, *M. bovis* infection is a cause of serious concern for the conservation of the highly endangered Iberian lynx *Lynx pardinus*.

Effect on livestock

Bovine TB is of significant importance to the cattle industry in terms of loss of production, control measures and trade restrictions. Presence of the disease may also lead to loss of consumer confidence in milk and beef products. Potential human health risks in the developing world, in particular, and the additional potential for infection in a wide range of hosts including free-roaming wildlife increases the need for control in domestic situations.

Effect on humans

Public health concerns arise from the possibility of human infection with *M. bovis* through the consumption of unpasteurised dairy products or meat from infected animals. Although rare in countries with bovine TB eradication programmes and pasteurised milk, it is still a significant concern in countries where the disease is poorly controlled. Incidence appears higher in personnel that work closely with cattle such as farmers and abattoir staff. It has also been documented that humans can be infected by exposure to other species, including goats, farmed elk and even rhinoceros. In countries where bushmeat is eaten wildlife species may be a particular source of infection. In some communities the close contact of humans and animals may facilitate disease transmission, for example, in some African countries cattle are an integral part of life and are present at ceremonies representing wealth and animals working in agriculture. People infected with HIV are also at increased risk from opportunistic bovine TB infections.

Economic importance

Annual economic losses to countries with bovine TB can reach many millions of US dollars. Bovine TB is also important due to potential impacts on the meat and live animal export trade, and expansion of the dairy industry may be severely limited at regional and national levels. Cost of control measures both in livestock and wildlife can be significant.

FURTHER INFORMATION

Useful publications and websites

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Brucellosis

Wetlands supporting groups of suspectible mammals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: Bang's disease, contagious abortion, enzootic abortion, epizootic abortion, Malta fever, Mediterranean fever, undulant fever

KEY FACTS

What is brucellosis?

A chronic and contagious bacterial disease of domestic and wild animals that may be transmitted to humans. In animals, it causes reproductive problems (*e.g.* abortions, stillbirth and infertility) and other signs, including arthritis in cows and pigs, mastitis and lameness in goats, and oozing skin lesions in horses. In humans, it causes influenza-like symptoms which can be severe and last for months and can be confused with malaria and typhoid.

Causal agent

Bacteria of the genus *Brucella*, infections are mainly caused by *B. abortus, B. melitensis, B. ovis, B. canis,* and *B. suis.*

Species affected

Many species of terrestrial and marine mammals, particularly cattle, swine, bison, elk *Cervus canadensis*, deer, goats, sheep, other ruminants and humans. Wildlife reservoirs do exist and can include feral pigs, bison, and elk amongst others.

Geographic distribution

Present to varying degrees in most countries of the world. High risk areas are the Mediterranean Basin (Portugal, Spain, Southern France, Italy, Greece, Turkey, North Africa), South and Central America, Eastern Europe, Asia, Africa, the Caribbean, and the Middle East.

Environment

Any environment supporting groups of suspectible mammals.

TRANSMISSION AND SPREAD

Vector(s)

Although the bacteria is not vector-borne, it may be spread mechanically by infected animals and contaminated objects such as equipment, clothing, shoes, feed or water.

How is the disease transmitted to animals?

Direct contact with infected animals or with an environment that has been contaminated with birthing tissues or, most commonly, fluids from infected animals (*e.g.* aborted foetuses, vaginal discharges). Animals may lick those materials or the genital area of other animals or ingest the disease-causing organisms with contaminated food or water. Venereal transmission is the most common means of spread but the bacteria can also be found in milk, blood, urine and semen.

How does the disease spread between groups of animals?

Brucellosis is usually spread from one animal group to another by an infected or exposed animal, *e.g.* by adding infected animals to a domestic herd or by infected animals mingling with brucellosis-free groups. Brucellosis can also be spread by contaminated objects (fomites) such as equipment, clothing, shoes, feed or water.

How is the disease transmitted to humans?

Direct contact with tissues or fluids from infected animals and by eating contaminated food, especially unpasteurised dairy products. Person-to-person transmission is very rare but has occurredthrough transplants, sexual intercourse, or from mother to child.

IDENTIFICATION AND RESPONSE

Field signs

There is no effective way to detect infected animals by their appearance. The most obvious sign is abortion or birth of weak young. Milk production may be reduced, and other signs include an apparent lowering of fertility with poor conception rates, retained afterbirths with resulting uterine infections, and (occasionally) enlarged, arthritic joints.

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals. Brucellosis caused by *B. abortus*, *B. mellitensis* or *B. suis* is notifiable to the OIE and suspected cases in livestock and humans should be reported to local and national authorities.

Diagnosis

Confirmation is made with prescribed laboratory tests to isolate and identify the bacteria, through serological testing, or a combination of both, following OIE guidelines.

PREVENTION AND CONTROL IN WETLANDS

Environment

Brucella can survive for months in the environment under optimum conditions but can be destroyed by heat and some common disinfectants.
►**Section 3.4.1. Disinfection and sanitation**

Livestock

The disease in livestock may be avoided by employing good sanitation and animal management practices *e.g.*

- Preventing the introduction of infection through movement controls, testing and quarantine.
- Detecting any infected animals in the population as early as possible through surveillance, and thoroughly investigating all suspect cases.
- Eliminating any confirmed infection found in livestock through the slaughter of infected and exposed animals.
- Vaccination with an approved vaccine can be effective.
- Cleaning and disinfection of calving areas and other places likely to become contaminated with infective material.
- Placing barriers around stored feed and utilising biosecurity measures to decrease interaction between wildlife and livestock in areas with a wildlife reservoir.

Wildlife

Control of the infection in wildlife requires management at the ecosystem scale. Eradication in wildlife is probably not feasible, but the following measures can help reduce prevalence:

- Preventing and controlling infection in domestic animals.
- Avoiding provision of artificial feeding grounds which concentrate susceptible animals (if existing, slowly phase-out).
- Protecting existing habitat and migration corridors (and increasing them where possible).
- Avoiding test-and-slaughter programmes as these have not been shown to control the disease but have been shown to exacerbate spread.
- Vaccination may be possible on a wildlife-appropriate scale if well thought-out and modelled beforehand.

Humans

Risks to humans can be reduced by:

- Not eating or drinking raw or unpasteurised dairy products.
- Wearing protective clothing (gloves, masks) when handling reproductive tissues (assisting delivery of newborn animals).
- Always washing hands after touching animals.

IMPORTANCE

Effect on wildlife

There is evidence of widespread infection in some populations. The disease causes little morbidity or mortality, but effects at the population level are largely unknown. It can result in a negative perception of wildlife and increase exposure of wildlife to brucellosis (and additional diseases) through practices used to control movement, *e.g.* provision of feeding sites and fencing.

Effect on livestock

Deaths are rare except in unborn animals, but the disease can be debilitating with obvious loss of productivity and welfare implications.

Effect on humans

Human infection frequently occurs in regions where brucellosis persists in domestic animals. It is an important human disease in many parts of the world especially in the Mediterranean countries of Europe, north and east Africa, the Middle East, south and central Asia and Central and South America and yet it is often unrecognised and unreported.

Economic importance

In developing countries, the disease in livestock has serious impacts on the livelihoods of farmers and may pose a barrier to trade or increase costs to farmers for testing and vaccination. The illness in humans is multisystemic and can result in economic losses due to the time lost from normal activities.

FURTHER INFORMATION

Useful publications and websites

- World Health Organization (WHO). **Brucellosis in humans and animals**. http://www.who.int/csr/resources/publications/Brucellosis.pdf [Accessed March 2012].
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- World Health Organisation (WHO). **Brucellosis**. www.who.int/zoonoses/diseases/brucellosis/en/ [Accessed March 2012].

Contacts

✉ **WHO Communicable Diseases Surveillance and Response (CSR)**.
Email: zoonotic_alert@who.int fmeslin@who.int and outbreak@who.int

🖱 **FAO Animal Production and Health Division**.
www.fao.org/ag/againfo/home/en/who.htm

Campylobacteriosis

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: *Campylobacter enteritis*, vibrionic enteritis, vibriosis

KEY FACTS

What is campylobacteriosis?

An infectious disease of humans and a range of animals including birds caused by their exposure to species of *Campylobacter spp.* bacteria. The bacterium is found commonly in the intestines of healthy livestock and poultry but also in most species of wild mammals and birds, other wildlife and the environment, surviving in mud slurries and polluted water for up to three months. The prevalence of infection in animals is much higher than the incidence of disease. Most *Campylobacter spp.* do not cause any signs of illness in the animal host although some may cause diarrhoea and sporadic cases of abortion in ruminants. The infection can spread rapidly between animals, particularly when they are gathered in dense concentrations.

Campylobacter spp. remain one of the main causes of gastroenteritis in humans globally. Humans usually contract the bacteria through the consumption and handling of contaminated meat and water but also through direct contact with infected animals and their faeces. Illness usually occurs in single, sporadic cases, but it can also occur in outbreaks, when a number of people become ill at one time.

Causal agent

Fourteen species of bacteria from the genus *Campylobacter: C. coli, C. concisus, C. curvus, C. fetus, C. gracilis, C. helveticus, C. hyointestinalis, C. jejuni, C. lari, C. mucosalis, C. rectus, C. showae, C. sputorum* and *C. upsaliensis.* Campylobacteriosis in humans is mainly caused by *C. jejuni* and, to a lesser extent, *C. coli.*

Species affected

Many species of domestic and wild animals including cattle, sheep, goats, pigs, dogs, cats, poultry (including ducks and geese), wild birds, rodents and marine mammals. Humans are very susceptible to illness caused by certain *Campylobacter spp.* bacteria.

Geographic distribution

C. jejuni, C. coli and *C. fetus* infections are found worldwide. The importance of each *Campylobacter spp.* differs between geographical regions. In humans, infections are particularly common in very young children in developing countries and young adults in developed countries.

Environment

Any environment supporting *Campylobacter spp.* and their animal hosts.

TRANSMISSION AND SPREAD

Vector(s)

The bacterium is not vector-borne but may be spread mechanically through infected animals and contaminated objects such as equipment, clothing, shoes, feed and water. Flies can also act as mechanical vectors for *Campylobacter spp.*

How is Campylobacter transmitted to animals?

Direct contact with infected faeces, vaginal discharges and abortion products and through ingesting water and food contaminated with bacteria. Water courses can easily become contaminated from infected faeces of livestock and wild birds. Flies can also act as mechanical vectors for *Campylobacter spp.*

How does Campylobacter spread between groups of animals?

Spread from one animal group to another by an infected animal which will shed the bacteria into the environment in its faeces. Bacteria may also be introduced to herds and flocks on shoes, equipment and other contaminated objects (fomites). Exactly how the infection spreads between and within herds and flocks is not fully understood due to the difficulties of detecting clinical signs in animals.

Few studies exist of the transmission between wild and domestic animals, but what evidence there is suggests this is rare.

How is Campylobacter transmitted to humans?

Most commonly transmitted by handling and ingesting contaminated food, particularly undercooked poultry, meat and unpasteurised milk, or from cross-contamination of other foods by these items, and through drinking contaminated water. Also transmitted through direct contact with infected animals and their faeces and may be spread through person to person contact if hygiene is poor. There is some evidence that feral and domestic pigeons in peri-domestic settings can carry *C. jejuni* and potentially transmit this agent to humans through the environment.

IDENTIFICATION AND RESPONSE

Field signs

Infected animals, both domestic and wild, may have diarrhoea but many will not show any symptoms and hence *Campylobacter spp.* can be difficult to detect. *Campylobacter spp.* may cause enteritis and infections by *C. fetus* may cause infertility and spontaneous abortion in sheep and cattle.

Humans may suffer from watery or bloody diarrhoea, abdominal pain, fever, headache, nausea and vomiting. Symptoms usually start 2–5 days after infection and last for 3–6 days. Some infected people do not show any symptoms at all.

Recommended action if suspected

Contact and seek assistance from human and animal health professionals immediately if there is suspected infection in people and/or livestock. An outbreak may mean that many humans and animals are exposed to a common contaminated food item or water source.

Diagnosis

Isolation of the causative agent by health professionals is needed for a definitive diagnosis. Faeces or blood cultures are used for isolating the bacteria in humans, and in mammals and birds, faeces, rectal swabs and/or caecal contents are required. Ideally, fresh faeces should be collected, preferably without traces of urine. Samples should be prevented from drying out. A medium should be used for transporting swabs.

In dead birds, the caecum is usually used for the detection of *Campylobacter spp.* and can be cut with sterile scissors from the remaining part of the intestines and submitted intact to the laboratory in a plastic bag or petri-dish. Samples from dead cattle, sheep and pigs are collected from the intestines by aseptically opening the gut wall. Samples should ideally be transported to the laboratory the same day but if not, within two days. Samples must be protected from light and not kept in high (>20°C) or low (<0°C) temperatures. Storage at 4°C is recommended.

PREVENTION AND CONTROL IN WETLANDS

Overall

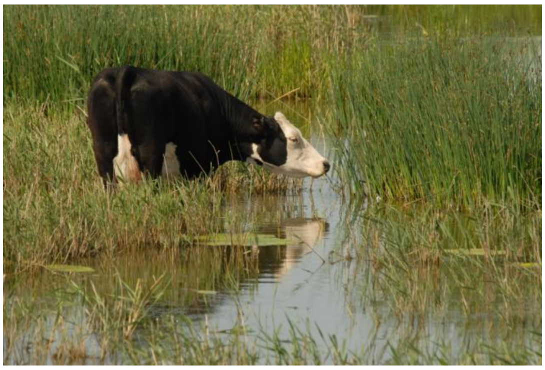
Prevention and control measures are limited in wetlands with free-living animals, many of which will carry the bacteria without any noticeable signs and untoward effects. Transmission of bacteria from animals to humans and between captive animals can be more easily prevented and controlled.

Monitoring and surveillance
Recording the incidence of outbreaks can identify trends in *Campylobacter spp.* infections and evaluate the feasibility of control programmes. Monitoring of outbreaks in animals and humans can also help assess the contribution of animals to human illness.

Livestock

The control of *Campylobacter spp.* along the food chain is most effective when the colonisation of living animals with bacteria can be prevented.

- **Good biosecurity** will help protect captive animals from bacteria and prevent cross-contamination:
  - Have disinfection facilities for hands, footwear, clothing, equipment and vehicles/trailers on entering or leaving areas with livestock and after contact with animals.
  - Wear protective clothing and footwear, either disposable or easily disinfected re-usable clothes (*e.g.* waterproof clothing, face shields, gloves and boots).
  - Have separate clothing and equipment for each person using areas with livestock.
  - Note that biosecurity does not guarantee a *Campylobacter spp.*-free flock or herd at the time of slaughter.
- **Vector control** - although not the most important mode of transmission, vector control will help prevent/reduce flies mechanically transferring *Campylobacter spp.* to other animals.
  ►Section 3.4.3. Control of vectors
- **Fence stream banks and watering holes** to limit access by livestock to water contaminated by faeces from infected animals and to reduce animals contaminating water courses. Provide clean drinking water in separate watering tanks located away from potentially contaminated water bodies.
- **Sewage treatment** to reduce release of bacteria into water courses.
- **Chlorinate** contained drinking water sources and prevent faecal contamination of food and water where possible. Do not chlorinate natural water bodies as this will have an adverse effect on the wetland ecosystem.
- **Avoid mixing** potentially infected and susceptible pregnant animals.
- **Vaccination** can prevent abortions in sheep and may be used as prophylaxis for bovine genital campylobacteriosis. Note that vaccinated cows may remain carriers of the bacteria.
- Use of **artificial insemination** techniques rather than natural insemination can control or prevent bovine genital campylobacteriosis.
- **Antibiotics** may be used to treat some cases of enteritis and may also prevent sheep and cattle from aborting during an outbreak.



If livestock are known to be infected with *Campylobacter spp*. they should not be allowed access to wetlands as this can pass on infection to other livestock, wildlife and humans. Fencing can be used and water provided in troughs (*James Lees*).

Wildlife

Campylobacter spp. are carried by most mammals and birds and are commonly found in water sources. Disease is largely uncommon in wild animals therefore control measures are limited. To protect wildlife, wetland management should focus on reducing sources of human and livestock faecal contamination of wetlands.

► **Humans**
► **Livestock**

Humans

- **Appropriate slaughtering and meat preparation** processes can reduce the risk of contaminating carcases with bacteria and can decontaminate infected meat.
- **Avoid consuming unpasteurised** dairy products and eggs and untreated surface water. Other foods, especially meat should be cooked thoroughly and fruit and vegetables should be peeled or washed thoroughly with uncontaminated water. Prevent contamination of food in the kitchen.
- **Good personal hygiene** including washing hands thoroughly with soap and warm water: before preparing and eating food; after handling raw food; after going to the toilet or changing a baby's nappy; after contact with animals; frequently if you have symptoms such as diarrhoea.
- If campylobacteriosis is suspected, thoroughly wash all dirty clothes, bedding and towels in hot water. **Clean** and **disinfect** toilets, sinks and taps.
- Most people who have *Campylobacter spp.* recover without treatment. It is important to **drink plenty of fluids** as diarrhoea or vomiting can lead to dehydration and loss of minerals. Re-hydration solutions may also be useful. Antibiotics may be given to treat severe infections.

IMPORTANCE

Effect on wildlife

Campylobacter spp. are not uncommonly found in most species of mammal and bird. However, the prevalence of infection in animals is much higher than the incidence of disease and many infected mammals and birds may not show any signs at all. That said, it can occasionally cause mortality in both taxa and may be of greater importance in hosts with con-current disease or subject to other stressors.

Effect on livestock

Whilst some infected animals may show mild signs such as diarrhoea, many will not show any signs at all. Mortality may be high in young farmed birds but low in older birds and adult sheep and cattle. Some infections may cause infertility and spontaneous abortion in sheep and cattle.

Effect on humans

Whilst most cases in humans are relatively mild, a small proportion may develop more severe illness. Death is rare in healthy individuals but may occur in cancer patients or those that have compromised immune systems. Worldwide, campylobacteriosis is responsible for around 5-14% of all cases of diarrhoea.

Economic importance

There is potential for significant economic losses to the livestock industry, with poultry particularly affected, due to illness of infected animals and likely trade restrictions imposed during and after an outbreak.

Illness in humans can result in significant economic losses due to the time lost from normal activities.

FURTHER INFORMATION

Useful publications and websites

- World Organisation for Animal Health (OIE). **Chapter 2.09.03: *Campylobacter jejuni* and *Campylobacter coli***. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.09.03_CAMPYLO.pdf [Accessed March 2012].
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Contacts

- **WHO Communicable Diseases Surveillance and Response (CSR)**. zoonotic_alert@who.int, fmeslin@who.int and outbreak@who.int
- **FAO Animal Production and Health Division**. www.fao.org/ag/againfo/home/en/who.htm

Coral diseases

Coral reefs

Wildlife ✓
Livestock ✗
Human ✗

Synonyms: Aspergillosis, black band disease, Caribbean ciliate infection, red band disease, ulcerative white spots, white band disease, white patch disease, white plagues, yellow band disease

KEY FACTS

What are coral diseases?	Coral diseases are a number of diseases that lead to the damage of corals and their structure. The diseases are multifactorial in nature and lead to the production of lesions on the coral. The exact origin and cause of these diseases is often unknown and where agents have been identified they are often part of complex interactions with the environment and other organisms. The diseases can be described as pigmented band diseases, focal or multifocal tissue loss without distinct pigmented band, annular or linear tissue loss without distinct pigmented band, discolouration and growth anomalies.
Causal agent	Virtually all of the most pervasive threats impacting coral reef ecosystems (including land-based and marine pollution, overfishing, global climate change, and ocean acidification) have been suggested as synergists or facilitators of infectious disease. Factors shown to stress the coral or lead to compromised health (*e.g.* predation) increase the likelihood of disease occurring. The causes of coral diseases are multifactorial and have often not yet been fully identified. Pathogens that have been suggested as causal agents of disease in corals include bacteria (*e.g. Vibrio spp.*), fungi (*e.g. Aspergillus spp.*) and protozoa.
Species affected	Many species are affected – most falling into either the Subclass Octocoralia (soft corals) or Order Scleratinia (true stony corals).
Geographic distribution	Worldwide (including the Western Atlantic, Indo-Pacific, East Africa, the Red Sea and Australia) with the Caribbean described as a hotspot because of rapid emergence and spread of virulent diseases. Diseases in Pacific-based corals have been increasingly reported as more surveys have been carried out in different locations.
Environment	Marine ecosystems.

TRANSMISSION AND SPREAD

Vector(s)	Coral predators and humans may transfer diseases between corals.
How is the disease transmitted to animals?	These diseases can be spread between corals by direct contact or, potentially, by coral predators and humans. Disease often occurs secondary to environmental changes or trauma.
How does the disease spread between groups of animals?	Direct contact between corals, water-borne contact, environmental changes, human interaction.
How is the disease transmitted to humans?	These diseases are not thought to be zoonotic.

IDENTIFICATION AND RESPONSE

Field signs

Lesions on coral – both of known or unknown cause. These lesions can include tissue loss, bleaching, pigmentation changes (*e.g.* in bands or patches) and growth anomalies.

Recommended action if suspected

If a lesion is present, record host affected, whether or not there is a known cause (*e.g.* fish predation, gastropod predation, galls, algal abrasion/overgrowth, Crown of Thorns Starfish predation, sediment damage *etc*.). Also record lesion type (*e.g.* tissue loss, growth anomaly, tissue discolouration, overlying pigmented material) and also lesion pattern (focal, multifocal or diffuse), rate of progression (rapid, moderate or not progressing), colour, and lesion margin (describe colour, thickness, shape and border type *e.g.* discrete or diffuse). Develop a monitoring programme to help address impacts of disease on coral communities (*e.g.* determine how widespread the disease is, how fast it is spreading and if the disease is fatal to the animals affected). Depending on local arrangements, report suspected cases to national authorities.

Diagnosis

Liaise with appropriate experts regarding collection of samples for laboratory investigations prior to any samples being taken. If tissue loss is visible look for potential predators in the surrounding area.

Samples may be taken for histology and microbiology. These can include coral tissue, coral surface mucus and water, and sediment together with other flora or fauna associated with the diseased corals.

Historic and background information should also be provided, together with photographic documentation of the lesions and area. All samples should be collected using the sterile techniques suggested by the experts to whom they are to be sent. Permits are often required for collection and transportation of samples and these vary between locations.

PREVENTION AND CONTROL IN WETLANDS

Environment

Management of the environment is a challenge for these disease processes, but certain aspects of coral life history may lend themselves to disease control if they are incorporated into a management strategy.

Corals, unlike most other wildlife species of concern, are immobile. Once a diseased colony has been found, it will not move and can be counted and monitored (and potentially treated, if viable methods are developed). Corals also have the potential to re-grow over dead skeleton by re-sheeting and in this way they function more like plants.

There has been some success in controlling the spread of black band disease (BBD) during warming anomalies by aspirating the band using large syringes or pumps. Clay or underwater epoxy putty can then be placed directly over the band.

By **reducing the amount of anthropogenic stressors** on reefs, it is also possible to try to optimise conditions favourable for reef health and coral growth.

Ensure that divers collecting samples or visiting sites always visit healthy sites before those considered to be diseased.

All samples should be placed in **double containment** and divers should

disinfect SCUBA gear and equipment in 5% bleach solution (or other disinfectant) and then rinse in fresh water between sites.

There is evidence to suggest corals that survive a bleaching episode may later succumb to opportunistic infections, as their resistance is lowered by the stress of bleaching. In such cases, imposing a **quarantine** on a reef acutely impacted by either bleaching or disease may be worthwhile. The reef can be closed to human activity by prohibiting diving, snorkelling and fishing for a period of time. Managers should make every effort to **disseminate** to the public locally-relevant information on coral diseases and their potential impacts. Managers may also focus their attention on target groups who interact regularly with the reef: fishers, recreational divers, and diving tourism operators and their clients

In the longer term a number of actions can help to prevent disease and its spread between corals:

- Restrict translocation of corals to prevent movement of disease.
- Provide guidance for proper handling and containment regimes during coral disease experiments.
- Monitor proposed coral management and research activities, as well as rehabilitation or remediation activities, to minimise or avoid ethical and legal problems with the potential spread of disease.
- Promote the use of universal precaution measures when dealing with diseases in the field.
- Encourage ethical behaviour and improved sanitary practices among divers and other users of the marine environment.
- Communicate and report disease outbreaks and interventions.
- Harnessing enthusiasm among divers will provide managers with additional observers underwater, and the only efforts that are necessary are some initial training and regular communication.

Livestock & humans

None

Wildlife

Experiments have shown that black band disease can be eliminated and the rate of appearance of new infections can be reduced through re-introduction of herbivorous urchins *Diadema antillarum* into habitats where they were formally abundant.

IMPORTANCE

Effect on wildlife

Infectious disease in corals has increased in frequency and distribution since the early 1970's and since then there has been an exponential increase in numbers of reported diseases, host species and locations with disease observations. This rate of change has resulted in a global reduction in coral cover. In addition to the loss of coral tissue, disease can cause significant changes in reproduction rates, growth rates, community structure, species diversity and abundance of reef-associated organisms.

Effect on livestock & humans

None

Economic importance

The revenue earned from fishing, tourism, recreation, education and research associated with coral reefs is of major importance to many local and national economies and can be severely affected by diseases of the coral in these areas.

FURTHER INFORMATION

Useful publications and websites

- Coral Reef Targeted Research & Capacity Building for Management. **Coral disease: an emerging threat to the world's remaining reefs**. http://www.gefcoral.org/LinkClick.aspx?fileticket=h6EdRoHvUgY%3d&tabid=3260&language=en-US [Accessed March 2012].
- Stoskopf, M.K. (2006). **Coelenterates**. In: Invertebrate medicine (1st Ed.). Lewbart, G. A. (ed.), Blackwell Publishing, (2006), pp.327.
- Great Barrier Reef Marine Park Authority, Australia. **A reef manager's guide to coral bleaching**. http://data.iucn.org/dbtw-wpd/edocs/2006-043.pdf [Accessed March 2012].
- The Global Coral Disease Database (GCDD). http://coraldisease.org/ [Accessed March 2012].
- The Coral Reef Targeted Research Program - Coral Disease Working Group**.** www.gefcoral.org [Accessed March 2012].

Contacts

- The Coral Disease and Health Consortium (CDHC)**:** cdhc.coral@noaa.gov
- For a full list of experts, see www.gefcoral.org [Accessed March 2012].

Crayfish plague

Wetlands supporting groups of susceptible animals

Wildlife ✓
Aquaculture ✓
Humans ✗

Synonyms: Crayfish aphanomyciasis, Kraftpest, Krebspest, la peste

KEY FACTS

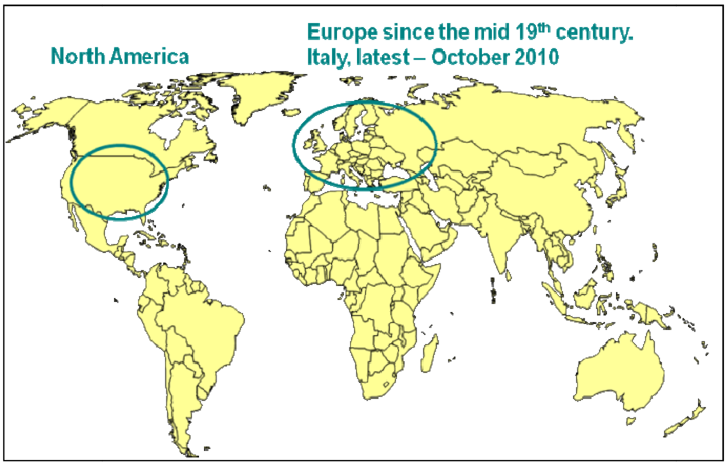
What is Crayfish Plague? Crayfish plague is a disease caused by an oomycete (water mould) that affects wild and farmed freshwater crayfish. The disease can cause large scale mortality.

Causal agent The oomycete *Aphanomyces astaci*. This is a close relative of *A. invadans* which is a species associated with epizootic ulcerative syndrome (EUS).

Species affected All species of freshwater crayfish are currently considered susceptible to crayfish plague. The outcome of infection varies depending on species:

- All stages of European crayfish species are considered highly susceptible.
- Laboratory challenges have shown that Australian crayfish species are also highly susceptible.
- North American crayfish do not usually present with clinical disease when infected with *A. astaci.*

Geographic distribution The native range of *A. astaci* infection is throughout North America. Crayfish plague spread to Europe in the 19th century and is now considered widespread throughout this continent.



Geographic distribution of crayfish plague.

Environment *A. astaci* is an obligate parasite of freshwater crayfish and does not survive well for long periods without a host. Crayfish plague is therefore found in the same freshwater, aquatic environments as its host.

TRANSMISSION AND SPREAD

Vector(s) Anthropogenic activity is often the most important vector of *A. astaci* as the disease is often spread by the translocation of animals (crayfish, fish, *etc.*) and *via* the movement of contaminated water and equipment (*i.e.* ropes, nets, traps, boots, fishing gear).

How is the disease transmitted to animals?

Transmission of the disease is primarily *via* the motile zoospores of *A. astaci* which have been shown to actively swim towards crayfish. Zoospores are also spread *via* flowing water, infected crayfish and less commonly by migratory and/or translocated fish.

How does the disease spread between groups of animals?

Introductions of North American crayfish (directly into the wild or into fish farms, from which escapes occurred) are believed to have initially spread crayfish plague to Europe.

The disease is spread to naïve crayfish populations by:
- the expansion of invasive, plague-carrying crayfish (*e.g.* signal crayfish *Pacifastacus leniusculus*)
- accidental releases of North American crayfish
- transmission from infected native crayfish
- viable zoospores (in water, on fish skin, or on contaminated equipment).

Other wildlife (*e.g.* otter, mink and heron) that can spread Infected crayfish to uncontaminated water bodies.

How is the disease transmitted to humans?

A. astaci does not have any human health implications.

IDENTIFICATION AND RESPONSE

Field signs

When the infection first reaches a naïve population of highly susceptible crayfish species, high levels of mortality are usually observed within a short space of time.

Initial field signs of crayfish plague include:
- presence of a number of crayfish during daytime (they are normally nocturnal)
- crayfish in open water with unsteady, uncoordinated movements
- crayfish falling over and unable to right themselves
- weakened rapid tail escape response
- numerous dead or weak crayfish in water bodies and water courses at the time of initial outbreak.

Note that there is no other disease, or pollution effect, that can cause total mortality of crayfish but leave all other animals in the same water unharmed. Clinical signs of crayfish plague are complicated. They depend on environmental conditions, number of zoospores and the density of susceptible crayfish in the area. Clinical signs can include:
- fungal growth on the soft parts of the shell
- brown or black spots on the carapace
- white necrotic musculature in the tail
- black lines on the soft shell underneath the tail
- blackening of most of the shell in chronically infected individuals
- death (within weeks in susceptible species).

Recommended action if suspected

Contact and seek assistance from appropriate animal health professionals. Crayfish plague is a notifiable disease and must be reported to local and national authorities and the OIE.

If crayfish plague is suspected take note of simple observations such as:
- abnormal behaviour of crayfish
- date and time of observed outbreaks

- species of crayfish affected and estimate of mortalities
- pattern of mortality (small number of crayfish dying every day, large number of crayfish dying at one time, *etc.*)
- any unusual events.

Guidance should be sought before collecting any samples.

Diagnosis

A confirmation of crayfish plague can be attained by molecular diagnostic tests (PCR, DNA sequencing). Isolation, confirmed by PCR and sequence analysis or bioassay, can be attempted. Note that isolation is only successful before or within 12 hours of the death of infected crayfish.

PREVENTION AND CONTROL IN WETLANDS

Environment

There is presently no practical way of eradicating crayfish plague or infected crayfish from a large or complex wetland system, although chemical eradication has proved an effective control mechanism in some smaller, closed water-bodies. Usually, the only effective way of preventing further spread and maintenance of crayfish plague is to control the spread of North American carrier crayfish. Emphasis should be placed on measures preventing future introductions of non-native or infected crayfish to unaffected water-bodies.

North American crayfish have been used in various European countries to replace the lost stocks of native crayfish. This is *not* recommended as restocking with North American crayfish can further the spread of *A. astaci*. Given the high reproductive rates and the tendency of several North American crayfish species to colonise new habitats, restocking with North American crayfish species would also largely prevent the re-establishment of native crayfish species.

Aquaculture

As above, actions should be directed at preventing the introduction of crayfish plague, as subsequent control can be very difficult.

- Movement of **water** or any **equipment** from affected to unaffected watersheds should be avoided or undertaken with disinfection precautions.
- Sodium hypochlorite and iodophores should be used to **disinfect equipment** and equipment should dried thoroughly (>24 hours).

If a **new crayfish farm** for a highly susceptible species is being planned, investigate whether North American crayfish species are:

- in the **vicinity** of the planned site; or
- present **upstream** (if North American crayfish are present, it is high likely that susceptible farmed crayfish will eventually become infected).

On an **established crayfish farm** (containing highly susceptible species), the following recommendations should be followed to avoid the introduction of *A. astaci* onto the site:

- Prevent movements of potentially infected live or dead crayfish.
- Prevent movements of potentially contaminated water, equipment or any other item that might carry *A. astaci* from an infected to an uninfected site.
- If fish transfers are to be undertaken, these must not come from streams or other waters that harbour potentially infected crayfish.
- Do not bring North American crayfish onto the site.

- Do not use fish obtained from unknown freshwater sources, sources where North American crayfish may be present, or from sources where a current outbreak of crayfish plague may be taking place.
- Do not use fish as bait or feed for crayfish, unless they have been subject to a temperature treatment that will kill *A. astaci*.
- Disinfect any equipment that is brought onto the site.
- Follow general biosecurity measures (*e.g.* controlled access to premises, disinfection of boots, investigation of mortalities if they occur).
- Conduct a risk analysis when making decisions to introduce live animals (crayfish, fish); introduce live animals only from sources known to be free of crayfish plague.

Wildlife

Contact between wildlife and aquaculture facilities should be minimised wherever possible.

Humans

Humans should make sure that they follow the guidelines described above to ensure that they do not move infectious agents or non-native crayfish to previously uninfected areas.

IMPORTANCE

Effect on wildlife

The spread of crayfish plague in Europe has resulted in the reduction of native European crayfish species. In the 125 years that crayfish plague has been recognised in Europe, no evidence of resistant populations of European crayfish has been found.

Although *A. astaci* does not directly affect biota other than the crayfish, the reduction of native crayfish species may indirectly affect the ecology of a wetland system.

Effect on aquaculture and fisheries

Large losses to fish farmers and fishermen through mortalities of crayfish.

Effect on humans

The agent causing crayfish plague has no direct human health implications.

Economic importance

Crayfish plague has caused significant financial damages to those who run crayfish farms and others who rely on catching in the natural water bodies for income.

FURTHER INFORMATION

Useful publications and websites

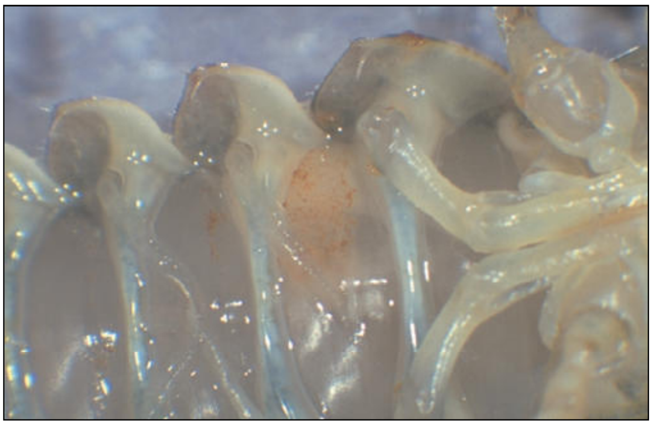
- World Organisation of Animal Health (OIE). **Chapter 2.2.01: Crayfish plague**. Manual of diagnostic tests for aquatic animals. http://www.oie.int/fileadmin/Home/eng/Health_standards/aahm/2010/2.2.01_CRAYFISH.pdf [Accessed March 2012].
- European Network On Invasive Alien Species (NOBANIS). **Invasive alien species fact sheet – *Aphanomyces astaci.*** http://www.nobanis.org/files/factsheets/aphanomyces_astaci.pdf [Accessed March 2012].
- Australian Government, Department of Agriculture, Fishery and Forestry. **Diseases of crustaceans; fungal diseases – crayfish plague**. http://www.scribd.com/doc/59272549/Crayfish-Plague. [Accessed March 2012].
- Food and Agriculture Organization (FAO) Fisheries technical paper. **Asia diagnostic guide to aquatic animal diseases**. http://www.fao.org/docrep/005/y1679e/y1679e00.HTM [Accessed March 2012].
- **Aquatic animal disease significant to Asia-Pacific; fungal diseases – crayfish plague**. http://library.enaca.org/Health/FieldGuide/html/cp001cra.htm [Accessed March 2012].

Contacts

- OIE reference laboratories and collaborating centres for diseases of amphibians, crustaceans, fish and molluscs: http://www.oie.int/fileadmin/Home/eng/Health_standards/aahm/2010/3_LIST_OF_LABS.pdf

Photos

Fresh microscopic mount of a piece of infected exoskeleton showing fungal spores (*D. Alderman, UK & FAO*).



Segment with brown markings shows signs of typical infection from fungus. (*D. Alderman, UK & FAO*).

Duck virus enteritis

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✖

Synonyms: Anatid herpesvirus, duck plague, DVE, endenpest, entenpest, peste du canard

KEY FACTS

What is duck virus enteritis?	Duck virus enteritis (DVE) is a contagious disease of ducks, geese and swans (order Anseriformes) caused by a herpesvirus. It can result in internal bleeding, diarrhoea and death but surviving birds can become carriers of the virus and intermittently excrete it for years. Disease appears to vary according to several factors, such as age, management practices and the presence of con-current disease agents.
Causal agent	Duck virus herpesvirus.
Species affected	Only ducks, geese, and swans are susceptible and species vary in their susceptibility to infection. During a disease outbreak, DVE may be suspected if there is no mortality in other shorebirds or waterbirds as it does not affect these species. DVE has not been reported in mammals, humans or other avian species.
Geographic distribution	DVE has been recorded in North America, countries in Asia, including India and China, and several countries in Europe.
Environment	Any environment supporting susceptible species.

TRANSMISSION AND SPREAD

Vector(s)	The disease is not vector-borne but may be spread through contact between birds or *via* exposure to contaminated objects/environments – see below.
How is the disease transmitted to animals?	Spread is by bird to bird contact or *via* exposure to a contaminated environment. Infected waterfowl shed and spread the virus in their droppings. The virus can persist in polluted and stagnant water and slow-moving pools, waterways and ponds. Swimming in and/or drinking infected water, or eating contaminated food, may infect susceptible birds as the virus may enter through breaks in the skin, the mouth, nose or cloaca. Some surviving birds may become carriers. These birds can shed the virus at other locations and therefore trigger future outbreaks. The disease occurs mostly in April, May, and June in Europe and North America, but can occur in any season. DVE in susceptible birds is linked to weather extremes and factors such as stress during breeding seasons. A DVE outbreak is perpetuated by environmental contamination, for example, from tissues and body fluids of decaying infected birds and also bird to bird contact.
How does the disease spread between groups of animals?	Carriers of DVE may shed the virus in faeces or on the surface of eggs, or fluids from decaying carcases of infected birds may contaminate the environment.
How is the disease transmitted to humans?	DVE is not transmitted to humans.

IDENTIFICATION AND RESPONSE

Field signs

DVE infected birds show variable signs which can include a combination of the following:

- loss of appetite
- listlessness, weakness and depression
- weight loss
- dehydration
- excessive water intake
- watery diarrhoea
- eye watering, and pasted eye-lids, associated with avoidance of bright light
- nasal discharge
- ruffled feathers and soiled vents
- a blue colouration to the bill
- prolapsed penis
- an ulcerative "cold sore" lesion under the tongue
- drop in egg production
- impaired movement, lack of muscle control and inability to fly
- bloody discharge from the bill and vent
- series of convulsions
- sudden death.

It is not possible to diagnose DVE on clinical signs alone but the disease should be suspected when acute deaths are seen in susceptible species of ducks, geese and swans. It is important to differentiate field signs from those of pesticide poisoning or other diseases such as avian cholera.

Recommended action if suspected

This is a potentially important poultry disease and if suspected a veterinary diagnostic laboratory should be contacted and submission of samples discussed. This may involve submitting freshly dead birds, recently euthanased birds or tissue samples.

Where possible whole birds should be submitted as opposed to tissues, but where this is not an option, remove the bird's liver, wrap in clean aluminium foil and place frozen in a plastic bag for shipping. Great care should be taken when packaging specimens to avoid contamination of packing materials and decomposition en route.

Any carcases should be incinerated and the area used to process the carcases and associated equipment disinfected.

In livestock settings, quarantine, depopulation, cleaning and disinfection of affected premises are crucial to prevent disease spread.

Diagnosis

Presumptive diagnosis of DVE is based on clinical signs, gross pathology and histopathology. Confirmation requires identification of the virus by viral isolation or PCR. The herpesvirus may be isolated from the liver, spleen and kidneys of infected birds. DVE carriers are in a state known as latency. It is during this period that the virus cannot be detected by standard methods of virus isolation.

PREVENTION AND CONTROL IN WETLANDS

Environment

DVE virus is resilient and can remain viable in the environment for many weeks under certain conditions. However, at pH 3 and below and at pH 11 and above, the virus is inactivated. Therefore, **decontamination** in domestic birds may be conducted *e.g.* by chlorination of contaminated water or by raising the pH or burning contaminated land. Burning of outbreak site materials and decontamination (including physical structures) should also be carried out. The **collection and disposal of carcases** by incineration should be meticulous and systematic. Personnel and equipment associated with carcase disposal should be decontaminated using chlorine bleach and phenol-based **disinfectants** before leaving the outbreak site to prevent mechanical spread to other waterfowl locations.

Livestock

The risks to commercial ducks and geese and captive wildfowl are greatest in free-range or open field systems especially if free-living wildfowl have access. To date no effective treatment for DVE exists.

In order to prevent rapid disease spread DVE requires rapid response and aggressive actions. The aim is to reduce exposure of the virus to populations of birds at risk, both as a source for potential infection and during outbreaks. Birds in a state of latency pose the greatest problem for disease prevention and control and being asymptomatic they are difficult to detect. Due to the fact that surviving birds are likely to become carriers, **eradication** of infected flocks (including eggs) may be required, and appropriate veterinary advice should be obtained.

A live **vaccine** is available to control DVE in birds over 2 weeks of age and ducks gain active immunity when vaccinated subcutaneously or intramuscularly.

Wildlife

The presence of domestic wildfowl in wetlands (especially highly susceptible muscovy ducks) greatly increases the risk of disease transmission to free-living wildfowl, hence this practice should be avoided if at all possible. Control in wildlife necessitates a system of rapid response to prevent spread by reducing exposure to the virus both in the environment and specifically at an outbreak site. Control actions include appropriate **disinfection** of an outbreak site, possible **drainage** of water bodies if appropriate and correct disposal of carcases.

In response to the potential devastating effects of DVE to continental wildfowl populations by direct losses and impaired reproductive capability, one of the wildlife agencies of the USA has developed a monitoring and control plan for DVE. Control measures in place for outbreak areas include: **disinfection** of contaminated soil, **chlorination** of affected waters, **quarantine** of epidemic areas, **removal and disposal of infected carcases** and **depopulation** of any captive flocks. Site specific responses are coordinated by a national DVE monitoring system in the USA, which includes state and federal agricultural and wildlife specialists. Although a live vaccine may be considered for control of captive flocks this is not an option in wild birds.

Any release or reintroduction programmes should not use birds or eggs from flocks with previous history of DVE unless certified DVE-free. Birds selected for release should be confined 2 weeks prior to liberating and any that die during that period should be submitted to a veterinary disease diagnostic laboratory. If DVE is confirmed then no remaining birds should be released.

Humans Not required.

IMPORTANCE

Effect on wildlife

In wild waterfowl populations, DVE may cause high mortality, together with secondary reproductive impairment. DVE effects may be endemic in wild species, although little information exists regarding the responses of wild waterfowl to different DVE strains.

Effect on livestock

In susceptible domestic waterfowl flocks this highly contagious disease can result in high mortality and reduced egg production. Flocks under the stress of egg production may suffer higher mortality compared with immature breeders. Although most commercial duck flock outbreaks have been in eastern Asia it has been recorded that migratory waterfowl are the source of DVE for captive waterfowl in regions such as North America and parts of Europe.

Effect on humans

DVE is not infectious to humans.

Economic importance

Significant economic losses may result from fatal outbreaks in commercial flocks and a drop in egg production.

FURTHER INFORMATION

Useful publications and websites

- Friend, M. & Franson, J.C. (2001). **Duck plague**. In: Field manual of wildlife diseases: general field procedures and diseases of birds. E. A. Ciganovich (ed.). pp. 141-152. U.S. Department of the Interior and U.S. Geological Survey, Washington, DC. www.nwhc.usgs.gov/publications/field_manual/chapter_16.pdf. [Accessed March 2012].
- World Organisation for Animal Health (OIE). **Chapter 2.03.07: DVE**. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.03.07_DVE.pdf. [Accessed March 2012].
- U.S Geological Survey (USGS) National Wildlife Health Center. **Duck plague**. http://www.nwhc.usgs.gov/disease_information/other_diseases/duck_plague.jsp [Accessed March 2012].
- Wildpro. **Duck plague**. http://wildpro.twycrosszoo.org/S/00dis/Viral/Duck_Plague.htm [Accessed March 2012].

Epizootic ulcerative syndrome (EUS)

Freshwater and estuarine wetlands supporting susceptible animals

Wildlife ✓
Aquaculture✓
Human ✗

N

Synonyms: Epizootic granulomatous aphanomycosis (EGA), mycotic granulomatosis (MG), ulcerative aphanomycosis (UA), ulcerative mycosis (UM), red spot disease (RSD)

KEY FACTS

What is EUS?

Epizootic ulcerative syndrome (EUS) is an infection caused by an oomycete (or water mould) – a fungus-like microorganism associated with seasonal epidemic conditions affecting wild and farmed freshwater and estuarine fish.

Causal agent

The lesions in EUS-affected tissues are caused by the oomycete *Aphanomyces invadans* or *A. piscicida.* Parasites and rhabdoviruses have also been associated with specific outbreaks and secondary bacteria invariably infect EUS lesions.

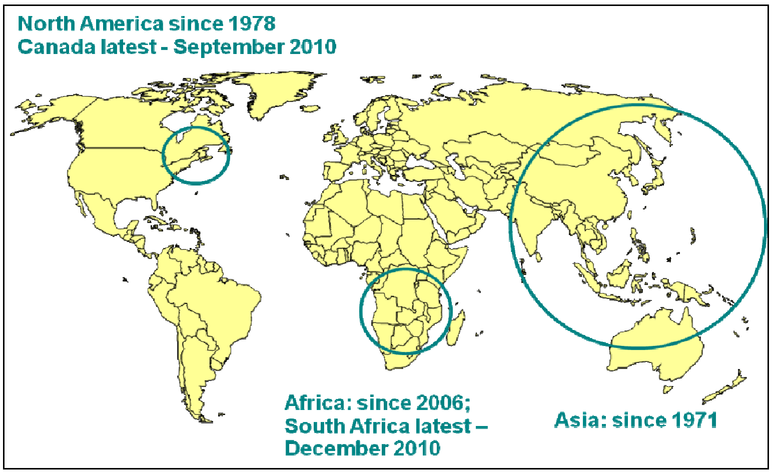
Species affected

Farmed and wild fish are affected worldwide, with infection confirmed in almost 80 finfish species, *e.g*. barbs, breams, catfish, gouramy, eel, mullet, pike, tigerfish, tilapias, seabass and snakehead. The range of susceptible species is very broad, thus many more species of fish are likely to be susceptible.

Some fish species, such as common carp *Cyprinus carpio* and Nile tilapia *Oreochromis niloticus*, have been reported not to develop clinical disease during outbreaks in other species. However, experimental studies demonstrate susceptibility so their potential role in spreading the disease is currently unclear.

Geographic distribution

EUS is a notifiable OIE-listed disease and now has a worldwide distribution. It was first reported in Japan in 1971, followed by subsequent confirmed reports from Australia (1972), the USA (1978), south and south east Asia (1986), southern Africa (2007) and Canada (2010). EUS now affects 25 countries in four continents: Africa, Asia, Oceania and North America.



Geographic distribution of EUS.

Environment

Any freshwater or estuarine habitats supporting susceptible species. The causative oomycete grows best at 20–30°C. Water salinity over two parts per thousand (ppt) can stop the spread of the agent.

TRANSMISSION AND SPREAD

Vector(s)

No data are available. However, it is possible that fish-eating birds can spread EUS.

How is the disease transmitted to animals?

EUS is transmitted horizontally from one fish to another. It is believed that only the zoospores are capable of attaching to damaged skin of fish and germinating into hyphae. If the zoospores cannot find susceptible species or encounter unfavourable conditions, they can encyst in the water or pond environment waiting for conditions that favour the activation of the spores.

Conditions which favour EUS occurrence include periods of lower temperatures (low for tropical climes, *e.g.* 18–22°C) and after heavy rainfall. Sporulation of *A. invadans* occurs under these conditions, whilst low temperatures have been shown to delay the inflammatory response of fish to oomycete infection.

A diverse group of biotic (*e.g.* parasites, bacteria, viruses) and abiotic (*e.g*. acid water) agents/factors are likely involved in initiating skin lesions in freshwater and estuarine fish species which are subsequently colonised by *A. invadans/piscicida*. A specific determinant is unlikely to be associated with EUS outbreaks; most probably, environmental determinants vary from outbreak to outbreak depending on the agent initiating the non-specific lesions, the aquatic environment at the site and the population at risk. EUS outbreaks in wild estuarine populations (*e.g.* Australia and the Philippines) have been reported to be associated with acidified run-off water from acid sulphate soil areas.

For EUS to occur, a combination of factors must ultimately lead to exposure of the skin, attachment to it by *A. invadans/piscicida*, and subsequent invasion by the fungus.

Successful invasion and establishment of EUS in fish requires tissue (epithelial) damage, a susceptible fish species and environmental conditions which favour sporulation of the oomycete.

How does the disease spread between groups of animals?

The disease occurs only among finfish. The spread from wild to cultured populations or *vice versa* can occur *via* several routes. Freshwater or estuarine fish migrations are thought to provide a potential pathway for pathogen movement. In addition, movements of fish (cross border and domestic) for aquaculture and the ornamental fish trade are proven pathways. In some countries outbreaks occur in wild fish first and then spread to fish ponds. Flooding also causes the spread of EUS (*e.g*. as in Bangladesh and Pakistan). Once an outbreak occurs in rivers/canals, the disease can spread downstream as well as upstream where susceptible fish species exist.

How is the disease transmitted to humans?

The agent causing EUS does not pose any human health implications. However, it is recommended not to eat EUS-infected fish unless it is properly and thoroughly cooked.

IDENTIFICATION AND RESPONSE

Field signs

EUS outbreaks have been associated with mass mortality of various species of freshwater or estuarine fish in the wild (*e.g.* in rice-fields, estuaries, lakes and rivers) and in farms often during periods of low temperatures (low for tropical climes, *e.g.* 18–22°C), but outbreaks have been observed across a broad temperature range (10-15 to 33°C).

The following abnormal behaviour may be seen: fish swimming near the surface, sinking to the bottom, loss of balance, flashing, cork-screwing or air gulping (for non air-breathers). Other behavioural signs include loss of appetite and darkening of skin. Infected fish may float near the surface of the water yet become hyperactive with a jerky pattern of movement.

Small to large red spots and open dermal ulcerative lesions may be seen.

African catfish *Clarias gariepinus* infected with EUS (*FAO*).

Straightfin barb *Barbbus Paludinosus* (*FAO*).

Recommended action if suspected

EUS is a notifiable disease and suspected cases must be reported immediately to local (nearest fisheries or veterinary authority) and national authorities and the OIE. Guidance concerning collection of samples should be sought.

Take note of simple observations such as:

- abnormal fish behaviour
- date and time of observed outbreaks
- total estimate of mortalities
- species of fish affected and estimate of mortalities per species
- pattern of mortality (small number of fish dying every day, large number of fish dying at one time, *etc.*)
- any unusual events.

Diagnosis

Presumptive diagnosis of EUS can be based on clinical signs and, in the laboratory, the observation of hyphae in squashed preparations of the muscle underlying gross lesions. EUS can be confirmed (1) when histological sections show the presence of typical lesions in affected tissues or organs; (2) by PCR identification; or (3) by isolation of *A. invadans/piscicida* from infected fish and confirmed by either bioassay, PCR or DNA sequence analysis.
►**Photos at the end of this factsheet.**

PREVENTION AND CONTROL IN WETLANDS

Environment

Control of EUS in natural water bodies is not possible.

Aquaculture

Actions should be directed firstly at prevention of the disease as subsequent control can be very difficult. No protective vaccine or effective drug/chemical treatment are available. The most important biosecurity measure to prevent the introduction onto farms is sourcing fish from safe, uninfected sources only.

A number of simple biosecurity measures can minimise or prevent the spread of EUS. These include:

- All possible carriers or vectors such as freshly dead fish, birds or terrestrial animals as well as contaminated fishing gear and fish transport containers should be prevented from entering water bodies or fish ponds.
- In outbreaks occurring in small, closed water bodies, liming of water and improvement of water quality, together with removal of infected fish.
- Increasing salinity in holding waters may also prevent outbreaks of EUS in aquaculture ponds.
- During dry and cold seasons (in tropical climes), close observation of wild fish should be made to determine the presence of EUS-diseased fish in neighbouring tanks or canals, in which case, exchange of water should be avoided.
- EUS-infected fish should not be thrown back to the open waters and should be disposed of properly by burying them in the ground or by incineration.
- Additional practical aquaculture biosecurity measures include:
  - Good farm hygiene (*e.g.* handwashing between tanks, separation of nets/tanks/stocks, regular and correct disinfection procedures, *etc.*)
  - Good husbandry practices
  - Good water quality management
  - Proper handling of fish to avoid stress
  - Regular monitoring of health status
  - Good record keeping (gross and environmental observations and stocking records including movement records of fish in and out of aquaculture facilities, *etc.*).
- Early reporting or notification to concerned authorities of a disease outbreak or suspicion of any abnormal appearance, behaviour or other observations in fish stocks.

Wildlife

The risk of EUS spread can be reduced by ensuring that water or wild fish do not come into contact with fish culture ponds. Contact between fish-eating birds and aquaculture facilities should be minimised to reduce the risk of disease spread from an infected to an uninfected area.

Humans

Do not eat EUS-infected fish unless it is properly and thoroughly cooked.

IMPORTANCE

Effects on wildlife

EUS is one of the most serious aquatic diseases affecting finfish. Indirect long-term effects may include threats to the environment and aquatic biodiversity through, for example, declining fish biomass and irreversible ecological disruption.

Effects on aquaculture and fisheries

High losses to fish farmers and fishermen through mortalities, market rejection and public health concerns due to the presence of ugly lesions and reduced productivity of all susceptible fish species.

Effects on humans

The agent causing EUS does not have direct human health implications although it is recommended not to eat EUS-infected fish unless it is properly and thoroughly cooked.

►**Effects on aquaculture and fisheries and Economic importance**

Economic importance

EUS has the potential to financially ruin those who run fish farms and others who rely on fishing for income. In addition, and perhaps more importantly, EUS outbreaks threaten food security for subsistence fishers and fish farmers and subsequently people's physical health, as fish are an important source of animal protein for people in the affected countries.

FURTHER INFORMATION

Useful publications and websites

- World Organisation for Animal Health (OIE). **Chapter 2.3.02: Epizootic ulcerative syndrome**. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/aahm/2010/2.3.02_EUS.pdf [Accessed March 2012].
- Food and Agriculture Organization (FAO). **What you need to know about epizootic ulcerative syndrome (EUS) - An extension brochure**. ftp.fao.org/FI/DOCUMENT/aquaculture/aq2008_09/root/i0777e.pdf [Accessed March 2012].
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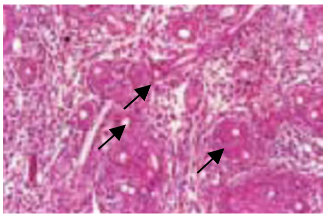
- Food and Agriculture Organization (FAO). **Report of the International Emergency Disease Investigation Task Force on a serious finfish disease in southern Africa**. 18-26th May 2007. www.fao.org/docrep/012/i0778e/i0778e00.htm [Accessed March 2012].
- Oidtmann, B. (2012). **Review of biological factors relevant to import risk assessments for epizootic ulcerative syndrome (*Aphanomyces invadans*)**. *Transboundary and Emerging Diseases*, 59 (1): 26–39.

Contacts

✉ **OIE reference laboratories and collaborating centres for diseases of amphibians, crustaceans, fish and molluscs**. www.oie.int/fileadmin/Home/eng/Health_standards/aahm/2010/3_LIST_OF_LABS.pdf

Additional photos



Typical severe mycotic granulomas (black arrows) from muscle section of EUS infected fish (*FAO*).

Typical *Aphanomyces* sporangium (Japanese isolate, *FAO*).

Escherichia coli poisoning

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: *E. coli*, colibacilliosis, colisepticaemia

KEY FACTS

What is *Escherichia coli* poisoning?

Escherichia coli is a bacterium that is commonly found living in human and animal intestines. Most of the hundreds of strains are harmless and some are even beneficial to humans and animals but others can cause illness. One such strain is *E. coli* O157, which is pathogenic in a number of species, produces a powerful toxin often referred to as Shiga toxin or verotoxin, and can cause severe illness and potentially death.

Once excreted from human and animal intestinal tracts, the bacteria may not survive, but some do find their way into lakes and streams, where they can persist for several weeks in water, sediment or sand. Frequent sources of *E. coli* include direct release of untreated sewage, leakage from sewage pipes, run-off from human developments, domestic animal faeces, and run-off from land or premises where animals are kept or grazed. Dog and cat faeces may be carried along by storm sewers, deposited directly into streams and pathogens may be released into groundwater by insufficiently maintained septic systems. Wild mammals and birds may directly release faeces into waterways.

The *E. coli* strain O157 which is carried mainly by ruminants can cause severe disease in vulnerable humans (particularly the elderly and children under five years old). It is likely that widespread use of antibiotics in livestock has helped increased prevalence of *E. coli* O157 in many parts of the world with some cattle, in particular, becoming 'super-shedders' of this zoonotic bacterium. The excretion of antibiotics into the environment directly from farms or even through sewage farms, contributes to genetically determined resistance in these and other bacteria in the environment. Infection occurs directly *via* contact with infected farm (or to a lesser extent wild) animals and their environments or from consumption of contaminated meat or unpasteurised milk.

A recent concern is the emergence of a new type of antibiotic resistance (called extended-spectrum beta-lactamase or ESBL) *E. coli*. Scientists are now finding strong evidence that a significant amount of antibiotic resistance in human *E. coli* infections comes from farm animals (particularly poultry but also pigs and cattle), contributing to increasing resistance in urinary-tract infections and blood poisoning in people.

Causal agents

- enterotoxigenic *E. coli* (ETEC)
- enteroinvasive *E. coli* (EIEC)
- enteropathogenic *E. coli* (EPEC)
- enterohaemorrhagic *E. coli* (EHEC)

Species affected

Mammals (including humans, pigs, sheep, goats, cattle, dogs, cats, horses and wild mammals) and to a lesser extent birds.

Geographic distribution

Occurs worldwide.

Environment

Wetlands inhabited by susceptible species, particularly domestic ruminants.

TRANSMISSION AND SPREAD

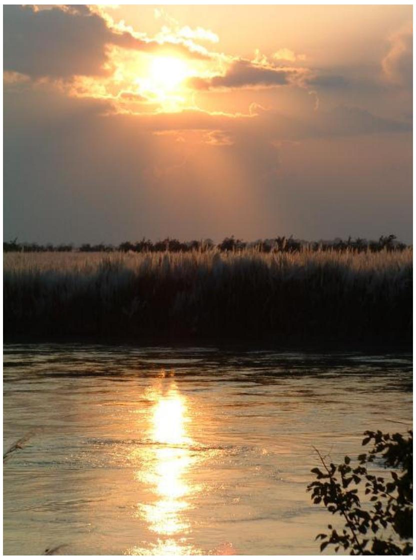
Vector(s)	*E. coli* is not vector-borne although some mechanical transfer from contaminated areas is possible.
How is the disease transmitted to animals?	Animals (livestock in particular) become infected with *E. coli* by exposure to items including food, water and inanimate objects (fomites) contaminated with faeces from which bacteria can be ingested. Susceptible animals include those which are immunocompromised, stressed, young, old, breeding or with associated environmental pressures.
How does the disease spread between groups of animals?	Animals can serve as carriers of the bacteria *i.e.* without the bacteria causing illness. The bacteria can be found in sheep, pigs, deer, cattle, dogs, poultry and other animals, although cattle are the main carriers. Infected animals, in particular young animals, shed the bacteria in their faeces, thus leading to exposure of other animals.
How is the disease transmitted to humans?	Most people are infected with *E. coli* from contaminated food (*e.g.* undercooked ground beef) or unpasteurised milk or contact with animal faeces from the environment. Animals do not have to be ill to transmit *E. coli,* including *E. coli* O157, to humans.

IDENTIFICATION AND RESPONSE

Field signs	Signs of *E. coli* infection in animals may include watery or bloody diarrhoea, fever and abdominal cramps, together with nausea and vomiting in animals such as cats and dogs. Resulting illness may be mild or severe. In humans, incubation period ranges from 1-8 days but the duration of the illness is usually approximately 3–5 days. However, the bacteria can continue to be passed in faeces for up to three weeks post infection. Symptoms vary from mild to severe and include diarrhoea, vomiting, stomach-ache and fever. In adults, for most strains, the infection clears on its own in about a week.
Recommended action if suspected	Alert the relevant authorities of any suspected cases.
Diagnosis	Many laboratory-based methods for detection of *E. coli* bacteria involve collection of environmental or faecal samples and isolating the bacteria or using polymerase chain reaction (PCR) methodologies to test water for bacteria. The latter method is rapid and can differentiate between *E. coli* of human and non-human sources.

PREVENTION AND CONTROL IN WETLANDS

Environment	Following laboratory confirmation, a **response system** may be activated if bacteria levels have risen to unacceptable limits based on bacterial water quality standards. Accepting that domestic ruminants pose the greatest risk of transmission of pathogenic strains of *E. coli*, **treatment wetland systems** can help treat water running off from agricultural premises and animal holdings.



Reedbed in Koshi Tapu Wildlife Reserve, Nepal. Natural or constructed treatment wetlands can significantly reduce bacterial contamination from livestock entering waterways (*WWT*).

Livestock

E. coli exposure can be limited in animals by **preventing faecal contamination** of feed and water, thus reducing the opportunity for ingestion of the bacteria.

Wildlife

Similarly, *E. coli* exposure can be limited in wildlife by **preventing faecal contamination** of wetlands, particularly by domestic ruminants, thus reducing exposure to the bacteria. If appropriate, wildlife can be kept away from possible sources of contamination *e.g.* by constructing physical barriers. Wetland treatment systems can also be used to reduce the risk of infection [►**Environment**]. Separating livestock from wildlife reduces risk to the latter.

Humans

Reducing exposure to *E. coli* by **preventing/reducing faecal contamination** of the environment including food and water plus **hygiene control measures** are key to reducing risk to humans. Hands should be frequently washed with soap after handling animals, or working in their environment, and disposable gloves should be worn if in contact with sick animals.

Medical attention should be sought for severe cases.

IMPORTANCE

Effect on wildlife

Wildlife in human agricultural landscapes, in particular species closely associated with livestock pastures *e.g.* wild rabbits, scavenging and feral species, have been shown to be infected, albeit at low levels, with *E. coli* O157 and in certain circumstances can act as a reservoir for *E. coli* O157. Wildlife populations may be in danger of fatalities or morbidity particularly if there are con-current infections or other stressors present. This is a problem of developed intensive agricultural systems and there is no evidence of widespread infection from extensive rangeland systems and natural environments.

Effect on livestock

Whilst domestic mammals generally only serve as carriers (or reservoirs) of the bacteria, some strains of *E. coli* do cause illness. For example, *E. coli* can cause illness in domestic animals either as a primary pathogen (diarrhoea in young pigs) or in association with other disease such as coronaviruses in cattle. *E. coli* mastitis in dairy cows can be very severe and potentially fatal, and adult pigs and cattle can be affected by urinary tract and other infections caused by pathogenic *E. coli*. Colibacillosis in pigeons and poultry is usually secondary to stress or con-current viral infection. *E. coli* in poultry can cause mortality, drops in weight gain and hatchability.

Effect on humans

Disease can be fatal; *E. coli* O157 can cause severe illness including deaths particularly in the young and old. Attacks of *E. coli* gastroenteritis may result in some infants developing a disaccharidase and lactose intolerance, which may become clinically manifested as chronic diarrhoea. There is now compelling evidence that animals reared for food are a reservoir for both antibiotic-resistant pathogenic and commensal *E. coli*, colonising or infecting humans, whilst also serving as a reservoir for resistance genes which can transfer to *E. coli* and can cause infections in humans.

Economic importance

Livestock infections can affect productivity *e.g.* in poultry [▶ **Livestock**].

FURTHER INFORMATION

Useful publications and websites

- Department for Environment, Food and Rural Affairs (DEFRA). **Summary profile for verocytotoxin producing *E. coli* (VTEC 0157).** archive.defra.gov.uk/foodfarm/farmanimal/diseases/vetsurveillance/profiles/documents/sp-vtec.pdf. [Accessed March 2012].
- Fairbrother, J. M. & Nadeau, É. (2006). ***Escherichia coli*: on-farm contamination of animals**. *Rev. sci. tech. Off. int. Epiz.*, 25, 2, 555-569. www.ecl-lab.ca/fr/news/documents/OIERev08-fairbrother555-569.pdf. [Accessed March 2012].
- World Organisation for Animal Health (OIE). **Chapter 2.09.11: Verocytotoxigenic *Escherichia coli***. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.09.11_VERO_E_COLI.pdf [Accessed March 2012].
- World Health Organization (WHO). **Enterohaemorrhagic *Escherichia coli* (EHEC)**. http://www.who.int/mediacentre/factsheets/fs125/en/ [Accessed March 2012].
- Wildpro. **Colibacilliosis.** http://wildpro.twycrosszoo.org/S/00dis/Bacterial/Colibacillosis.htm [Accessed March 2012].
- Wetlands International. **Wetlands & water, sanitation and hygiene (WASH) - understanding the linkages (2010)**. http://www.wetlands.org/WatchRead/Currentpublications/tabid/56/mod/1570/articleType/downloadinfo/articleId/2467/Default.aspx [Accessed March 2012].
- Nunan C. & Young R. (2012). ***E. coli* superbugs on farms and food**. The Soil Association, Bristol, UK pp.88. http://www.soilassociation.org/LinkClick.aspx?fileticket=yCT9su5iViQ=&tabid=313

Harmful algal blooms

Both saltwater and freshwater wetlands

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Cyanobacterial blooms, exceptional algal blooms, HABs, micro-algal blooms, phycotoxins, phytoplankton blooms, red tide toxicosis, red tides, toxic algae

KEY FACTS

What are harmful algal blooms (HABs)?

Blooms of toxin-producing algae which may kill fish, shellfish, other wildlife and livestock and cause illness and sometimes death in humans. High biomass harmful algal blooms (HABs) cause harmful effects when they occur in high concentrations, and cause discolouration of the water *e.g.* 'red tides'. Low biomass HABs cause harm when they occur in low concentrations and do not necessarily cause discolouration of the water, which can appear clear.

Causal agent

Toxin-producing species of algae, including: *Alexandrium fundyense, Dinophysis spp, Gambierdiscus toxicus, Gymnodinium catenatum, Karenia brevis, Karenia brevisulcatum, Karlodinium veneficum, Lyngbya, Pfiesteria piscicda, Pfiesteria, Prorocentrum lima, Protoperidinium crassipes, Pseudo-nitzchia and Pyrodinium bahamense* var. *compressum*

Species affected

Many aquatic species, marine and terrestrial mammals, birds and humans.

Geographic distribution

Occurs worldwide.

Environment

Occur in both saltwater and freshwater environments, particularly where there are high nutrient levels (in particular high levels of nitrogen and phosphorus) but can also occur frequently in low nutrient environments.

TRANSMISSION AND SPREAD

How are algal blooms caused?

Algal blooms are a natural phenomenon, however, they occur more commonly when offshore algal populations are transported to inshore regions or following agricultural run-off and other pollution events of freshwater and marine wetlands. These events can cause increased nutrient loading of phosphorous and nitrogen which then encourages the growth of algae, including toxin-producing algae in the case of HABs.

How do algal blooms cause harm?

- Production of toxins. Toxins may kill fish or shellfish directly, or may cause human illnesses following consumption of contaminated seafood. Livestock may drink contaminated water or lick themselves after bodily exposure and become ill.
- Mechanical damage to aquatic life such as blocking gills of fish.
- Affecting water quality by causing oxygen depletion from respiration and bacterial degradation, and blocking of sunlight.

IDENTIFICATION AND RESPONSE

Field signs

Sudden mortality of a broad range of taxa *e.g.* birds, amphibians, fish and/or marine mammals. This may appear in conjunction with occurrence of a marine reddish/orange tide or freshwater bloom (which initially appear green and may later turn blue sometimes forming a scum/foam in the water). Signs such as irritation of the skin, vomiting, paralysis, lethargy and loss of muscle co-ordination may be observed in birds. Birds and domestic mammals that ingest toxic blooms of *Microcystis* may develop necrotic lesions and haemorrhages in the liver. Not all toxic algal blooms are visibly noticeable and so a sample of organisms from the bloom may be useful or necessary for diagnosis.

Recommended action if suspected

Contact and seek assistance from animal and human health professionals immediately if there is any illness in birds, fish, marine mammals and/or people. Report suspected cases to local or national authorities.

Diagnosis

Confirmative diagnosis is difficult and relies on circumstantial evidence and supportive clinical and pathologic findings. There are also currently no established toxic thresholds for wildlife species and even when these exist it may be difficult to assess their significance.

Collection of algal samples may be necessary for diagnosis. Collect samples during the die-off event as soon as possible after carcases are found. Contact a diagnostic laboratory for advice on appropriate sample collection and transport.

PREVENTION AND CONTROL IN WETLANDS

Overall

Reduce the release of nutrients into waterways

- Use vegetated buffer zones. Plants such as reeds and willow, and constructed treatment wetland systems can remove sediments and pollutants especially in places which release high volumes of nutrients, such as animal and human sewage outlets.
- Reduce the use of fertilisers.
- Improve animal waste control.
- Improve sewage treatment.

Note that control methods remain largely untested on major blooms.

Monitoring and surveillance

Careful monitoring and early detection of potentially toxic algal blooms could allow time to initiate actions to prevent or reduce harmful effects *e.g.* bird mortality.

- Monitor for changes in nutrient load of water discharges, particularly sewage discharges (including septic tanks and cesspits) and agriculture.
- Patrol to observe and map discoloured water or dead fish for early detection of potentially toxic algal blooms.

Livestock

Keep livestock from drinking/bathing in lakes with blooms.

Wildlife

If possible, try to reduce access to contaminated areas *e.g.* using streamers and flags to dissuade birds from using an affected wetland and consider moving endangered species to safe areas with no HABs.

Humans

- Do not fish in an algal bloom/discoloured water and never eat fish which are dead when caught.
- Be aware of intoxication symptoms when eating shellfish and fish. If symptoms are experienced, keep sample of the food for toxicity tests.
- When swimming, look for warnings of algal blooms and avoid swimming if you cannot see your feet when the water level is at your knees.
- Wear rubber/latex gloves when handling carcases associated with HABs.

IMPORTANCE

Effect on wildlife

May cause mass mortality of aquatic species (including turtles and marine mammals such as manatees and dolphins), especially fish and shellfish, and accounts for more than half of unusual marine mortality events. Ingestion of toxin may not cause mortality but have other less obvious physiological effects such as affecting immune, neurological and reproductive capability.

Effect on livestock

Mostly not harmful unless ingested through eating contaminated seafood/fish, drinking contaminated water or licking their coats following exposure to the skin.

Effect on humans

Mostly not harmful unless ingested through eating contaminated seafood/fish or drinking contaminated water. Some organisms irritate the skin and others release toxic compounds into the water and, if aerosolised by wave action, these compounds may cause problems when inhaled.

Economic importance

May have significant economic impacts on freshwater and marine aquaculture industries, fisheries and coastal tourism.

FURTHER INFORMATION

Useful publications and websites

- Asia Pacific Economic Program, Singapore, and Intergovernmental Oceanographic Commission. Technical Series No. 59, (2001). **Monitoring and management strategies for harmful algal blooms in coastal waters**. www.whoi.edu/fileserver.do?id=24193&pt=10&p=19155. [Accessed March 2012].
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- NOAA Coastal Ocean Program Decision Analysis Series No. 10. **Harmful algal blooms in coastal waters: options for prevention, control, and mitigation**. (1997). www.cop.noaa.gov/pubs/das/das10.pdf [Accessed March 2012].
- Intergovernmental Oceanographic Commission (IOC) of UNESCO: Harmful Algal Bloom Programme ioc-unesco.org/hab/ [Accessed March 2012]
- Wildpro. **Blue-green algae toxicity in waterfowl**. http://wildpro.twycrosszoo.org/S/00dis/toxic/Biotoxin/Blue-Green_Algae_Toxicity.htm [Accessed March 2012].

Contacts

- **IOC Science and Communication Centre on Harmful Algae**. University of Copenhagen, Øster Farimagsgade 2D, 1353 Copenhagen K, Denmark. hab.ioc@unesco.org ☎ Tel: +45 33134446, Fax: +45 33134447.
- **IOC-IEO Science and Communication Centre on Harmful Algae**. Instituto Español de Oceanografía, Centro Oceanografico de Vigo, Cabo Estay-Canido, 36390 Vigo, Spain. vigohab@vi.ieo.es ☎ Tel: +34 986492111 ; Fax: +34 986492003.
- **Regional HAB networks:**
  - IOC FANSA: an IOC regional working group and network on harmful algal blooms in South America. www.ioc-unesco.org/hab/index.php?option=com_oe&task=viewDoclistRecord&doclistID=60 [Accessed March 2012].
  - IOC ANCA: the regional working group and network on harmful algae in the Caribbean. www.ioc-unesco.org/hab/index.php?option=com_oe&task=viewDoclistRecord&doclistID=61 [Accessed March 2012].

- IOC HANA – An IOC working group and network on harmful algae in North Africa (HANA). This list contains HANA working documents and HAB profiles of North African countries participating in HANA. www.ioc-unesco.org/hab/index.php?option=com_oe&task=viewDoclistRecord&doclistID=63 [Accessed March 2012].
- EUROHAB - The European Commission cluster of HAB research projects: cordis.europa.eu/eesd/ka3/cluster5.htm [Accessed March 2012].
- HARRNESS – US national plan for algal toxins and harmful algal blooms. www.esa.org/HARRNESS [Accessed March 2012].
- ECOHAB – US National Research Agenda on the Ecology and Oceanography of Harmful Algal Blooms. http://www.whoi.edu/science/B/redtide/nationplan/ECOHAB/ECOHABhtml.html
- IOC Western Pacific Network – WESTPAC/HAB (IOC Sub-Commission for the Western Pacific / Harmful Algal Blooms). www.ioc-unesco.org/hab/index.php?option=com_content&task=view&id=20&Itemid=0
- IOC-ICES Northern Atlantic network - WGHABD (ICES-IOC Working Group on Harmful Algal Bloom Dynamics). www.ioc-unesco.org/hab/index.php?option=com_content&task=view&id=11&Itemid=0
- CEOHAB - Chinese Ecology and Oceanography of Harmful Algal Blooms Programme. www.china-hab.cn/english

Samples whereby species are difficult to identify or species that requires special techniques can be sent to: www.ioc-unesco.org/hab/index.php?option=com_content&task=view&id=15&Itemid=0

Lead poisoning

Any wetland where lead is deposited

Wildlife ✓
Livestock ✓
Human ✓

Synonym(s): Pb poisoning

KEY FACTS

What is lead poisoning?

Lead poisoning arises through the absorption of hazardous levels of lead in body tissues. Lead is a highly toxic poison which can cause morbidity and mortality in humans, livestock and wildlife. Waterfowl, birds of prey and scavenging birds are at greater risk of exposure to lead than other bird species and mammals due to feeding habits that involve ingesting lead gunshot as grit or consuming prey animals that have been shot with lead ammunition. Lead poisoning in waterbirds is a very serious and large-scale environmental problem. Birds can die from lead poisoning throughout the year but mortality is more likely after waterfowl hunting seasons. Lead exposure may also cause a variety of health effects in humans, particularly for children, foetuses and pregnant women.

Causal agent

Lead.

Species affected

Many species of birds, particularly waterbirds, birds of prey, scavenging birds, and mammals.

Geographic distribution

Occurs worldwide, *i.e.* wherever lead is deposited in the environment.

Environment

Any environment where lead is deposited and accessible.

EXPOSURE

How is the environment contaminated by lead?

Wetlands are most commonly contaminated by spent lead ammunition and abandoned lead fishing weights which build up in the sediments of lakes and marshes. Any species using an area where shooting with lead ammunition occurs or has occurred previously is at some risk of exposure and, potentially, poisoning. Lead-based paint, mine wastes, lead contaminated industrial effluents and other objects provide additional sources of contamination.

How are animals exposed to lead?

Waterfowl usually become poisoned after ingesting spent lead shot, mistaking them for food items or grit, which is usually picked up to facilitate digestion. Predators or scavengers may become poisoned after consuming animals that have been shot with lead ammunition. Lead from ammunition and fishing weights may slowly dissolve and enter groundwater, making it potentially harmful for plants, animals and perhaps humans if it enters water bodies or is taken up in plants. Lead poisoning in livestock often occurs after swallowing point sources of lead such as lead from inside vehicle/machine batteries or lead paint, but also through consuming contaminated water and food supplies. Cattle are at most risk due to their inquisitive natures and they often ‘taste-test’ objects.

How are humans exposed to lead?

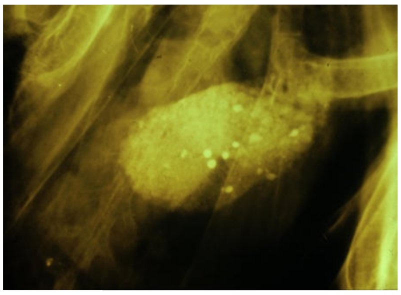
Exposure to lead may occur through ingestion of contaminated food, such as lead shot game, and through inhalation and absorption through the skin from sources such as gasoline, industrial activities and water pipes made from lead. Toxic effects may or may not be recognised as such.

IDENTIFICATION AND RESPONSE

Field signs

Sick and dead birds are usually seen in low numbers, although many are likely to go undetected. Large scale die-offs only occasionally occur. Signs include weakness, lethargy, reluctance to fly or inability to sustain flight, weight loss causing emaciation (the breast-bone becomes prominent), green-stained faeces and vent and fluid discharge from the bill. Birds are often mistaken for cripples during or after hunting seasons. Those suffering from acute poisoning do not attempt to escape but will often seek isolation and protective cover making them difficult to find. In some species, the head and neck position may appear 'crooked' or bent during flight. The wings may be held in an arched position which is followed by wing droop. A lot of green faeces in areas used by waterfowl may suggest lead poisoned birds and warrants further searches. Those suffering from acute poisoning may die with few clinical signs or lesions, but there are usually several weeks between exposure and death.

Lead poisoned mute swan *Cygnus olor* with typical kinked neck and drooped wings (*Martin Brown*).



Radiograph of dense pieces of lead shot in the gizzard of a lead poisoned swan (*Martin Brown*).

Dead animals are usually the first sign of lead poisoning in livestock. Live animals show signs of central nervous system damage. They may stop grazing and appear unresponsive and lethargic. These symptoms may be accompanied by muscle twitches (which may be more obvious around the face), blindness, staggering and gazing at the sky ('star-gazing').

Obvious symptoms in humans usually don't appear until sufficient amounts of lead have accumulated. Symptoms in children include: loss of appetite, weight loss, fatigue, abdominal pain, vomiting, constipation and learning difficulties. Symptoms in adults may include pain and numbness, muscular weakness, headache, abdominal pain, memory loss, miscarriage or premature birth in pregnant women and fatigue. A blue line around the gums and a metallic taste in the mouth may indicate lead poisoning. Other less 'identifiable' symptoms include affects on cognitive function, blood pressure and kidney function.

Recommended action if suspected

Contact and seek assistance from animal and/or human health professionals if there is any illness in birds, animals and/or people. Depending on local arrangements, suspected cases in livestock should be reported to national authorities.

Diagnosis

Confirmation of lead poisoning as a cause of death can only be determined by a combination of pathology, toxicological findings, clinical signs and field observations.

It is useful to record whether dead birds have lead shot or lead particles in the gizzard although this does not provide a confirmative diagnosis. For dead birds, whole carcases should be submitted to a diagnostic laboratory but if this is not possible, liver and/or kidneys can be submitted, frozen and wrapped separately in aluminium foil. Lead levels in live birds can be determined through blood screening and through indirect measurements using blood enzymes. For this, appropriate veterinary advice should be sought.

Post mortem examination should confirm lead poisoning through the detection of toxic levels of lead in kidney and/or liver tissue of affected animals. Blood samples can be taken from live animals suspected of having lead poisoning to confirm diagnosis.

For humans, a blood test can screen for harmful levels of lead in the body and confirm diagnosis.

PREVENTION AND CONTROL IN WETLANDS

Overall

To reduce the risk of lead poisoning in wildlife, livestock and humans, lead should be prevented from entering the environment.

Livestock

- Ensure that livestock do not have access to potential sources of lead such as old batteries, broken battery cases and spilled contents, lead paint, sump oil, contaminated soil from lead mining, and other farm machinery/rubbish.
- Check for these sources before putting stock onto new land and by checking areas ahead when driving stock.
- Animals in the early stages of poisoning are more likely to respond to treatment than those severely affected.

Wildlife

- Ensure that non-toxic shot is used for hunting. This is the only long-term solution for significantly reducing wild bird mortality from lead poisoning.
- Pick up and safely dispose of birds known, or suspected to be, contaminated by lead so that scavenging species do not ingest them.
- Exclude birds from heavily contaminated areas.
- Habitat management to temporarily reduce the availability of lead shot:
  - Lower water levels in feeding grounds after the hunting season to deter waterfowl from an area or increase water levels so that shot is out of reach of certain waterfowl species.
  - Turn the soil so that lead shot lies below the soil surface (>15 cm) so that it is not readily available to birds.
  - Plant food crops other than grains which may worsen the effects of lead ingestion.
  - Provide supplementary grit for waterbirds to ingest for digestion instead of shot.
  - Note that these actions can be expensive, labour intensive and of

limited effectiveness and should therefore not be relied upon as effective long-term solutions. These methods require knowledge of where the birds are picking up lead and knowledge of the wetlands' hunting history and historical lead exposure. Differences in feeding habitat should be considered for the broad spectrum of wildlife using the area.

- Treatment of poisoned birds is generally impractical but endangered species or those of high value may warrant treatment, which involves the use of lead-chelating chemicals under veterinary supervision.

Humans

Humans should reduce their exposure to lead by whatever means including reducing the amount of food consumed containing lead shot or other ammunition. Hunters should be encouraged by whatever means (legislation or education) to only use non-toxic shot when hunting.

IMPORTANCE

Effect on wildlife

Lead poisoning through the ingestion of lead gunshot is one of the most significant causes of death of wildfowl across the world and may also cause sub-lethal effects such as reduced survival and productivity. Lead poisoning is a particular problem in dabbling ducks, diving ducks and grazing species and accounts for an estimated 9% of waterfowl mortality in Europe alone. Morbidity and mortality also occurs in bird species that predate and scavenge animals shot with lead ammunition and has also been reported in upland bird species, reptiles and small mammals. The impacts of lead poisoning on threatened animal species and populations are also a great cause for concern.

Effect on livestock

Lead is a common cause of morbidity and mortality in cattle but is less frequently reported in sheep, goats and other livestock. Domestic animals are most vulnerable when they have access to the sources of lead listed above. Mortality in exposed groups can be high if animals are not removed from the source promptly.

Effect on humans

Lead can cause damage to various body systems including the nervous and reproductive systems and the kidneys and can cause anaemia and high blood pressure. High exposure to lead can cause convulsions, coma and death. Children, foetuses and pregnant women are particularly vulnerable to its toxic effects and there is now considered to be no safe level of lead exposure below which toxic effects do not occur.

Economic importance

There is potential for significant economic losses to the livestock industry due to death and illness of poisoned animals and restrictions on the sale of produce. Even low levels of exposure, which may not cause clinical illness, can cause concentrations of lead residues in milk, offal and meat to exceed residue limits and be deemed unfit for human consumption. The effects of lead on cognitive function of humans, together with other health impacts, have socioeconomic impacts.

FURTHER INFORMATION

Useful publications and websites

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- USGS National Wildlife Health Center. **Concerns rise over known and potential impacts of lead on wildlife**. www.nwhc.usgs.gov/disease_information/lead_poisoning/index.jsp. [Accessed March 2012].
- Centers for Disease Control and Prevention (CDC). **Lead**. www.cdc.gov/lead. [Accessed March 2012].
- World Health Organisation (WHO). **Water-related diseases**. www.who.int/water_sanitation_health/diseases/lead/en. [Accessed March 2012].
- Wildpro. **Lead poisoning in waterfowl**. http://wildpro.twycrosszoo.org/S/00dis/toxic/MetalMineral/Lead_Poisoning.htm [Accessed March 2012].

Contacts

National Wildlife Health Center (USGS).
US enquiries: +1 608 270 2400
AskNWHC@usgs.gov

Leptospirosis

Wetlands with stagnant water and animal ponds

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Autumn fever (*akiyami*), cane-cutter's fever, canicola fever, haemorrhagic jaundice, mud fever, redwater of calves, rice-field fever, sewerman's flu, Stuttgart disease, swamp fever, swineherd's disease, Weil's disease or syndrome

KEY FACTS

What is leptospirosis?

A bacterial infection that affects humans and animals following exposure to species of *Leptospira spp.* bacteria. Bacteria are excreted into the environment in the urine of infected animals and can survive for up to several months in contaminated soil and for several weeks in contaminated mud slurries, although they do not survive well in river water. The primary reservoir hosts for most *Leptospira* species are wild mammals, particularly rodents, in which they cause little or no clinical disease.

Leptospirosis is most commonly transmitted indirectly through contact with contaminated water or soil but can also be transmitted directly between mammalian hosts. It is mainly endemic in countries with humid subtropical or tropical climates and is a notable cause of morbidity and mortality in humans and animals in the western hemisphere. It occurs most commonly during the rainy season in the tropics and in the summer and autumn in temperate regions. Conditions leading to an increase of contaminated surface water or soil, such as rain, floods and disasters increase the risk of leptospirosis and may result in epidemics. In addition, during periods of drought, risks of infection may increase in association with the attraction of both humans and animals to water bodies.

In humans, the range of symptoms is very wide and variable, from mild non-specific signs to lethal infection.

Causal agent

Species of bacteria from the genus *Leptospira*, including *L. grippotyphosa*, *L. canicola*, *L. hardjo*, *L. pomona, L. bratislava, L. icterohaemorrhagiae, L. interrogans, L. noguchii, L. santarosai, L. meyeri, L. borgpetersenii, L. kirschneri, L. weilii, L. inadat, L. fainei* and *L. alexanderi*. Taxonomy is complex, but strains are commonly described as serovars. There are over 200 pathogenic serovars with many being host adapted to wildlife species in which they cause little clinical disease.

Species affected

All terrestrial and marine mammals appear to be susceptible. Most commonly found in many species of wild and domestic animals including rodents, cattle, sheep, goats, pigs, horses and dogs. Humans, particularly those working in or close to water, are very susceptible to illness caused by certain strains. Infection in reptiles, amphibians and birds is rare.

Geographic distribution

Occurs worldwide but most commonly in temperate or tropical climates with high rainfall. The highest concentrations of cases are often in developing countries where wet farming and rodent populations combine and where freshwater floods may occur.

Environment	Any environment supporting species of *Leptospira spp.* and their animal hosts. Leptospirosis is particularly prevalent in warm and humid climates, marshy or wet areas, and in regions with an alkaline soil pH. The importance of each species differs between geographical regions.

TRANSMISSION AND SPREAD

Vector(s)	Infected terrestrial and marine mammals.
How is the disease transmitted to animals?	Infection is acquired through direct contact with infected urine or indirect contact with urine-contaminated water/soil/vegetation or food. Bacteria gain entry across intact mucous membranes or broken skin. Occasionally, infection can spread through the inhalation/ingestion of aerosolised urine or water. Transmission may also occur through contact with infected normal, aborted or stillborn foetuses, or vaginal discharge and placental fluids.
How does the disease spread between groups of animals?	Infection is spread from one animal group to another by an infected animal which will shed the bacteria into the environment, most commonly in urine. Infection is maintained through survival of bacteria in the kidney of a reservoir host, where they are protected from the host's immune response.
How is the disease transmitted to humans?	Infection is acquired through contact with water, food or soil contaminated with urine from infected animals, especially rats. Bacteria may be ingested or may gain entry across intact mucous membranes or broken skin. Direct person to person transmission is rare but possible. Transmission occurs less commonly through the bite of a rodent.

IDENTIFICATION AND RESPONSE

Field signs	In reservoir wildlife hosts infection is likely to be asymptomatic, with little clinical disease. In accidental hosts symptoms may be very variable, and depend, in part, on the bacterial strain involved. Initial clinical signs are generally non-specific and include lethargy and anorexia, associated with fever. In dairy cattle, reduced milk production may be observed. Disease may progress to septicaemia and in some cases may result in death of the host. Infection during pregnancy may result in abortion, still-birth, weak offspring or infected but healthy offspring. In horses, many infections are subclinical and eye disease is the most common symptom. Seals and sea lions may suffer from fever, abortions and neonatal deaths. In humans, the disease picture is also highly variable. During the initial incubation period of roughly seven days (range 2-19), signs are non-specific and include fever, headache, chills, a rash and muscular pain. The kidneys and liver are common target organs and symptoms might include vomiting, anaemia and jaundice. Meningitis, eye pathology and haemorrhage in the lungs have also been reported.
Recommended action if suspected	Contact and seek assistance from human and animal health professionals immediately if there is any illness in people and/or livestock. The disease is notifiable and suspected cases must be reported to local and national authorities and the OIE.
Diagnosis	Clinical diagnosis is not straightforward due to the non-specific nature and wide variability in symptoms observed. Demonstration of the presence of the organism or an antibody response to the organism are required. Bacteria may

be isolated from blood and cerebrospinal fluid in the first seven days, and from urine during the second and third week of illness. An antibody response may be detected in the blood from 5-7 days after infection. A rising antibody level confirms current infection. In dead animals, the liver, lung, brain, kidney, genital tract and the body fluid of foetuses can be used for detecting bacteria.

PREVENTION AND CONTROL IN WETLANDS

Overall

Monitoring and surveillance - recording the incidence of outbreaks can identify trends in *Leptospirosis spp.* infections and assist in evaluating the feasibility of control programmes. Monitoring of outbreaks in animals and humans can also help assess the contribution of animals to human illness.

Selective rodent control can prevent infections in livestock and humans, particularly in urban areas.

Minimise contact with reservoir host species, rodents in particular, and minimise contact with potentially contaminated food/water/bedding.

Livestock

- **Good sanitation** and the **prevention of contact** with contaminated environments or infected wildlife, particularly rodents, can decrease the risk of infection.
- Prevention of contamination of food and bedding by rodents.
- **Fence stream banks and watering holes**, to limit access by livestock to water bodies contaminated by urine from infected animals, and to reduce contamination of water courses. Provide clean drinking water in separate watering tanks located away from potentially contaminated water sources.
- **Chlorinate** contained drinking water sources and prevent urine contamination of food and water where possible. Do not chlorinate natural water bodies as this will have an adverse effect on the wetland ecosystem.
- Keep **livestock wastes** away from pastures, animal housing and feeding sites and **away from water courses** in so far as possible.
- **Isolate** infected animals.
- **Separate** young animals from older animals where practical.
- **Replacement stock** should be selected from herds that have tested negative for leptospirosis. Animals not known to be *Leptospira*-free should be quarantined for four weeks and tested before being added to the herd.
- **Vaccination** of pigs, cattle and dogs may prevent infection caused by certain bacterial strains and prevent abortions in cattle. Note that vaccination of animals may not completely prevent infection and the animals may remain carriers of the bacteria.
- **Antibiotics** may be used to treat infections caused by certain bacterial strains and may prevent disease and abortion in cattle.
- Fluid therapy, blood transfusion and other supportive care may also be necessary.

Wildlife

Sporadic cases occur in free-ranging wildlife, but are likely to go unnoticed. Wildlife species are more important as asymptomatic carriers of infection. Rodent control from a pest perspective may be important in this context, although prevention of contamination of feed, bedding and water, and water treatment, as discussed, may be more appropriate.

Humans

Prevent or minimise contact with contaminated or potentially contaminated freshwater bodies and infected animals where possible:

- Do not let animals urinate in water that humans contact.
- **Protect food** from sources of infection, particularly rodents, and always cook food thoroughly. Do not eat fish taken from contaminated water.
- **Wash fruit and vegetables** thoroughly, particularly if they are eaten raw. Ideally, vegetables and fruit should be peeled.
- **Avoid consuming untreated surface water**. All drinking water should be boiled unless it is known to be absolutely safe.
- **Good personal hygiene**, especially if working in or near water and with animals. Have disinfection facilities for hands, footwear, clothing, equipment and vehicles/trailers on entering or leaving areas with livestock and after contact with animals.
- **Wash hands** thoroughly with soap and warm water:
  - before preparing and eating food
  - after contact with potentially contaminated water sources
  - after contact with animals
  - after working outside.
- **Wear protective clothing** especially if working in or near water or with animals:
  - wear protective clothing and footwear, either disposable or easily disinfected re-usable clothes (*e.g.* gloves, face shields, waterproof clothing and boots)
  - have separate clothing and utensils for each person using areas with livestock
  - use waterproof dressings to cover broken skin.
- Do not allow water to enter the mouth (*via* the hands, or *via* food or clothing).
- **Avoid swimming** and other water-based activities in contaminated water. Look out for symptoms following such activities and seek early treatment if needed.
- **Mark areas** that have an increased risk of exposure (*e.g.* water bodies used by animals, open sewage works, areas flooded with fresh water) with warning signs.
- **Vaccination:** annual vaccination may provide protection against some bacterial strains, particularly for those working in or close to water and with animals.

Antibiotic treatment: preventative use can be considered for short periods, particularly for those in high risk groups, and is most effective if given early in the infection. Supportive care may also be necessary.

Be aware of symptoms and seek early treatment.

IMPORTANCE

Effect on wildlife

Infections are usually asymptomatic in wild animals, including rodents, although outbreaks on the west coast of the USA are not uncommon in marine mammals, with depression, fever, abortions and neonatal deaths in seals and sea lions.

Effect on livestock

Mortality may be high in calves and young or weak piglets but low in adults, many of which will have mild symptoms or show no signs of infection at all. Some infections may cause infertility and spontaneous abortion in cattle.

Effect on humans

Whilst most cases in humans are asymptomatic or relatively mild, a small proportion may develop more severe life-threatening illness, also known as Weil's disease. Death is uncommon, although it is more likely to occur in the elderly. Those working in or close to contaminated water are most likely to develop infection.

Economic importance

There is potential for significant economic losses to the livestock industry due to illness, abortions and reduced milk yield of infected animals and likely trade restrictions imposed during and after an outbreak.

Illness in humans can result in significant economic losses due to the time lost from normal activities.

FURTHER INFORMATION

Useful publications and websites

- World Organisation for Animal Health (OIE). **Chapter 2.01.09: Leptospirosis**. Manual of diagnostic tests and vaccines for terrestrial animals. http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.09_LEPTO.pdf. [Accessed March 2012].
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- World Health Organization/International Leptospirosis Society. **Human leptospirosis: guidance for diagnosis, surveillance and control**. (2003) whqlibdoc.who.int/hq/2003/WHO_CDS_CSR_EPH_2002.23.pdf [Accessed March 2012].
- Wetlands International. **Wetlands & water, sanitation and hygiene (WASH) - understanding the linkages (2010)**. http://www.wetlands.org/WatchRead/Currentpublications/tabid/56/mod/1570/articleType/downloadinfo/articleId/2467/Default.aspx [Accessed March 2012].
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- **The Leptospriosis Information Center**. www.leptospirosis.org. [Accessed March 2012].
- World Health Organisation (WHO). **Leptospirosis**. www.who.int/zoonoses/diseases/leptospirosis/en. [Accessed March 2012].

Contacts

- **WHO Communicable Diseases Surveillance and Response (CSR)**. zoonotic_alert@who.int, fmeslin@who.int and outbreak@who.int
- **FAO Animal Production and Health Division**. www.fao.org/ag/againfo/home/en/who.htm

Oyster diseases

Shellfish reefs

Wildlife ✓
Aquaculture ✓
Human ✓

N

Synonyms: bonamiosis, marteiliosis, perkinsosis (dermo disease), marteiliodosis, microcell disease, hemocyte disease, winter mortality, aber disease, digestive gland disease, QX disease

KEY FACTS

What is Oyster disease?

Oysters are subject to a number of diseases which can impact the local population and reduce harvests in a commercial setup. A number of these diseases are associated with parasitic infections.

Oysters that are produced in areas contaminated with biotoxins or heavy metals could potentially cause health concerns for humans. Humans are also at risk when consuming raw oysters which contain levels of *Vibrio* (Gram-negative bacteria).

Causal agent

There are a number of causal agents recognised for oyster diseases. Examples of major oyster diseases and their causal *protozoan* agents are:

- bonamiosis (*Bonamia exitiosa, B. ostreae*)
- marteiliosis (*Marteilia refringens*)
- perkinsosis (*Perkinsus marinus, P. olseni*)

Bacteria of particular concern for human health include *Vibrio parahaemolyticus*, *V. vulnificus* and choleragenic *V. cholera.* Illness in humans is linked to the consumption of raw oysters.

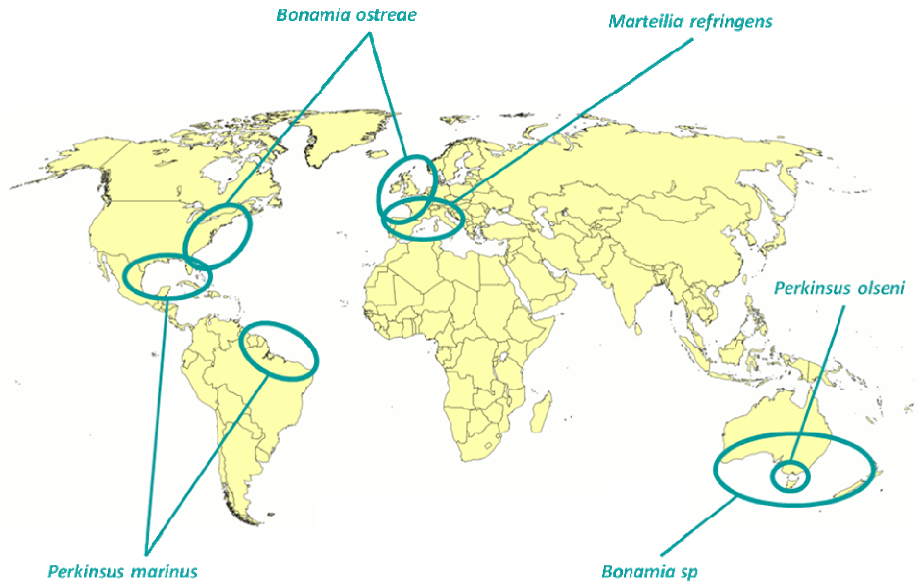
Species affected

Farmed and wild oysters worldwide are affected by diseases and those species known to be susceptible are:

Scientific name	Common name
Ostrea angasi	Australian mud oyster
O. chilensis	Chilean flat oyster
O. edulis	European flat oyster
O. puelchana	Argentinean flat oyster
O. denselammellosa	Asiatic oyster
Crassostrea gigas	Pacific oyster
C. virginica	Eastern oyster
C. ariakensis	Suminoe oyster

Geographic distribution

The above-mentioned oyster diseases (infection with *B. exitiosa*, *B. ostreae*; infection with *M. refringens*; infection with *P. marinus*, *P. olseni*) are notifiable OIE-listed diseases and now occur worldwide.



Geographic distribution of oyster diseases and their causal agents.

Environment

The causative pathogens live in aquatic environments in both tropical and temperate zones. High temperatures and salinities favour the proliferation of some of the pathogens.

TRANSMISSION AND SPREAD

Vector(s)

No data are currently available with respect to possible vectors.

How is the disease transmitted to animals?

The mode of transmission differs depending on the disease and its causal agents.

1. Bonamiosis: infection with the protozoan parasites *B. exitiosa* or *B. ostreae*

There is marked variation in susceptibility to this infection between bivalve genera. Prevalence and intensity of infection tends to increase during the warm water season. The parasite is difficult to detect prior to the proliferation stage of its development or in survivors of an epidemic. Infections may be detected in the first year of growth in areas where the disease is endemic but prevalence of infection and mortality is noticeably higher during the second year of growth.

Clean oysters living in close proximity to infected oysters (and artificial tissue homogenate/haemolymph inoculations) can precipitate infections indicating that transmission is direct (no intermediate hosts are required). There is a pre-patent period of 3-5 months between exposure and appearance of clinical signs of *B. ostreae* infection. In New Zealand, the pre-patent period for *Bonamia spp*. infection may be as little as 2.5 months and rarely exceeds 4 months.

2. Marteiliosis: infection with *M. refringens, M. sydneyi*

Marteilia refringens has a broad host range and transmission appears to be restricted to periods when water temperatures exceed 17°C. High salinities may impede *Marteilia spp*. multiplication within the host tissues. *Marteilia sydneyi* also has a seasonal period of transmission with infections occurring generally from mid- to late-summer (January to March). Heavy mortalities and sporulation occur all year round. The parasite enters the oyster through the epithelium of the palps and gills and develops and proliferates within the digestive tract.

The route of infection and life-cycle outside the mollusc host are unknown although the life cycle within oysters has been well documented. Since it has not been possible to transmit the infection experimentally in the laboratory, an intermediate host is suspected (possibly a copepod). This is reinforced by recent observations showing spores do not survive more than 7-10 days once isolated from the oyster. Cold temperatures prolong survival (35 days at 15°C). Spore survival within fish or birds is limited to 2 hrs, suggesting they are an unlikely mode of dispersal or transmission.

3. Perkinsosis: infection with *P. marinus, P. olseni*

Proliferation of *Perkinsus spp*. correlates with warm water temperatures (>20˚C) and this coincides with increased clinical signs and mortalities. Effects appear cumulative with mortalities peaking at the end of the warm water season in each hemisphere. The infective stage is a biflagellate zoospore which transforms into the feeding trophozoite stage after entering the host's tissues where they multiply. *P. marinus* shows a wide salinity tolerance range and *P. olseni* is associated with full-strength salinity environments.

Direct transmission of *Perkinsus spp*. has been demonstrated by exposure of susceptible hosts to infected hosts, including cross-species transmission for *P. olseni*. There is currently no evidence of cross-genus transmission of *P. marinus*.

How does the disease spread between groups of animals?

Transmission of the parasite directly from host to host is possible and transmission by infective stages carried passively on currents between oyster beds is suspected. *Bonamia exitiosa* often infects wild populations of susceptible species. Transmission of marteiliosis by an intermediate host may also take place.

How is the disease transmitted to humans?

The majority of agents that cause oyster disease do not pose any human health risk. However, it is recommended not to eat oysters from areas of poor sanitation because they may be infected with *Vibrio spp*. bacteria that can cause illness in humans when ingested.

IDENTIFICATION AND RESPONSE

Field signs

Clinical signs of oyster diseases may include cessation of growth, gaping oysters and occasionally mass mortality of oysters in the wild and in farms. A decline in body condition may be seen and discolouration of the digestive glands, mantle and gills may be visible in heavily infected individuals at gross *post mortem* examination.

Recommended action if suspected

The oyster diseases mentioned within (infection with *B. exitiosa*, *B. ostreae*; with *M. refringens*; with *P. marinus*, *P. olseni*) are notifiable and a suspected outbreak must be reported immediately to local (nearest fisheries or veterinary authority) and national authorities and the OIE. Guidance concerning collection and submission of samples must be sought.

Diagnosis

Presumptive diagnosis of most of the oyster diseases can be based on clinical signs and through cytological and tissue imprints in the laboratory. A confirmative diagnosis can be obtained using histopathology and/or transmission electron microscopy. The currently accepted procedures for a conclusive diagnosis of oyster diseases are summarised in the Manual of Diagnostic Tests for Aquatic Animals 2011 (OIE, 2011).

PREVENTION AND CONTROL IN WETLANDS

Environment

No protective vaccine or effective drug/chemical treatment is available for control of the above oyster diseases in natural water bodies.

Aquaculture

There is currently no available vaccine or chemical control agent for these diseases.

Good farming practices can help reduce stress and thus the negative impact of disease. Sources of stress include exposure to extreme temperatures and salinity, starvation, handling and infection with other parasites.

Actions should be directed firstly at prevention of the disease as subsequent control can be very difficult.

A number of simple measures can minimise or prevent the spread of oyster diseases. These include:

- **Reduction in stocking densities** and/or **restocking** and **lowering of water temperatures** may suppress clinical manifestation of the disease although no eradication procedures have worked successfully to date.
- Development of **resistant stocks** of oysters.
- **Early harvesting** at 15-18 months of production and **subtidal culture** may also minimise the effects of disease on oyster production and profitability.
- **Prevention of introduction or transfer** of oysters from waters where causal agents are known to be enzootic into historically uninfected waters.
- The use of **increased salinities** which appear to suppress clinical manifestation of the disease caused by *Marteilia spp*.

Wildlife

Wild oyster beds should be monitored for signs of disease as, if infected, they may transmit disease to other beds both wild and farmed.

Humans

Humans must ensure that all biosecurity measures are followed to reduce the chance of spreading the infectious agents to previously uninfected sites.

IMPORTANCE

Effect on wildlife

Whilst most of the causal agents are naturally present in coastal water, oyster diseases do occur in wild populations. Direct impacts on wildlife are not clear, although indirect long-term effects may include threats to the environment and aquatic biodiversity through, for example, declining biomass and irreversible ecological disruption.

Effect on Aquaculture and Fisheries

High losses (up to 80-90% with bonamiosis) to oyster farmers through mortalities, and reduced growth/productivity. Increased operational cost of additional biosecurity measures.

Effect on humans

The agents causing oyster diseases do not pose any direct human health implications. However, oysters could potentially pose a health concern for humans in cases where they contain high levels of *Vibrio spp*. (*V. parahaemolyticus*, *V. vulnificus*, and choleragenic, *V. cholera*) and are consumed raw, or where the oysters are produced in an area containing biotoxin or heavy metal contamination.

Economic importance

Oyster disease has the potential to financially decimate those who run oyster farming operations. Subsequently, oyster diseases can negatively affect the community and industries depending on the oyster trade.

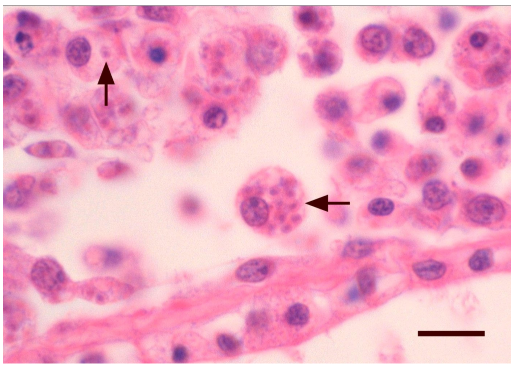
FURTHER INFORMATION

Useful publications and websites

- Australian Government Department of Agriculture Fisheries and Forestry. **Aquatic animal diseases significant to Asia-Pacific: identification field guide**. http://library.enaca.org/Health/FieldGuide/index.htm [Accessed April 2012].
- Food and Agriculture Organization (FAO) Fisheries. **Asia diagnostic guide to aquatic animal diseases**. **Technical Paper 402/2**. www.fao.org/docrep/005/y1679e/y1679e00.HTM [Accessed April 2012].
- FAO/WHO. **Risk assessment of *Vibrio vulnificus* in raw oysters: interpretative summary and technical report**. Microbiological risk assessment series No. 8. http://www.who.int/foodsafety/publications/micro/mra8.pdf [Accessed April 2012].
- OIE. **Manual of diagnostic tests for aquatic animals**. http://www.oie.int/en/international-standard-setting/aquatic-manual/access-online [Accessed April 2012].
- Levine J.F., Law M & Corsin F (2006). **Bivalves**. In: Invertebrate medicine (1st Ed.). Lewbart, G. A. (Ed.), Blackwell Publishing, (2006), pp.327.

Photos

Oysters infected with *Bonamia ostreae*, illustrating classic symptoms of *Bonamia ostreae* infection, *e.g.* gaping (*D. Alderman*).



Arrows point to *Bonamia ostreae* parasites inside haemocytes (blood cells) in the mantle of oysters (*The National Aquatic Animal Health Program (NAAHP) of Canada*).

Peste des petits ruminants

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✗

N

Synonyms: Contagious pustular stomatitis, goat plague, kata, pest of small ruminants, PPR, pneumoenteritis complex, pseudorinderpest of small ruminants, small ruminant plague, stomatitis-pneumoenteritis syndrome

KEY FACTS

What is peste de petits ruminants (PPR)?

A highly contagious viral disease, primarily affecting goats and sheep. It is characterised by the sudden onset of fever, depression, eye and nasal discharge, immunosuppression, lesions of the mouth, laboured breathing or coughing, diarrhoea and death. Although often characterised by high morbidity and mortality rates, pathogenicity can vary significantly, with clinical disease ranging from mild to severe. The outcome of infection may often be complicated by the involvement of pre-existing secondary pathogens.

Causal agent

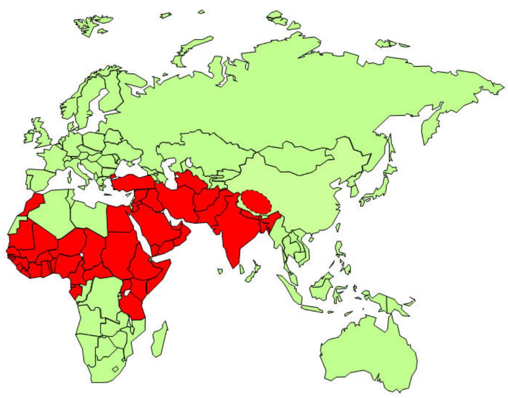
Peste des petits ruminants virus (PPRV), a member of the morbillivirus genus that includes measles virus and rinderpest virus (RPV).

Species affected

Small ruminants, predominantly sheep and goats, although many other species have been reported to be infected and develop clinical disease. The role of wildlife species in the transmission of the virus remains unclear although zoological collections in Saudi Arabia and various wildlife species across Africa have been shown to be susceptible (*e.g*. Arabian oryx *Oryx leucoryx*, Dorcas gazelle *Gazella dorcas*, Laristan sheep *Ovis orientalis laristanica*, gemsbok *Oryx gazella*, Nubian ibex *Capra nubiana*, Thomson's gazelle *Eudorcas thomsonii*, grey duiker *Sylvicapra grimmia*, kobs *Kobus kob* and Bulbal hartebeest *Alcelaphus buselaphus)*. Camels are also susceptible to infection and can display signs of clinical disease. Infection of other large ruminants (*e.g.* cattle and buffalo) and pigs has been reported although infection is generally subclinical in these species and viral excretion is unlikely.

Geographic distribution

PPR has historically been associated with outbreaks across West, Central and East Africa, India and the Middle East. However, PPRV is now also considered to be endemic across North Africa, China and parts of the Far East. Increased awareness of the disease and reporting systems have highlighted the presence of PPR in areas previously thought to be clear of the virus.



Historical and recent distribution of PPR

Environment

- Any areas that support the existence of susceptible animals, including wetlands.

TRANSMISSION AND SPREAD

Vector(s)

Although PPRV is not vector-borne, it may be spread mechanically by infected animals and contaminated objects (see below).

How is the disease transmitted to animals?

PPRV is most effectively transmitted between animals by direct contact, often through the inhalation of infective droplets. However, the virus is known to be excreted in eye and nasal discharge as well as, to a lesser extent, in urine and faecal matter. The UV lability and temperature sensitivity of the virus reduce the likelihood of transmission *via* routes other than droplet spread.

Transmission *via* infected bedding, water, feed troughs and other inanimate objects (fomites) is possible but is thought to occur at a very low level. There is currently no evidence for vertical transmission of PPRV (*i.e.* mother to offspring).

How does the disease spread between groups of animals?

PPRV is considered to be highly infectious, often spreading rapidly between groups of susceptible animals. Wherever animals are in close contact the potential for transmission exists *e.g.* markets. The variability in virulence between different isolates of the virus is currently poorly understood. However, animals can excrete and therefore spread the virus in the absence of clinical disease, often allowing the spread of virus to naïve populations when groups of animals are moved. Clinical disease is often preceded by a 4-5 day incubation period where animals must be considered to be contagious.

The appearance of clinical PPR in an area may be associated with: the introduction of animals from another area; the general movement of animals; contact with livestock returning unsold from market; contact with traded livestock or nomadic animals (*e.g.* shared grazing, water, housing); and husbandry changes.

How is the disease transmitted to humans?

PPRV is not known to be infectious for humans.

IDENTIFICATION AND RESPONSE

Field signs

PPR can quickly spread in populations of naïve small ruminants and cause the following symptoms:

- fever
- dry muzzle and dull coat
- discharge from the eyes, mouth and nose
- profound immunosuppression leading to the development of secondary infections
- sores on mucous membranes particularly in the mouth
- sudden onset of restless behaviour and depressed appetite
- scabs or nodules may be seen around the lips and muzzle in later stages
- laboured breathing, coughing and sneezing
- severe depression
- diarrhoea
- death (high mortality of up to 90% which can occur within 5-10 days after the onset of fever).

As well as causing high morbidity and mortality, the virus can also circulate in a mild form and can be very difficult to diagnose in the field. Factors affecting the outcome of infection include breed, age, immunological competence, general health, and the presence of secondary infections.

Recommended action if suspected

PPR is a notifiable disease and suspected cases must be reported immediately to local and national authorities and the OIE.

Subsequent and additional measures:
- quarantine affected area and restrict movement of animals
- avoid introduction of healthy animals
- collect samples (where appropriate and as directed)
- dispose of carcases (burning or burying as directed)
- disinfect in-contact fomites; most common disinfectants can be used.

Diagnosis

A tentative diagnosis can be made based on the clinical signs described above.

Laboratory confirmation is required for a definitive diagnosis of PPR as clinical signs are similar to many other diseases including bluetongue virus, contagious caprine pleuropneumonia, foot and mouth disease, contagious ecthyma, Nairobi sheep disease, capripox virus, pasteurellosis and others.

Laboratory tests may detect the PPR virus itself, evidence of the presence of PPRV (virus antigen or genetic material) or antibodies against PPRV found in blood serum. Rapid laboratory diagnosis is achieved through immunocapture enzyme-linked immunosorbent assay (ELISA), counter immunoelectrophoresis, agar gel immunodiffusion and in some instances, polymerase chain reaction (PCR).

PREVENTION AND CONTROL IN WETLANDS

Environment

- Under ideal conditions (*i.e.* dark and cool) outside the body the virus is generally considered to be viable for less than four days and is able to spread only relatively short distances. The virus is inactivated by UV light and most lipid-solvent based detergents and is both thermo- (>70°C) and pH-labile (inactivated at pH <5.6 and > 9.6).
- The virus may survive for short periods in carcases and in refrigerated meat, and may survive for several months in salted or frozen meat.
- It is not well understood how the virus is maintained between outbreaks.

Livestock

Livestock stakeholders are advised to monitor susceptible animals closely and frequently for any signs of disease or developing illness. Where possible, any newly acquired small ruminants should be quarantined for a minimum of 21 days and monitored, before being released.

Epidemic

When PPR appears in a previously unaffected area, the following is advised:
- **Rapid identification and confirmation of the disease.**
  Contact a veterinarian immediately if unusual illness is noticed.
- **Humane slaughter and disposal of affected animals.**
  Infected animal carcases should be burned or buried deep, along with their contact fomites (bedding, feed *etc*).
- **Strict quarantine and control of movements.**
  Quarantine affected areas and avoid the introduction of healthy animals; isolate affected animals from the rest of the herd; do not allow contact

between sick animals and neighbouring livestock; restrict the movement of small ruminants to and from affected areas.

- **Disinfection and cleaning**
  Thoroughly clean and disinfect all contaminated areas and items (including holding pens, physical perimeters, clothing and equipment) with lipid solvent solutions of high or low pH and disinfectants.
- **Monitor** all livestock and interaction with susceptible wild animals closely.
- **Vaccination**
  Consider and seek advice on the best use of vaccine; strategically 'ring' vaccinate and/or vaccinate high-risk populations.

Endemic

In PPR-affected areas, disease outbreaks are controlled by a combination of **quarantine** and **vaccination:**

- **Ring vaccination** in areas surrounding a PPR outbreak. This involves vaccinating susceptible animals in a given zone, forming a buffer of immune individuals that then limit disease spread.
- **Vaccination** of **high-risk** populations in high-risk areas (prophylactic immunisation).

Both vaccinated animals and small ruminants that recover from infection with PPRV generate a long lasting immunity that may last the lifetime of the animal.

Treatment

There is no specific treatment for PPR but antibiotics and other supportive treatment may prevent secondary infections and decrease mortality.

Wildlife

The role of wildlife in the maintenance and transmission of PPR remains unclear. However, numerous wildlife populations are susceptible and caution must be taken, by restricting interaction of livestock with wildlife species, and restricting movement of livestock where virus is known to be circulating.

Humans

Livestock stakeholders such as veterinarians, traders, community animal health workers and members of pastoral communities, play an important role in the prevention and control of PPR. Raising awareness of the disease (signs of the disease, how the virus is spread, the role of trade and disease diagnosis), its reporting and prevention, and how best to control outbreaks, is fundamental to PPR control.

IMPORTANCE

Effect on wildlife

The host range of PPR in wild animals is still not fully understood, and the conservation status of some susceptible wildlife species could be at risk.

Effect on livestock

PPR causes heavy losses to goat and sheep stock and is a major factor that affects the development of sustainable agriculture and food security.

Effect on humans

There is no evidence to suggest direct public health implications exist although outbreaks threaten food security, especially for subsistence farmers, causing a substantial reduction in the availability of animal protein, as well as essential micro-nutrients, for human consumption.

Economic importance

Direct and severe economic losses may be observed as a result of PPR, especially for pastoralist households and populations that rely on small ruminants as trade commodities. Disease outbreaks are a substantial threat to livelihoods which may already be under strain due to recurrent droughts and other pressures.

The presence of PPR in a region also seriously constrains export, trade and the development of livestock production.

FURTHER INFORMATION

Useful publications and websites

- World Organisation for Animal Health (OIE). **Chapter 2.07.11: Peste des petit ruminants**. Manual of diagnostic tests and vaccines for terrestrial animals. http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.07.11_PPR.pdf [Accessed March 2012].
- Food and Agricultural Organization (FAO). **Recognizing peste des petits ruminants**. ftp://ftp.fao.org/docrep/FAO/003/X1703E/X1703E00.PDF [Accessed March 2012].
- Banyard, A. C., Parida, S., Batten, C., Oura, C., Kwiatek, O. & Libeau, G. (2010). **Global distribution of peste des petits ruminants and prospects for improved diagnosis and control**. *Journal of General Virology*, 91 (12): 2885-2897. http://jgv.sgmjournals.org/content/91/12/2885.full.pdf+html [Accessed March 2012].
- World Organisation for Animal Health (OIE). **Technical disease card: PPR**. http://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/PESTE_DES_PETITS_RUMINANTS_FINAL.pdf [Accessed March 2012].
- The Centre for Food Security and Public Health (CFSPH). **Peste des petits ruminants**. http://www.cfsph.iastate.edu/Factsheets/pdfs/peste_des_petits_ruminants.pdf [Accessed March 2012]. World Organisation for Animal Health (OIE). **World Animal Health Information Database (WAHID) Interface**. http://web.oie.int/wahis/public.php?page=home [Accessed March 2012].
- Merck & Co. Inc. **The Merck veterinary manual: peste des petits ruminants**. http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/56100.htm [Accessed March 2012].

Contacts

- **FAO Animal Production and Health Division**. www.fao.org/ag/againfo/home/en/who.htm [Accessed March 2012].
- **WHO Communicable Diseases Surveillance and Response (CSR)**. zoonotic_alert@who.int, fmeslin@who.int and outbreak@who.int

Laboratory confirmation

Samples for diagnostic confirmation can be submitted to:

- **FAO Reference Laboratory For PPR (CIRAD-EMVT), Campus international de Baillarguet,** Montferrier-sur-Lez, BP 5034**,** 34032 Montpellier**,** Cedex 1**,** France**,** +33 4 67593705**,** diallo@cirad.fr.
- **FAO World Reference Laboratory for Rinderpest Reference Laboratory for PPR, Institute for Animal Health, Pirbright Laboratory,** Ash Road**,** Pirbright, Woking**,** Surrey GU24 0NF**,** United Kingdom**,** +44 1483 232441**,** ann.boddy@bbsrc.ac.uk.
- Detailed instructions for the collection and dispatch of PPR samples can be found in the publication **Collection and submission of diagnostic specimens to the FAO World Reference Laboratory for Rinderpest**. www.fao.org/docrep/007/v9813e/v9813e00.htm.

Ranavirus infection

Any freshwater wetland supporting susceptible animals

Wildlife ✓
Livestock ✓
Human ✗

N

Synonyms: Ranaviral disease, ranavirosis

KEY FACTS

What is ranavirus?

Ranavirus is a genus of iridoviruses that can infect amphibians, reptiles, and/or fish. Ranaviruses can lead to high levels of mortality in certain species and subclinical carrier status in others. Signs include swelling of the limbs or body, reddening and ulceration of the skin, and internal haemorrhage. Death in susceptible amphibians can occur within a few days following infection or may take several weeks. Amphibian species differ in their susceptibility to ranaviruses. The occurrence of recent widespread amphibian population die-offs from ranaviruses may be an interaction of suppressed or naïve host immunity, anthropogenic stressors, habitat degradation and the introduction of novel virus strains.

Causal agent

Ranaviruses. There are several different types of ranaviruses, some of which may be more host specific than others.

Species affected

Amphibians of the orders Anura and Caudata: salamanders (*e.g. Ambystoma spp*.), toads (*e.g. Bufo spp*.), frogs (*e.g. Limnodynastes spp*., *Rana spp.*) and others. Ranaviruses also infect fish and reptiles, and some ranavirus isolates may be able to infect animals from more than one class.

Susceptible age groups: larvae and metamorphs are most commonly affected in North America. Adult morbidity and mortality is reported more commonly in Europe. The effect on eggs remains unknown.

Geographic distribution

The disease has been reported in North and South America, Asia, the Pacific and Europe.

Environment

Any freshwater environment inhabited by amphibians, fish or reptiles.

TRANSMISSION AND SPREAD

Vector(s)

Infected animals, especially those exhibiting carrier status. Mechanical transport on the feet of livestock or fomites (inanimate objects).

How is the disease transmitted to animals?

Horizontal transmission: direct contact, cannibalism, through the water. Vertical transmission (parent to offspring): suspected but remains unknown. Clinical carrier status with ranaviruses can occur. Movement of ranaviruses into an area will most probably happen by movement of infected amphibians, fish or reptiles or *via* equipment and other inanimate objects that have been contaminated with ranaviruses. Generally, ranaviruses have low host specificity (*i.e.* they can infect a wide range of species). The viruses are highly infectious and capable of surviving for extended periods of time in the environment, even in dried material.

How does the disease spread between groups of animals?

Environmental persistence of ranavirus virions outside a host may be several weeks or longer in aquatic systems. Transmission occurs by indirect and direct routes, and includes exposure to contaminated water or soil, contact with infected individuals, and ingestion of infected tissue during predation, cannibalism or necrophagy (consumption of carcases/carrion).

How is the disease transmitted to humans?

Ranaviruses are not zoonotic.

IDENTIFICATION AND RESPONSE

Field signs

Field signs can vary from numerous dead amphibians visible in, and surrounding, water bodies to no dead amphibians visible (especially in areas where they are swiftly scavenged). Diseased larval amphibians often have swollen bodies and signs of internal and cutaneous haemorrhage. Affected adult amphibians may have reddening of the skin, skin ulceration, bloody mucus in the mouth and might pass blood from the rectum; often there is systemic internal haemorrhaging (which also may be seen in affected fish and reptiles). Anorexia, lethargy and/or ataxia might also be evident. These signs are all typical of the disease syndrome 'red leg': ranaviruses are not the only possible cause of 'red leg' in amphibians and other differential diagnoses should be borne in mind.

Chronically infected, inapparent carriers have been described. Seasonal variations in disease outbreaks have been reported, with both their prevalence and severity being greater during the warmer months, therefore temperature is considered a likely factor influencing disease outbreaks.

Recommended action if suspected

The disease is notifiable in amphibians (as are certain fish ranaviruses) and suspected cases must be reported immediately to local and national authorities and the OIE. Dead animals should be submitted to a suitable diagnostic laboratory for *post mortem* examination. Surveillance of live animals should be carried out if possible and sick animals submitted for testing.

Diagnosis

Liver and/or kidney samples from dead animals should be sent to an appropriate laboratory for diagnostic testing. Toe or tail clips from live animals might also be used for diagnosis, but the reliability of these has not been validated.

Tests carried out on samples include: PCR, real-time PCR, electron microscopy, virus isolation (followed by immunofluorescence, PCR or electron microscopy) and histology (followed by immunohistochemistry or electron microscopy).

Before collecting or sending any samples from animals with a suspected disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorised laboratories to prevent the spread of the disease. Although ranaviruses are not known to be zoonotic, routine hygiene precautions are recommended when handling animals. Also, suitable precautions must be taken to avoid cross contamination of samples or cross-infection of animals.

PREVENTION AND CONTROL IN WETLANDS

Environment

Ensure that the site is regularly scanned for dead amphibians, fish and reptiles. Ideally any site containing a reasonable population of amphibians should be monitored for sick and dead animals as a matter of course. If sick or dead animals are found, they should be tested for ranavirus infection so that the site's ranavirus status can be determined.

People coming into contact with water, amphibians, reptiles or fish should ensure where possible that their equipment and footwear/clothing has been cleaned and fully dried before use if it has previously been used at another site.

To properly clean footwear and equipment:

- first use a brush to clean off organic material *e.g.* mud and grass
- rinse with clean water
- soak in disinfectant
- rinse with clean water and allow to dry.

If any clothing is particularly soiled during activities, then washing at 40°C with detergent should be sufficient to remove any contamination with ranavirus. Ideally, different sets of footwear should be used at the site than are used by staff at home.

Biosecurity measures should be increased to reduce the chance of spread if disease is confirmed.

Livestock

It is important to reduce the chance that livestock moving between sites (especially those travelling from known infected sites) will carry infected material on their feet or coats. This can be accomplished by ensuring that feet are clean before transport. Foot baths can be used and animals should be left in a dry area after the bath for their feet to fully dry before transport.

Wildlife

Do not allow the introduction of amphibians, reptiles or fish without thorough screening and quarantine for ranavirus. This screening may still not pick up all subclinically infected individuals but will reduce the risk of actively infected animals being introduced to the site. Also, remember that the virus can be introduced with water or aquatic plants.

Humans

Humans must ensure that all biosecurity measures described above are followed to prevent introduction of the infectious agent into previously uninfected areas.

IMPORTANCE

Effect on wildlife

May cause epidemics with very high mortality rates, dependant on virus and host species. The disease has been shown to cause significant population declines of common frog *Rana temporaria* in the United Kingdom, apparently following virus introduction from North America. Ranavirus infection might be implicated in declines elsewhere, but data are lacking.

Effect on livestock

None other than farmed amphibians and fish. ► Economic importance

Effect on humans

None

Economic importance

Fish ranaviral diseases can cause major economic losses of high value species, such as rainbow trout *Oncorhynchus mykiss*. Ranaviruses also are considered to be of some economic importance due to disease and mortalities in farmed American bullfrogs *Lithobates catesbeianus* and harvested edible frogs *Rana esculenta.* There are potential economic losses due to potential risk of disease spread to fish.

FURTHER INFORMATION

Useful publications and websites

- World Organisation for Animal Health (OIE). **Disease card: infection with ranavirus**. http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/Ranavirus_card_final.pdf [Accessed March 2012].
- European Association of Zoo and Wildlife Veterinarians (EAZWV). **Transmissible disease fact sheet: ranavirus infection in amphibians**. www.eaza.net/activities/tdfactsheets/050%20Ranavirus%20Infection%20In%20Amphibians.doc.pdf [Accessed March 2012].
- Robert, J. (2010). **Emerging ranaviral infectious diseases and amphibian decline**. *Diversity*, 2,3, 314–330. http://www.mdpi.com/1424-2818/2/3/314/ [Accessed March 2012].
- Speare, R. (2003). **Summary of formidable infectious diseases of amphibians**. www.jcu.edu.au/school/phtm/PHTM/frogs/formidable.htm [Accessed March 2012].
- World Organisation for Animal Health (OIE). **Diagnostic manual for aquatic animal diseases**. http://www.oie.int/doc/ged/D9568.PDF [Accessed March 2012].

Contacts

- OIE reference laboratories and collaborating centres for diseases of amphibians, crustaceans, fish and molluscs: http://www.oie.int/fileadmin/Home/eng/Health_standards/aahm/2010/3_LIST_OF_LABS.pdf.

Rift Valley fever

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonym: RVF

KEY FACTS

What is Rift Valley fever?

An insect-borne viral disease that primarily affects animals but can also affect humans. The virus is mostly transmitted by the bite of infected mosquitoes, mainly of the *Aedes* species, which acquire the virus when feeding on infected animals. The main amplifying hosts are sheep and cattle. The disease can cause abortions and high mortality in young animals throughout its geographic range. In humans it causes a severe influenza-like illness, with occasionally more serious haemorrhagic complications and death.

Causal agent

Rift Valley fever virus (RVFV) from the genus *Phlebovirus.*

Species affected

Many species of terrestrial mammal, particularly sheep, cattle and wild ruminants, although most indigenous livestock species in Africa are highly resistant to the disease. Humans are very susceptible.

Geographic distribution

Endemic in tropical regions of eastern and southern Africa, with occasional outbreaks in other parts of Africa. Rift Valley Fever (RVF) was detected outside Africa for the first time in 2000, with cases in Saudi Arabia and Yemen

Environment

An epidemic can occur when there is a susceptible livestock population, a large population of vector mosquitoes and the presence of the RVFV. Major epidemics occur at irregular intervals of 5-35 years: in Africa, outbreaks typically occur in savannah grasslands every 5-15 years, and in semi-arid regions every 25-35 years. Epidemics are associated with the hatching of mosquitoes during years of heavy rainfall and flooding.

TRANSMISSION AND SPREAD

Vector(s)

Mainly mosquitoes (*e.g. Aedes, Anopheles, Culex, Eretmapodites* and *Mansonia* species) and other biting insects.

How is the disease transmitted to animals?

Most commonly spread by the bite of an infected mosquito. Mosquitoes become infected when they feed on infected animals and the female mosquito can also transmit the virus directly to her offspring *via* eggs. In mammalian species the virus can also be transmitted to the foetus of an infected female.

How does the disease spread between groups of animals?

The main amplifying hosts are sheep and cattle and once livestock are infected, many species of mosquitoes (*e.g. Aedes, Anopheles, Culex, Eretmapodites* and *Mansonia* species) and biting insects can then spread the disease to other animals and humans. Transmission can also occur through direct contact, which may become relatively more important as an outbreak progresses.

How is the disease transmitted to humans?

Humans can be infected through the bite of an infected mosquito, but most reported cases occur through contact with the blood or organs of infected animals, through the handling of animal tissue during slaughtering or butchering, assisting with animal births, conducting veterinary procedures, or from the disposal of carcases or foetuses. The disease may be spread by ingesting the unpasteurised or uncooked milk of infected animals. The virus can also be transmitted vertically to the human foetus.

IDENTIFICATION AND RESPONSE

Field signs

There may be a sudden onset of large numbers of abortions in sheep ('abortion storms' with up to 100% of a flock affected), goats, cattle or camels and deaths in lambs, kids or calves, a high neonatal mortality, and the presence of liver lesions which may be particularly severe in foetuses and newborn animals. Jaundice may be noted in surviving lambs. There is a higher risk of an outbreak in irrigated areas or if there is surface flooding in savannah or semi-arid areas followed by prolonged rains, if the mosquito populations are high, and if there is concurrent illness.

Humans may suffer from influenza-like symptoms which can include fever, headache, muscular pain, weakness, nausea, sensitivity to light, loss of appetite and vomiting. Recovery usually occurs within 4–7 days. Complications can lead to ocular disease (with loss of vision), meningoencephalitis, hepatitis, haemorrhagic fever and occasionally death.

Recommended action if suspected

Contact and seek assistance from animal and human health professionals immediately if there is any illness in livestock and/or people. RVF is a notifiable disease and suspected cases must be reported immediately to local and national authorities and the OIE

Diagnosis

Isolation of the causative agent by health professionals is needed for a definitive diagnosis. For dead animals, whole blood, liver, lymph nodes and spleen are preferable tissues for detecting the virus. In live animals and humans, diagnosis is usually made by testing blood/serum.

PREVENTION AND CONTROL IN WETLANDS

Overall

Environmental (habitat) management

Encourage mosquito predators and their access to mosquito breeding habitats:

- Connect shallow water habitat (mosquito breeding areas) with deep-water habitat > 0.6m (favoured by larvivorous fish) with steep sides, through meandering channel connections, deep ditches and tidal creeks.
- Include at least some permanent or semi-permanent open water.
- Construct artificial homes or manage for mosquito predators such as bird, bat and fish species.

Reduce mosquito breeding habitat:

- Reduce the number of isolated, stagnant, shallow (2-3 inches deep) areas.
- Cover or empty artificial containers which collect water.
- Manage stormwater retention facilities.
- Strategic manipulation of vegetation.
- Vary water levels.

- Construct a vegetation buffer between the wetland and adjacent land to filter nutrients and sediments.
- Install fences to keep livestock from entering the wetland to reduce nutrient loading and sedimentation problems.

In ornamental/more managed ponds:

- Add a waterfall, or install an aerating pump, to keep water moving and reduce mosquito larvae. Natural ponds usually have sufficient surface water movement.
- Keep the surface of the water clear of free-floating vegetation and debris during times of peak mosquito activity.

Vector control (chemical)

It may be necessary to use alternative mosquito control measures if the above measures are not possible or ineffective:

- Use larvicides in standing water sources to target mosquitoes during their aquatic stage. This method is deemed least damaging to non-target wildlife and should be used before adulticides. However, during periods of flooding, the number and extent of breeding sites is usually too high for larvicidal measures to be feasible.
- Use adulticides to spray adult mosquitoes.
- The environmental impact of vector control measures should be evaluated and appropriate approvals should be granted before it is undertaken.

Biosecurity

Protocols for handling sick or dead wild animals and contaminated equipment can help prevent further spread of disease:

- Avoid contact with livestock where possible.
- Wear gloves whilst handling animals and wash hands with disinfectant or soap immediately after contact with each animal.
- Change or disinfect gloves between animals.
- Change needles and syringes between blood collection from different animals.
- Wear different clothing and footwear at each site and disinfect clothing/footwear between sites.
- Disinfect field equipment between animals and sites.

Monitoring and surveillance

- Regular inspection of sentinel herds (small ruminant herds located in geographically representative areas) in high risk areas such as locations where mosquito activity is likely to be greatest (*e.g.* near rivers, swamps and dams). As a general guide, sentinel herds should be sampled twice to four times annually, with an emphasis during and immediately after rainy seasons.
- In livestock, clinical surveillance for abortion with laboratory confirmation and serology, and disease in humans in areas known to have had outbreaks.

Livestock

- **Vaccination**
  - Animal vaccination must be implemented *prior* to an outbreak. Consider vaccination of all trade animals at 9-12 months of age. Vaccination in outbreak areas is *not* recommended.
- **Restrict or ban the movement of livestock** to slow the expansion of the virus from infected to uninfected areas:
  - Livestock should not be moved into/out of the high-risk epizootic areas during periods of greatest virus activity, unless they can be moved to an area where no potential vector species exist (such as at high altitudes).
  - All trade should cease once pre-epidemic conditions have been recognised and until at least six months after the last evidence of virus activity.
- **Bury animals** rather than butchering them as freshly dead animals are a potential source of infection.

Wildlife

RVF is thought to occur in endemic cycles between wild African ruminants and mosquitoes, with little apparent disease. For control of disease in captive collections of wild ruminant species, guidelines above for livestock, habitat and vector management may be applicable.

Humans

In the epidemic regions, **thoroughly cook** all animal products (blood, meat and milk) before eating them.

Avoid contact with livestock where possible [▶ Biosecurity section above].

Reduce the chance of being bitten by mosquitoes:

- Wear light coloured clothing which covers arms and legs.
- Use impregnated mosquito netting when sleeping outdoors or in an open unscreened structure.
- Avoid mosquito-infested areas or stay indoors when mosquitoes are most active.
- Use colognes and perfumes sparingly as these may attract mosquitoes.
- Use mosquito repellent when outdoors. Note that some repellents cause harm to wildlife species, particularly amphibians. Wash hands before handling amphibians.
- Use citronella candles and mosquito coils in well ventilated indoor areas.
- Use mesh screens on all doors and windows.

IMPORTANCE

Effect on wildlife

RVF is thought to occur in endemic cycles between wild African ruminants and mosquitoes with little apparent disease. African buffalo and domestic buffalo are considered 'moderately' susceptible with mortalities of less than 10%. Camels, equids and African monkeys including baboons are all considered 'resistant' with infection being inapparent. Birds, reptiles and amphibians are not susceptible to RVF.

Effect on livestock

Pregnant livestock are most severely affected with abortion of nearly 100% of foetuses. Lambs and kids are most at risk with mortalities of 70–100%, followed by sheep and calves (20–70%), and then adult cattle, goats and domestic buffalo (<10%).

Effect on humans

Whilst most cases in humans are relatively mild, a small proportion may develop more severe illness such as ocular (eye) disease (0.5-2% of people), haemorrhagic fever (<1%) or meningoencephalitis (<1%). Few infected humans die of the disease (1%).

Economic importance

There is potential for significant economic losses in the livestock industry due to death and abortion of infected animals and possible trade restrictions imposed during and after an outbreak. Illness in humans can result in economic losses due to the time lost from normal activities.

FURTHER INFORMATION

Useful publications and websites

- Food and Agriculture Organization (FAO). **Animal health manual No. 17: recognising Rift Valley fever**. www.fao.org/docrep/006/y4611e/y4611e00.htm#Contents [Accessed March 2012].
- Food and Agriculture Organization (FAO). **Animal health manual No. 15: preparation of Rift Valley fever contingency plans**. www.fao.org/DOCREP/005/Y4140E/y4140e00.htm#TopOfPage [Accessed March 2012].
- World Organisation for Animal Health (OIE). **Chapter 2.01.14: Rift Valley fever**. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.14_RVF.pdf [Accessed March 2012].
- World Health Organization (WHO). **Rift Valley fever factsheet**. www.who.int/mediacentre/factsheets/fs207/en/ [Accessed March 2012].

Further information on disinfectants

- FAO, Rome. **Manual on procedures and for disease eradication by stamping out**. (2001). www.fao.org/DOCREP/004/Y0660E/Y0660E03.htm [Accessed March 2012].

Contacts

- **International Rift Valley fever experts and laboratories (FAO)**. www.fao.org/DOCREP/005/Y4140E/y4140e13.htm#P1_10 [Accessed March 2012].
- **Rift Valley fever vaccine sources (FAO)**. www.fao.org/DOCREP/005/Y4140E/y4140e14.htm#TopOfPage. [Accessed March 2012].

Salmonellosis

Wetlands supporting groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: non-typhoidal salmonellosis, paratyphoid, Salmonella

KEY FACTS

What is salmonellosis?

An infectious zoonotic disease found in a range of animals including birds, caused by their exposure to species of *Salmonella spp.* bacteria. The bacteria are found in the intestines of humans and animals but are also widespread in the environment and are commonly found in farm effluents, human sewage and any material that is contaminated with infected faeces. The bacteria can survive for several months in the environment, particularly in warm and wet substrates such as faecal slurries.

The disease can affect all species of domestic animals, and many animals, especially pigs and poultry, may be infected but show no signs of illness. The infection can spread rapidly between animals, particularly when they are gathered in dense concentrations. Salmonellosis can occur at any time of year, however, salmonellosis outbreaks may be more common in certain seasons (*e.g.* European garden bird salmonellosis outbreaks occur most frequently during the winter months).

Humans usually contract the bacteria through the consumption and handling of contaminated foods of animal origin and water, but also through direct contact with infected animals and their faeces. Salmonellosis is one of the most common and widely distributed food-borne diseases in humans globally, constituting a major public health burden and representing a significant cost in many countries.

Causal agent

Two species of bacteria from the genus *Salmonella: Salmonella enterica*, and *S. bongori*. Within these, there are over 2,300 strains which are grouped into 'serovars'.

Species affected

Many species of domestic and wild animals including birds, reptiles, amphibians, fish and invertebrates can be infected with *Salmonella spp*. The importance of each *Salmonella* serovar (and phage type) differs between the host species. Some *Salmonella* serovars (and phage types) have a broad host range and others are thought to be highly host-adapted. Infection is most commonly seen in poultry, pigs and reptiles. All species seem to be susceptible to salmonellosis but clinical disease is more common in some animals than others. For example, disease is common in cattle, pigs and horses, but uncommon in cats and dogs.

The frequency of occurrence of *Salmonella spp.* infection and salmonellosis varies amongst wild bird species. Salmonellosis outbreaks, caused by certain phage types of *S. typhimurium*, commonly affect passerine species that are gregarious and seed-eating (*e.g.* finches and sparrows). Outbreaks of passerine salmonellosis are typically observed in the vicinity of supplementary feeding stations in garden habitats. Salmonellosis outbreaks have also been reported in colonial nesting birds, such as gulls and terns. Birds of prey can become infected with *Salmonella spp.* bacteria from prey items.

Humans are very susceptible to illness caused by certain *Salmonella spp*. Children, the elderly, and people with weakened immune systems are at greatest risk of developing severe disease.

Geographic distribution
Found worldwide but most common in areas of intensive animal husbandry, especially in pigs, calves and poultry reared in confined spaces. The importance of each serovar differs between geographical regions. Eradication programmes have nearly eliminated salmonellosis in domestic animals and humans in some countries but wild animal *Salmonella spp.* reservoirs remain.

Environment
Any environment supporting *Salmonella spp.* and their animal hosts.

TRANSMISSION AND SPREAD

Vector(s)
Salmonellosis can be spread mechanically by animals and insects. In general infection is transmitted by infected hosts, their faeces or contaminated inanimate objects.

How is *Salmonella* transmitted to animals?
Direct contact with infected faeces and through ingesting water and food (including pastures) contaminated with bacteria (often through faecal contamination). In mammals, the bacteria can be transmitted from an infected female to the foetus, and in birds, from an infected adult to the egg. Carnivores may be infected through ingesting infected animals and their products. Bacteria may also be inhaled in closely confined areas.

How does *Salmonella* spread between groups of animals?
Spread by infected animals which shed the bacteria into the environment in their faeces. Bacteria may also be introduced to herds and flocks on shoes, equipment and other contaminated objects (fomites). Birds, rodents and insects can spread bacteria to other animals. How the infection spreads between and within herds and flocks is not fully understood due to the difficulties of detecting clinical signs in animals infected with *Salmonella spp*.

How is *Salmonella* transmitted to humans?
Most commonly transmitted by handling and ingesting contaminated water and food, particularly undercooked foods of animal origin, such as meat, eggs or unpasteurised milk and dairy products, or from cross-contamination of other foods by these items. Also transmitted through direct contact with infected animals and their faeces, particularly those of reptiles, chicks and ducklings, but also of livestock, dogs, cats, adult poultry and cage birds. The bacteria may be spread through person-to-person contact if hygiene is poor.

IDENTIFICATION AND RESPONSE

Field signs
Many infected animals will not show any clinical signs and hence *Salmonella spp.* can be difficult to detect. Infected livestock may develop enteritis and septicaemia and commonly show signs of diarrhoea, dehydration, depression, abdominal pain and rapid weight loss. Pregnant animals may abort, either with or without other clinical signs. Clinical signs usually last for 2-7 days but death can occur within 24-48 hours in some species. Loss of condition, emaciation and lethargy may be seen in surviving livestock. In poultry, disease is usually seen in very young birds. Clinical signs may include ruffled feathers, lethargy, diarrhoea and increased thirst. Chronically infected birds often appear severely emaciated. Some may show poor coordination, tremors, convulsions and blindness.

Clinical disease usually appears when animals are stressed by factors such as transportation, crowding, food shortage or deprivation, weaning, giving birth, exposure to cold, a concurrent viral or parasitic disease, sudden change of feed, or overfeeding following a fast.

Infection in humans often causes gastroenteritis but a wide range of clinical signs may be seen and death can occur in severe cases. Illness usually occurs in single, sporadic cases, but outbreaks can also occur. Humans may suffer from fever, abdominal pain, diarrhoea, nausea and sometimes vomiting. Infection may last for 1-7 days. The elderly, children and those with weakened immune systems may suffer from severe dehydration and more severe illnesses, such as septicaemia. Some infected people do not show any symptoms at all.

Recommended action if suspected

Salmonellosis in sheep and goats is a notifiable disease and suspected cases must be reported immediately to local and national authorities and the OIE. In general, contact and seek assistance from human and animal health professionals immediately if there is any illness in people and/or livestock. An outbreak may mean that many humans and animals have been exposed to a common contaminated food item or water source.

Diagnosis

Isolation of the causative agent by health professionals is needed for a definitive diagnosis. Faeces or blood cultures are used for isolating the bacterium in humans, and in animals and birds, faeces, rectal swabs and/or caecal contents are required. Ideally, fresh faeces should be collected, preferably without traces of urine. Samples should be prevented from drying out. A medium should be used for transporting swabs.

For dead animals, whole carcases should be submitted to a diagnostic laboratory. If the whole carcase cannot be submitted, submit the intestine, and if possible, the liver and heart. Wrap each sample in a separate piece of aluminium foil. Place the foil-wrapped specimens in tightly sealed plastic bags, and ship them frozen. After an abortion, samples should be collected from the placenta, vagina and foetal stomach. Whole eggs, egg shells and shell membranes can also be cultured for bacteria providing that the egg fragments have not been subjected to environmental conditions that would destroy the bacteria.

PREVENTION AND CONTROL IN WETLANDS

Environment

Prevention and control measures are limited in wetlands with free-living animals, many of whom will carry the bacteria without any noticeable clinical signs and untoward effects. Transmission of bacteria from animals to humans and between captive animals can be more easily prevented and controlled.

Monitoring and surveillance
Recording the incidence of outbreaks can identify trends in salmonellosis infections and evaluate the feasibility of control programmes. Monitoring of outbreaks in animals and humans can also help assess the contribution of animals to human illness.

Livestock

The control of *Salmonella spp.* along the food chain is most effective when the colonisation of living animals with bacteria can be prevented.

A number of measures can be taken to help prevent or control infection:

- Good biosecurity will help protect captive animals from bacterial infection and prevent cross-contamination:
  - Have disinfection facilities for hands, footwear, clothing, equipment and vehicles/trailers on entering or leaving areas with livestock and after contact with animals. *Salmonella spp.* are susceptible to many disinfectants including 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, iodine-based disinfectants, phenolics and formaldehyde.
  - Wear protective clothing and footwear, either disposable or, if reusable, easily disinfected (*e.g.* waterproof clothing, face shields, gloves and boots).
  - Have separate clothing and utensils for each person using areas with livestock.

  Note that biosecurity does not guarantee a *Salmonella spp.*-free flock or herd at the time of slaughter.
- Disease can be reduced by good hygiene and optimal animal husbandry and by minimising stressful events.
- Rodent control will help prevent/reduce transfer of bacteria from rodents to animals.
- Fence stream banks and watering holes to limit access by livestock to water contaminated by faeces from infected animals and to reduce animals contaminating water courses. Provide clean drinking water in separate watering tanks located away from potentially contaminated water bodies.
- Treat sewage to reduce the release of bacteria into water courses.
  - **Chlorinate** contained drinking water sources and prevent faecal contamination of food and water where possible. Do not chlorinate natural water bodies as this will have an adverse effect on the wetland ecosystem.
  - **Feed sources** should be *Salmonella spp.*-free. Store feed in rodent and insect-proof sealed containers.
  - **Avoid mixing** potentially infected and susceptible animals.
  - **Isolate** newly acquired animals.
- Buy animals or eggs from *Salmonella spp.*-free sources.
- During a herd outbreak, animals carrying bacteria should be identified and either isolated and treated, or culled. Contaminated materials should be disposed of.
- Vaccination can reduce the level of colonisation and shedding of the bacteria into the environment, as well as clinical disease. Vaccines are available for some serovars such as *Salmonella dublin*, *S. typhimurium*, *S. abortusequi* and *S. choleraesuis*, in some countries.
- Re-test treated animals several times to ensure that they no longer carry *Salmonella spp.*
- Adequate colostrum intake is important in preventing disease in young animals.
- Antibiotics may help with overcoming an outbreak but will not eliminate carriers, and transmission of bacteria from an infected adult to the egg or foetus may result in new outbreaks and disease spread.
- Maintain low densities of livestock to reduce cycles of salmonellosis within populations.

Wildlife

- Eliminating point sources of infection should be the key activity for preventing and controlling salmonellosis in wild bird and other animal populations:
  - Feeding stations encourage birds to congregate, sometimes in large densities, thereby increasing the potential for disease to spread between individuals when outbreaks occur. Ensure that garden bird feeding stations are regularly cleaned. Remove spilled and soiled feed from the area under the feeder. Rotate the locations of feeders to help avoid accumulation of faeces and contamination of particular areas. If bird baths are used, ensure that water is clean and fresh on a daily basis.
  - Regularly disinfect feeders using a dilution (1:10 ratio) of household bleach and water or an aviary-safe disinfectant. Ensure that feeders are rinsed with clean water and air-dried before re-use.
  - Thoroughly disinfect feeding stations and discontinue use temporarily if a salmonellosis outbreak occurs. This is to reduce the opportunity for transmission of *Salmonella spp.* which might be increased when garden birds feed together in high densities at shared food and water sources.
- Avoid contaminating wetlands with wastewater known to harbour bacteria *e.g.* by use of constructed treatment wetland. This often happens when:
  - existing wetlands receive wastewater discharges
  - agricultural fields receive manure and slurries as fertiliser
  - development of landfill, livestock, and poultry operations are proposed.
- Ensure that waste, sewage wastewater, and wastewater discharges are properly treated, secure and contained away from livestock, poultry and wetlands:
  - wastewater should be stored in lagoons and treated for a combined period of 20 days to eliminate bacteria *e.g.* a primary lagoon for eight days, secondary lagoon for five days, detention pond for two days, and recycle pond for five days.

Humans

- Avoid consuming un-pasteurised dairy products (*e.g.* milk, cheese and colostrum), eggs and untreated surface water.
- Cook food thoroughly, especially eggs, meat and poultry. All meat should be cooked so that it is without blood and no longer pink.
- Wash fruit and vegetables thoroughly, particularly if they are eaten raw. Ideally vegetables and fruit should be peeled.
- People with weakened immune systems should avoid contact with reptiles, young chicks and ducklings.
- Good personal hygiene:
  - wash hands thoroughly with soap and warm water: before preparing and eating food; after handling raw food; after going to the toilet or after/before changing a baby's nappy; after contact with animals and (especially) reptiles or contact with items they have touched; after working outside; and frequently if you have symptoms such as diarrhoea.
- Prevent contamination of food in the kitchen.

No human vaccines to prevent salmonellosis exist.

Most people who have salmonellosis recover without treatment within 2-7 days. It is important to drink plenty of fluids as diarrhoea or vomiting can lead to dehydration and loss of minerals. Re-hydration solutions may also be useful. Antibiotics may be given to treat severe infections.

IMPORTANCE

Effect on wildlife

Many infected animals will not show any clinical signs at all and clinical disease is uncommon in healthy, unstressed adult birds and mammals. The prevalence of bacteria in most wild bird populations is generally low although large-scale mortalities of birds using feeding stations have become common in the United States and also occur with some frequency in Canada and Europe.

Effect on livestock

Many infected animals will not show any clinical signs at all and disease is uncommon in healthy, unstressed adult birds and mammals. In mammals, clinical disease is most common in very young, pregnant or lactating animals, and often occurs after a stressful event. Outbreaks in young ruminants, pigs and poultry can result in a high morbidity rate, and sometimes, a high mortality rate. In outbreaks of septicaemia, the morbidity and mortality rates may approach 100%. There are reports of domestic cats suffering gastroenteritis with the *S. typhimurium* phage types that affect garden birds. This is thought to occur when cats predate sick passerine prey.

Effect on humans

Salmonellosis is common in humans and is a major cause of food-borne illness throughout the world. Most people recover from infection without treatment. Infection often causes gastroenteritis but a wide range of clinical signs may be seen and death can occur in severe cases. The incidence and severity of the disease is higher in younger children, the elderly and those with weakened immune systems. The overall mortality rate for most forms of salmonellosis is less than 1%.

Economic importance

There is potential for significant economic losses to the livestock industry, with ruminants, pigs and poultry particularly affected, due to illness and loss of infected animals and likely trade restrictions imposed during and after an outbreak.

Illness in humans can result in significant economic losses due to the time lost from normal activities and medical costs incurred.

FURTHER INFORMATION

Useful publications and websites

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- Wildpro. **Salmonellosis**. http://wildpro.twycrosszoo.org/S/00dis/Bacterial/Salmonellosis.htm [Accessed March 2012].

Contacts

- **WHO Communicable Diseases Surveillance and Response (CSR)**. zoonotic_alert@who.int fmeslin@who.int and outbreak@who.int.
- **FAO Animal Production and Health Division**. www.fao.org/ag/againfo/home/en/who.htm.

Schistosomiasis

Wetlands supporting groups of susceptible animals and freshwater snails

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Bilharzia, blood flukes, Katayama fever, snail fever, swimmer's itch

KEY FACTS

What is schistosomiasis?

Also known as bilharzia, schistosomiasis is a disease caused by trematode worms which inhabit the blood circulatory system of their host. The worms require freshwater snails as an intermediate host to develop infectious larvae that penetrate the skin of a wide range of animal hosts following contact with infested water bodies. Infected animals pass worm eggs out in their urine or faeces which, if in contact with freshwater, hatch out and infect freshwater snails, producing another larval stage which is infective to the final animal host thus completing the life cycle.

Eighty-five percent of the 207 million people who are infected with schistosomiasis worldwide live in developing African countries. Poor sanitation greatly increases prevalence and severity.

Causal agent

Parasitic flatworms called blood flukes of the genera *Schistosoma* and *Orientobilharzia.* Many domestic farm animals and birds have their own species-specific schistosomes, each with varying impacts on health and subsequent economic importance.

Species affected

Schistosomes have a broad host range encompassing many species of wild animals including waterbirds, however, humans and livestock aremost at risk of clinical disease.

In Africa, cattle, sheep and goats are infected by three species (*S. mattheei*, *S. bovis* and *S. curassoni*). Schistosomes are also prevalent in wild mammals, including: antelope (*S. margrebowiei*), zebra, bushbuck and rodents (*S. rodhaini)* in tropical areas. In addition to humans, *S. mansoni* also infects rodents, baboons and some insectivores.

In Asia, 40 different species of wild and domestic animals are known to be infected by *S. japonicum* including pigs, dogs, cats, rodents, monkeys, oxen and water buffalo (*Bubalus bubalis*). *S. japonicum* also infects humans and animal hosts are likely to act as a reservoir for human infection. *S. indicum* occurs in the Indian subcontinent infecting horses, buffalo, sheep, goats and camels.

Humans are infected by three main species: *Schistosoma haematobium* (Africa)*, S. mansoni* (Africa and South America) and *S. japonicum* (Asia). Locally important species include *S. mekongi* and *S. intercalatum*, which are localised to parts of Cambodia and Laos, and central and west Africa, respectively.

Geographic distribution

- Africa: all freshwater in southern and sub-Saharan Africa, including the great lakes and rivers as well as smaller bodies of water, is considered to present a risk of schistosomiasis transmission. Transmission also occurs in the Nile River valley in Egypt.
- South America: including Brazil, Suriname and Venezuela.
- Caribbean: Antigua, Dominican Republic, Guadeloupe, Martinique, Montserrat, Saint Lucia (lower risk).
- The Middle East: Iran, Iraq, Saudi Arabia, Yemen.

- Southern China.
- South East Asia: Philippines, Laos, Cambodia, central Indonesia, Mekong delta.

Environment	Freshwater, particularly associated with irrigation schemes, reservoirs and water holes. Parasite distribution is dependent on habitats suitable for the snail intermediate host which range from still to slow-moving water.

TRANSMISSION AND SPREAD

Vector(s)	Intermediate hosts include freshwater snails mainly of the genera *Bulinus, Biomphalaria* and *Oncomelania.*
How is the disease transmitted to animals?	Eggs laid by mature flukes in the blood vessels surrounding the gut and the bladder of the host are eventually passed in faeces and urine. When the eggs reach freshwater they hatch into infectious free-living miracidia and infect only suitable snail vectors. Within the snail, the parasite propogates by asexual reproduction and several thousand free-swimming larvae, known as cercariaeare, are released and remain infectious to the final animal host for up to 48 hours.
How does the disease spread between groups of animals?	Eggs shed in the faeces and urine of infected animals and humans contaminate water sources inhabited by snail intermediate hosts, which in turn are shared by different animal groups. Risk of infection is exacerbated by increased host density and by the wide definitive host range of schistosome species. As an example, hosts of *S. japonicum* in Asia include dogs, cats, rodents, pigs, horse, goats, water buffalo, cattle and humans.
How is the disease transmitted to humans?	In contaminated freshwater bodies, infective schistosome cercariae penetrate the skin. Schistosome infections are maintained by a range of mammals, however, field transmission is increased when water sources such as dams and irrigation ditches are shared with infected human populations (*e.g. S. mansoni* in Africa). Herein lies the potential for a human settlement with poor sanitation to significantly impact on the health of surrounding livestock and wildlife. Human population displacement and refugee movements can introduce the disease to new areas (*e.g.* Somalia and Djibouti). Schistosomes which only infect domesticated ruminants (*e.g. S. mattheei*, *S. bovis*, *S. curassoni*) or waterbirds (*e.g. Heterobilharzia americana*, *Orientobilharzia turkestanica*, and *O. turkenstanicum*) may be present in water bodies near human settlements. The infective cercariae of these non-human species can penetrate the skin of humans but rarely develop further. A condition known as 'swimmer's itch' may develop from these infections.

IDENTIFICATION AND RESPONSE

Field signs	In ruminants symptoms may include haemorrhagic enteritis, anaemia and emaciation due to mechanical damage of blood vessels by the spiked eggs of schistosomes. Severely affected animals usually die within a month or two of infection. Older cattle may develop immunity in areas where the disease is endemic. In humans, there are no symptoms when first infected. Skin irritation or a rash may develop after a few days. After 1-2 months, fever, chills, cough and muscle aches may occur. Intestinal schistosomiasis can result in abdominal

pain, diahorroea and blood in the stool. Urogenital schistosomiasis is associated with blood in the urine.

The infectious larval stages of some 'animal' *Schistosoma spp*. in either tropical or temperate countries may penetrate the skin of humans and cause an allergic reaction known as 'swimmers itch'. 'Swimmers itch' may develop in approximately one third of those infected, however, the larval worms die in the skin and cannot migrate or mature in infected humans.

Recommended action if suspected

Contact and seek assistance from human and animal health professionals immediately if there is suspected infection in people and/or livestock. The disease is not notifiable.

Diagnosis

Diagnosis is based on identification of characteristic schistosome eggs by microscopic examination of faeces and urine samples, or biopsy specimens. Serological tests may be sensitive and specific but do not provide information about the size of worm burden or clinical status.

PREVENTION AND CONTROL IN WETLANDS

Environment

Adult schistosomes have a high degree of fecundity as the infective cercariae are sensitive to dessication and have an average life span of 48 hours. In areas where mammalian host density is low, this high fecundity enables the parasite to maintain a low level population without causing disease in humans or livestock. In environments where water sources supporting populations of susceptible snails are contaminated with high levels of infected human and livestock excreta, rates of transmission will also rise along with the probability and severity of disease.

Control measures should therefore focus on **preventing contamination of water sources** through improved sanitation, as well as public health education, large scale medical treatment of infected individuals [► Humans], ring-fencing contaminated water bodies and reducing snail populations.

Reduce snail populations
► Section 3.4.3. Vector control - snail control

Strategies should be implemented with specific knowledge of the ecology of the causative snail. Water impoundments of all shapes and sizes (*e.g.* irrigation systems, lakes and dams) provide fertile breeding grounds and good habitat for freshwater snails and encourage close and frequent contact between people and infected water. The following habitat alterations may help reduce snail populations.

Alter flow rate and water levels to disturb snail habitats and their food sources:

- Include V-shaped banks in irrigation channels.
- Remove vegetation/silt in channels to avoid a drop in velocity which may lead to further vegetation growth and good habitat for snails. Note that personnel involved in the manual removal of vegetation are increasing their exposure to snails. Frequent removal may be needed.
- Flow rate should only be addressed with knowledge of the ecology of the snail in question *e.g.* for *Biomphalaria* and *Bulinus* flows greater than 0.3 m/sec would suffice but most snails can withstand flows up to 0.5 m/sec.

- Borrow-pits, small pools and ponds serving no special purpose (for humans, wildlife or livestock) may be drained to eliminate breeding sites.

Expose snail habitat:

- Remove littoral vegetation from the sides of canals feeding irrigation projects to expose snail habitat. Heavy rain can also cause removal.
- Thought should be given to downstream conditions and the potential for the liberated snails to recolonise new habitat.
- Where possible dry out littoral zones to strand snail populations, however take into account the specific ecology and the resilience of the target species.

Chemical control:

- Use of molluscicides may cause environmental damage and should be avoided. Use should be targeted rather than wide-spread. Applications are usually restricted to places frequently used by people for swimming, bathing *etc.*

Biological control of intermediate snail hosts using larger, more voracious aquatic snails which do not harbour schistosome infection and out-compete local snails, has also been successful but should only be used after expert consultation due to their effects on local biodiversity.

Livestock

Prevention of contamination of wetland habitat with livestock excreta should be the main priority. This is especially important for schistosome species such as *S. japonicum* which parasitises wild animal, livestock and human hosts.

To reduce the risk of infection, **susceptible livestock should be removed** from wetlands and replaced with non-susceptible species (or by farm machinery if the purpose of livestock is mechanical management).

Agricultural run-off must be prevented from contaminating water bodies.

Infected and susceptible livestock should be treated with **flukicides** such as praziquantel. However, re-infection may occur quickly if the source of contamination is left uncontrolled.

Wildlife

High density populations of susceptible wildlife increase the potential for disease transmission. Interaction between livestock and wildlife should be prevented wherever possible and **supplementary feeding** of wild animals close to water sources should also be avoided.

Humans

The following practices may help reduce the likelihood of infection in humans:

- Avoiding contact with snail-infested waters and using water supplied from covered pipes or pit-wells.
- Avoiding swimming, wading, washing or bathing in water suspected of infestation. It is safest to consider all freshwater bodies in endemic areas as potential transmission sites if sites are otherwise unidentified.
- For agricultural workers at constant risk of infection, periodic examination and treatment may be the most feasible approach to disease control.

- Ensuring good sanitary practices. A clean water supply and improved sanitation (including for people onboard boats) must be provided to stop human excrement entering wetlands.

Treat infected individuals
Anthelmintics such as praziquantel and oxamniquine (for *S. mansoni*) are effective treatments for schistosomiasis. If the local economic situation allows, consider mass treatment programmes for non-infected individuals following episodes of flooding. It is important that anthelmintic treatment be applied in conjunction with sanitation improvements to prevent widespread re-infection and subsequent cycles of treatment/re-infection thus increasing the potential for drug resistance to develop. Schistosomes contain cross-reacting antigens and vaccine development programmes are currently in progress. Frequent exposure of humans to schistosomes of domesticated animals can impart a degree of immunity to disease-causing species.

Public health education
Many countries and regions may lack funds for public education especially to isolated human settlements. However, an informed public are able to make personal decisions over their contact and use of standing water and thus reduce the risk of infection to themselves and their livestock.

IMPORTANCE

Effect on wildlife

In general this disease has a subclinical impact on wildlife. Problems may arise in areas where wildlife mixes with high density livestock and/or human populations.

Effect on livestock

An estimated 165 million animals are infected in Africa and Asia. In these regions most infections are subclinical but, depending on the schistosome species, can still cause serious morbidity and mortality (*e.g. S. japonicum* in Asian cattle and goats). ► **Economic importance**

Effect on humans

Because of considerable economic and health impacts, schistosomiasis is considered the second most important parasitic disease after malaria. Worldwide, 207 million people are infected with schistosomiasis and it is especially important because of its prevalence in children and capacity to hinder growth and learning. Chronic schistosomiasis is debilitating and can affect people's abilty to work. In sub-Saharan Africa over 200,000 people die of the disease every year. ► **Economic importance**

Economic importance

Farmers suffer significant economic losses due to schistosome burdens in livestock, productivity is reduced whilst susceptibilty to other environmental stresses is increased (particularly *S. bovis* in African cattle and *S. mattheei* in sheep). Similarly, schistosomiasis impacts on economic development in developing countries by reducing the productivity of human workforces. Eradication programmes including widespread administration of praziquantel and implementation of improved sanitation are costly and beyond the means of many developing nations.

FURTHER INFORMATION

Useful publications and websites

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Tick-borne diseases (TBDs)

Wetlands inhabited by ticks and susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: TBDs include: African swine fever, anaplasmosis, babesiosis, Crimean-Congo haemorrhagic fever, ehrlichiosis, equine piroplasmosis, heartwater (or Cowdriosis), louping ill, Lyme disease, Nairobi sheep disease/Ganjam virus, Powassan encephalitis virus, rickettsiosis (including Q fever & Rocky Mountain spotted fever), theileriosis (including East Coast fever & tropical theileriosis), tick-borne encephalitis (TBE), tick-paralysis, tick-borne relapsing fever, tularemia.

KEY FACTS

What are tick-borne diseases?

Tick-borne diseases (TBDs) encompass a wide range of disease-causing pathogens that all have a tick vector. These include bacterial (*e.g.* heartwater and Lyme disease), protozoan (*e.g.* theileriosis) and viral diseases (*e.g.* TBE), which are maintained and transmitted by ticks to numerous wild and domestic animal hosts.

Ticks are among the most important arthropod vectors of disease. These blood-feeding ectoparasites are found in almost every region of the world, typically in grassy, wooded habitat. They can act as vectors and/or reservoirs of disease, transmitting pathogens from an infected vertebrate to another susceptible animal, or human, whilst feeding.

There are two major tick families: the Argasidae (soft ticks) and the Ixodidae (hard ticks), the latter (Ixodidae) having a number of attributes that enhance their potential to transmit disease, including long feeding durations (often days), firm attachment whilst feeding, a usually painless bite and the utilisation of a variety of hosts.

Aside from disease transmission, ticks are also responsible for severe toxic conditions (tick paralysis or toxicosis), irritation, secondary infections and physical damage associated with their bites.

Causal agents

A wide variety of pathogens (including bacteria, viruses and protozoa) are harboured and transmitted by ticks. Salivary neurotoxins, produced by some tick species, are the causal agents of tick paralysis.

Species affected

TBDs affect a wide variety of vertebrate species including domestic animals, wildlife and humans.

Geographic distribution

TBDs occur worldwide as their tick vectors also have a global distribution. Most individual TBDs are geographically localised, occurring in foci with favourable conditions for the ticks and animal hosts involved in the transmission of the pathogen.

Environment

Each tick species is well adapted to its habitat, environment and host. Depending on the species of tick, they are mostly found in deciduous woodland, coniferous forest, wetland and meadows.

Areas with leaf litter, weeds, long grass or brush often have higher densities of ticks as this vegetation is used by most species (hard and some soft ticks) to ‘quest’ for a suitable host animal. When questing, a tick climbs vegetation, extends its first pair of legs and uses them to grasp a host when it passes. Conversely, most soft ticks inhabit environments commonly used by potential hosts (*e.g.* bedding or cracks in dens, stables or caves) and often feed when the host animal sleeps.

TRANSMISSION AND SPREAD

Vector

Ticks of the Argasidae (soft ticks) and Ixodidae (hard ticks). An estimated 10% of the currently known 867 tick species act as vectors of diseases of domestic animals and humans.

A tick species is only considered as a vector for a pathogen if it:

- feeds on an infectious vertebrate host;
- acquires the pathogen during the blood meal;
- maintains the pathogen through one or more life stages; and
- transmits the pathogen on to other hosts when feeding again.

How is the disease transmitted to animals?

TBDs are transmitted to animals when an infected tick feeds on a susceptible animal. Usually, a pathogen must infect and multiply within a tick before the tick is able to transmit disease to a host *via* its salivary glands and mouthparts (hypostome).

Ticks become infected with pathogens by:

- **feeding** on an infected animal host
- **transstadial transmission**
  Pathogen passed through tick life stages (*i.e.* from larvae to nymph to adult)
- **transovarial transmission**
  Pathogen passed from parent tick to offspring *via* the female ovaries (increasing vector potential by several thousand times).

Ticks are often a robust and long-lasting reservoir of infection. For example, they can remain infected with *Ehrlichia ruminantium* (the causative agent of heartwater) for at least 15 months and can harbour the pathogen responsible for theileriosis for up to two years.

Pathogens harboured in a tick are transmitted to an animal host through salivary secretions, regurgitations or tick faeces when the ectoparasite feeds. The likelihood of disease transmission increases with tick attachment time.

Some TBDs (*e.g.* TBE) can also be transmitted between ticks co-feeding on a host, without that host becoming systemically infected. This is important for the epidemiology and has implications for disease surveillance.

Infrequently, some TBDs are transmitted indirectly *via* fomites and mechanical vectors contaminated by infected blood or plasma.

How does the disease spread between groups of animals?

The spread of TBDs requires the dispersal of the **tick vectors** and/or the **reservoir hosts**. For a TBD to spread to a *new* area, the vector ticks or reservoir hosts must find respective hosts or ticks that are susceptible to infection and can maintain the pathogen.

TBDs may be dispersed by:

- **Tick movement:** ticks may walk short distances (seldom exceeding 50m).
- **Hosts:** whilst attached to a host, ticks may travel larger distances (particularly in the case of migratory animals).
- **Anthropogenic activity**
  - Movement and trade of livestock (infected with TBD or tick-carrying)
  - Changes in agricultural practices
  - Tick-habitat modification.
  -

How is the disease transmitted to humans?	Direct routes, as with animals, involve humans being bitten by disease-transmitting ticks. Indirect routes of transmission are also possible, such as contamination of cuts or the eyes following crushing of ticks with the fingers.

IDENTIFICATION AND RESPONSE

Field signs

Due to the wide range of pathogens transmitted by ticks, there are no signs specific for TBDs. Signs can include: fever, diarrhoea or incontinence, lack of appetite and weight loss, weakness, lethargy, muscle and/or joint pain (reduced mobility), neurological signs (convulsions, head pressing *etc.*), anaemia (weakness, paleness of gums and mouth), discharge from the eyes or nose, or jaundice (yellowing of skin and eyes).

Infected animals may not have all of the signs, and many are associated with other diseases. The development and severity of TBD will depend on numerous factors (host susceptibility, agent virulence and infective dose).

Important TBDs of domestic animals, include:

- **Bovine babesiosis** (Redwater disease).
  Fever, weight loss, anaemia, jaundice, depressed or unusual behaviour, occasional muscle tremors and convulsions, red-coloured urine.
- **Heartwater** (Cowdriosis).
  Fever, loss of appetite, listlessness, shortness of breath, purple spots (petechiae) on mucous membranes, occasional diarrhoea (particularly in cattle), high-stepping gait, unusual behaviour, convulsions and frothing at the mouth. Death usually occurs within a week of infection.
- **Anaplasmosis** (Gall sickness).
  Fever, anaemia, jaundice, weakness, loss of appetite and co-ordination, shortness of breath, constipation, death (mortality is usually between 5-40% but can reach 70% in a severe outbreak). Pregnant cattle may abort.
- **Theileriosis** (including East Coast Fever and Tropical Theileriosis)**.**
  Swelling of the lymph nodes, high fever, shortness of breath and high mortality (can be up to 100% in susceptible cattle). Tropical theileriosis may additionally present with jaundice, anaemia and bloody diarrhoea.
- **Equine piroplasmosis.**
  High fever, reduced appetite, congestion of mucous membranes, dark red urine.
- **African swine fever.**
  Fever, anorexia, reddening of skin, cyanosis, vomiting and diarrhoea, abortion, or sudden death.

Many TBDs may cause little or no detectable disease in the reservoir host (*e.g.* African swine fever in wild African suids). This can be significant for zoonotic diseases such as TBE (reservoir hosts include forest rodents), where human cases can occur without detectable disease in wild or domestic animals.

Recommended action if suspected	Seek advice from animal health professionals. Many TBDs are listed as notifiable by OIE and suspected or confirmed cases must be reported to local and national authorities and the OIE.
Diagnosis	Ticks can carry more than one pathogen, which can make diagnosis of a TBD difficult. For a definitive diagnosis of a TBD, laboratory confirmation is required.

National laboratories will provide guidance on the samples that are required, which often include: tissue (brain, lymph node), whole blood, serum and ticks.

Some tick-borne pathogens may be directly observed by the microscopic examination of stained tissue and/or blood samples. Abnormal blood test results in TBD cases may include low platelet count, low serum sodium levels, abnormal white blood cell counts or elevated liver enzyme levels.

Serological assays (including indirect immunofluorescence assay (IFA), ELISA or EIA, latex agglutination and dot immunoassays) are often used to aid in the diagnosis of a TBD and molecular methods such as PCR can be used for rapid detection.

For more detailed information regarding laboratory diagnostic methodologies, refer to the latest edition of the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals.

PREVENTION AND CONTROL IN WETLANDS

Environment

A well planned and thorough monitoring programme should form the basis of integrated tick control. A number of tick survey methods may be implemented to monitor tick densities. These include: tick walks and drags, carbon dioxide trapping, tick flags and host trapping and examination.

Habitat modification.
The free-living stages of most tick species are often restricted to specific conditions within the ecosystems inhabited by their hosts. **Reduction** of **leaf-litter** and **understory vegetation** will remove tick microhabitats and reduce the abundance of ticks.

The removal of the structural vegetation used by ticks to quest (*i.e*. weeds, high grass and brush) has also proved a successful method of tick-control in recreational areas. **Controlled burning** of habitat has been shown to reduce tick numbers for up to a year, yet the long-term impacts of burning on tick populations are unclear.

Avoiding areas with large populations of ticks can be used to reduce TBDs where possible (*e.g.* select grazing areas for domestic animals).

Biological Control.
Predators naturally control tick numbers in some areas of the world and habitat modification to encourage tick predators may provide a method of free-living tick control. However, most tick predators are generalists with a limited potential for tick control. Some wasp species parasitise and kill ticks, but are not thought to reduce tick numbers significantly (although inundative releases have shown potential value). Research has suggested several species of bacteria, entomopathogenic fungi and nematodes that are pathogenic to ticks and may have potential as biocontrol agents.

Chemical control.
Control of ticks with an appropriate acaricide is a widely used method to control TBDs. Acaricides have been used against free-living ticks in the environment by treating vegetation at specific sites (*e.g.* along paths or animal trails). This method is not recommended for wider use due to the environmental implications and the cost of treating large areas. However, the free-living stages of soft ticks are more frequently and effectively treated with acaricides, as they are usually found in specific foci (*i.e.* animal holding pens, livestock runs, poultry housing and in human dwellings).

*The **environmental consequences** of undertaking any form of habitat modification must be carefully evaluated **before** being implemented as a method to control tick populations.*

For further information ►**Section 3.4.2. Control of Vectors.**

Livestock

The **exposure** of livestock to ticks may be reduced by the use of **repellents**, **acaricides** and regular **inspections** of premises and animals. A variety of tick control programmes may be integrated into livestock management:

Chemical control.
Tick control in livestock is most commonly achieved by **acaricide** treatment. Acaricides are most effectively applied through total immersion of livestock in a dip-vat. They may also be applied as sprays, dusts, pour-ons, spot-ons and more recently *via* slow release technologies such as impregnated ear tags, or systemically from implants or boluses. Fowl are usually treated with a dust application. The frequency of acaricide treatment depends on the targeted tick species, the TBD present and the livestock-management practices followed. Treatment may vary from every three days (as followed in east Africa for the protection of cattle against East Coast fever transmission by *Rhipicephalus appendiculatus*) to every six months (for the control of *Rhipicephalus* (formerly *Boophilus*) *microplus* tick populations).

Organochlorines, organophosphates, carbamates, amidines, avermectins and pyrethroids have been used for tick control. The development of acaricide resistance in ticks has necessitated the development of new compounds, such as phenylpyrazoles.

Acaricide usage is not considered sustainable as they are expensive, can cause environmental damage, may leave potentially harmful residues in meat and milk and ticks can develop resistance over time. More sustainable methods for the control of some TBDs may involve a combination of strategic tick control and vaccination, however, these are yet to be successfully applied on a large scale in endemic areas.

N.B. Tick eradication with acaricide is not recommended in some situations. Where TBDs are endemic, it may be preferable to allow tick populations to remain at high levels. This permits the re-infection of immune livestock, boosting immunity and leading to endemic stability.

Tick-resistant livestock.
Zebu (*Bos indicus*) and Sanga (*B. indicus* crossed with *B. taurus*) are indigenous cattle breeds of Asia and Africa which are very resistant to hard ticks after initial exposure. Conversely, European cattle (*B. taurus*) usually remain susceptible. Tick-resistant cattle and their cross breeds may be exploited as a method to control the parasitic stages of ticks. Although these breeds continue to support tick populations, they are not conducive to large tick infestations. The use of Zebu cattle has been successful in Australia and the introduction of tick-resistant cattle is becoming an increasingly important method of tick control in the Americas and Africa.

Pasture spelling.
Pasture rotation or pasture spelling can be used as a method to control one-host tick species (such as *Rhipicephalus microplus*, an economically important parasite of livestock that spreads the pathogens responsible for babesiosis). Larval ticks are starved due to the absence of their host, so the duration of pasture spelling is determined by the lifespan of the free-living larvae. This

technique requires the existence of well maintained pasture boundaries and the absence of suitable alternate hosts. Pasture-vacation schedules must be rigidly followed. This method has minimal application to soft ticks (nymphs can survive for long periods without food) and multi-host tick species.

Vaccines.
TBD control in livestock may also be achieved by the use of live, attenuated vaccines. Notable vaccination programmes include the development of an East Coast fever vaccine in Kenya and the implementation of a vaccine for tick fever in Australia. Furthermore, live attenuated vaccines have been used to control tropical theileriosis (caused by *Theileria annulata*) and heartwater (caused by *Ehrlichia*, formerly *Cowdria ruminantium*).

A potential alternative is to vaccinate against the tick species itself. Recently, a vaccine against *R. microplus* has been developed that stimulates the host production of an antibody which damages tick gut cells, causing tick mortality or reduced reproductive potential. One-host ticks such as *R. microplus* are good candidates for livestock vaccines, yet vaccine development for multi-host ticks, which infest both cattle and wild ungulate species, may not be feasible.

Quarantine.
The control of livestock movements through quarantine can help control TBD spread. In all tick-borne disease-free areas or countries, it is recommended that livestock are inspected for ticks before allowing entry. Area quarantine, on areas with large infestations, ensures all livestock are inspected for ticks and given precautionary treatment before leaving. Premises quarantines act to prevent the spread of infested livestock from individual pastures, farms or ranches with suitable physical barriers.

Antibiotics.
Livestock moved into endemic areas of TBDs may be protected from bacterial disease by prophylactic treatment with broad-spectrum antibiotics. Antibiotic administration can also be effective for the treatment of bacterial TBDs in their early stages and the secondary infection of lick lesions.

Manual tick removal may also provide an effective control method for small numbers of animals.

Wildlife

Control of wildlife populations may be difficult, but the interaction of livestock and wildlife should be prevented where possible. This will minimise the transmission of TBDs and ticks to and from susceptible wild animals.

Humans

Reducing exposure to ticks is the best method to prevent TBDs which affect humans.

Avoid and repel ticks:

- Walk in the centre of trails to avoid contact with overgrown vegetation.
- Where possible avoid tick habitat, especially during peak tick seasons.
- Wear clothing to cover arms, legs and feet whenever outdoors.
- Apply repellents containing DEET (20% or more) to exposed skin and clothing.

Find ticks:

- Wear light-coloured clothing to enable ticks to be observed easily.
- Check yourself, your children and gear thoroughly for ticks after being outdoors.
- Companion animals should be routinely checked for ticks; cats and dogs can be treated with commercially available acaricide dusts or washes.

Remove ticks:

- Using tweezers, grasp the tick as close to the skin as possible.
- Pull the tick out it one, steady movement. Do **not** twist or jerk.
- Wash hands and disinfect the bite. Freeze tick, if possible, to aid with the identification of a TBD if symptoms develop.
- If a rash, flu-like symptoms or other illness develop, seek medical advice.

► Section 3.4.3. Control of vectors: tick control

Educational talks and **informative material** (such as brochures and pamphlets) can also help reduce the likelihood of tick bites and zoonotic disease transmission, especially for high-risk employees such as reserve wardens. **Signage**, warning people they are entering tick-infested areas, may also help reduce the incidence of tick bites.

Treatment

Seek advice from medical health professionals. Early diagnosis is essential. Antibiotic treatment is indicated in cases of clinical bacterial TBDs such as anaplasmosis, Lyme disease, tularemia, Rocky Mountain spotted fever, and ehrlichiosis.

IMPORTANCE

Effect on wildlife

Ticks and TBDs have co-evolved with numerous wild animal hosts, often living in a state of equilibrium with little detectable clinical disease. Where TBDs emerge in new areas or naïve species, wildlife can be clinically affected (*e.g.* African swine fever in European wild boar *Sus scrofa*).

Effect on livestock

The multiple TBDs can cause a wide range of clinical syndromes leading to variable morbidity and mortality**.** Major TBDs of livestock include bovine babesiosis, bovine anaplasmosis, theilerioses and heartwater, African swine fever, louping ill and equine piroplasmosis. In addition to other diseases, these TBDs can result in mass herd die-offs and cause severe losses to the livestock industry.

Livestock may also suffer direct impacts from feeding ticks:

- Tick paralysis and toxicosis.
- Discomfort and irritation, leading to production losses (milk and weight gain).
- Blood loss, resulting in reduced live weight and anaemia.
- Damage to hides.
- Reduced suckling efficiency due to scarring on udders and teats.
- Formation of lesions susceptible to secondary infections.

Effect on humans

Ticks and TBDs constitute a serious public health threat, particularly in the northern hemisphere. Lyme disease is the most frequently reported zoonotic tick-borne disease globally and viral TBDs, characterised by haemorrhagic fevers and encephalitis, cause the highest morbidity and mortality in humans of the tick-borne zoonoses.

TBD resulting in livestock mortality affects humans indirectly, due to the reduction in animal protein available for human consumption.

Economic importance

TBDs are responsible for severe economic losses worldwide, primarily due to their impacts on livestock production and human health.
TBDs are a significant impediment of export, trade and the development of livestock production. TBDs affecting companion animals are only of economic significance in industrialised countries and TBDs of equines constitute important constraints to international trade and sporting events involving these animals.

FURTHER INFORMATION

Useful publications and websites

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Contacts

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Trematode infections of fish

Wetlands supporting susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

Synonyms: Dicrocoeliasis, fascioliasis, foodborne trematode (FBT) infections, fishborne parasitic zoonoses, helminth infection, paramphistomiasis.

KEY FACTS

What are trematode infections of fish?

Trematodes are a group of flatworms (or flukes) that parasitise members of all vertebrate classes but most commonly fish, frogs and turtles; they also parasitise humans, domestic animals and invertebrates such as molluscs and crustaceans. Some are external parasites (ectoparasites); some attach themselves to internal organs (endoparasites); others are semi-external, attaching themselves to the lining of the mouth, to the gills or to the cloaca. Some attack a single host, whilst others require two or more hosts. Some species are zoonotic, causing lung, liver and intestinal fluke diseases in humans, and trematodes have been reported to affect the health of more than 40 million people throughout the world.

The principal human diseases are: (i) trematodiasis (*e.g.* liver fluke diseases such as clonorchiasis, opisthorchiasis and metorchiasis; lung fluke disease such as paragonimiasis; and intestinal trematodiases such as heterophyiases and echinostomiases); (ii) nematodiases (*e.g.* capillariasis, gnathostomiasis, anisakiasis); and (iii) cestodiases (*e.g.* diphyllobothriasis). The trematodiasis group are considered as some of the most medically important parasitic zoonoses where a large number of fish species, both marine and freshwater, are potential sources of infection. Some trematodes are potentially pathogenic and the main pathway for human infection is through consumption of raw or inadequately cooked fish.

Causal agent

Clonorchiasis is caused by *Clonorchis sinensis* (Chinese liver fluke); opisthorchiasis is caused by two species: *Opisthorchis viverrini* (Southeast Asian liver fluke) and *O. felinius* (cat liver fluke), and metorchiasis is caused by *Metorchis conjunctus* (Canadian liver fluke). Infections of the bile duct, gall bladder and pancreas (*e.g.* cholangitis, choledocholithiasis, pancreatitis and cholangiocarcinoma) are the major clinical problems associated with the chronic pattern of these liver fluke infections. They belong to Phylum Platyhelminthes, Class Trematoda and Family Opisthorchidae.

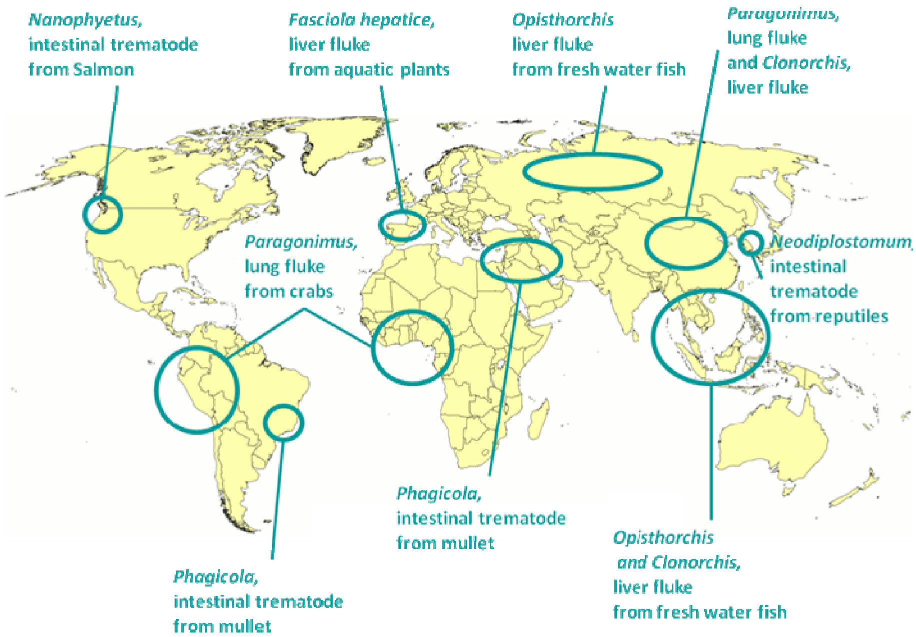
Intestinal trematodiases are caused by intestinal trematode parasites belonging to the families Heterophyidiae and Echinostomatidae and several genera such as *Metagonimus*, *Heterophyes* and *Haplorchis*.

Schistosome species that cause bloodfluke infections are mainly restricted to the tropical and subtropical areas and belong to the genera *Schistosoma* and *Orientobilharzia*. These include both zoonotic and non-zoonotic species and typically occur in cattle, buffaloes, goats and pigs.

A large number of gastro-intestinal trematode species (paramphistomes) have been described. They are usually thick, short (4-12 mm), fleshy, maggot-like worms. They can infect all ruminants but young calves and lambs are the most susceptible. Not all species are pathogenic but clinical outbreaks of paramphistomiasis have been caused by *Paramphistomum microbothrium* (Africa), *Cotylophoron cotylophorum* (Asia), *P. ichikawar, C. calicophorum* (Australasia) and *P. cervi* (Europe).

Species affected Freshwater snails (Phylum Mollusca; Class Gastropoda) and various fish are intermediate (in some cases definitive) hosts, and human and other vertebrates such as wild animals, livestock (sheep, cattle, goats and pigs) and fowl are usually definitive hosts.

Geographic distribution Trematode infections have a worldwide distribution but are not notifiable OIE-listed diseases. Trematode infections are reported to affect the health of more than 40 million people throughout the world and are particularly prevalent in South East Asia and Western Pacific Regions.



Geographic distribution of fish-borne trematode infections

Environment Trematodes have complex life cycles and part of the life cycle takes place in water (freshwater to marine water depending on the species) in both tropical and temperate zones. Habitats of secondary intermediate hosts include freshwater habitats with stagnant or slow-moving water (ponds, rivers, aquaculture, swamps and rice fields).

TRANSMISSION AND SPREAD

Vector(s) Most trematodes have a lifecycle in which larval stages parasitise one or more species that are different from the host of the adults. Infective larval stages of the parasites include miracidium, redia, cercaria and metacercaria. The vectors include molluscs (*e.g.* snails), fish, crustaceans (*e.g.* crayfish and crabs), herpetafauna (*e.g.* frogs and snakes), terrestrial arthropods (*e.g.* ants), wild and farmed animals (*e.g.* sheep, cattle, goats, pigs, cervids and fowl).

How is the disease transmitted to animals? The transmission mechanisms of zoonotic trematodes are generally the same, *e.g. C. sinensis* is transmitted through ingestion of trematode eggs by the intermediate host (*i.e.* snail), followed by a free-swimming cercariae encysted stage that adheres to the skin of the host fish.

How does the disease spread between groups of animals? Some species attack a single host, whilst others require two or more hosts, but the mechanism of spread between groups of animals is essentially the same. Embryonated eggs are discharged in the biliary ducts and through the faeces and ingested by a suitable snail intermediate host where they undergo several developmental stages (sporocysts, rediae and cercariae). The cercariae are released from the snail and after a short period in a free-swimming stage in the water, they come into contact with a suitable fish where they encyst in the flesh as metacercariae.

How is the disease transmitted to humans?

The mode of transmission to the definitive host is through consumption of raw, undercooked, or improperly pickled or smoked infected fish. Major dietary sources of infection in Asia include the following examples: for *C. sinensis* - (i) morning congee with slices of raw freshwater fish (southern China, Hong Kong SAR) or slices of raw freshwater fish with red pepper sauce (Korea); (ii) half roasted or undercooked fish (China); (iii) raw shrimps (China). For *O. viverrini* – (i) raw fish dishes called 'Koi pla', 'Pla ra', 'Pla som', *etc*.. Men in the 25-55 year age group are a highly affected group; a contributing factor for this is the practice of men eating raw or pickled fish (usually accompanied with alcohol).

IDENTIFICATION AND RESPONSE

Field signs

As many trematodes are endoparasites, it is difficult to diagnose the infection based on gross external examination of the fish. However, heavy infestation can lead to retarded growth.

Recommended action if suspected

Notification is not mandatory since these diseases are not listed as notifiable by the OIE. However, as infections are a serious concern for public health, the recommendations listed in the next section should be adhered to in order to protect the health of households and the local communities in general. Metacercariae can persist in the fish muscle for a considerable time (*e.g.* for weeks in dried fish, a few hours in salted or pickled products) but they may be killed by adequate cooking.

Diagnosis

Parasitological examination, using a microscope to observe the eggs, is one of the reliable techniques used to demonstrate infection; however, this requires well-trained laboratory staff.

Several different diagnostic techniques are available for animals, such as a pepsin digestion method to induce the release of metacercariae from infected animals. The selection of particular techniques is determined by the available resources, the type of animal/products to be analysed, the organ suspected to be infected, the training and experience of the inspector and the degree of certainty required by any inspections.

PREVENTION AND CONTROL IN WETLANDS

Environment

It is important to ensure that proper hygiene measures are followed to prevent human waste entering, and contaminating, the environment.

Aquaculture

Actions should be directed, firstly, at prevention of the disease in the fish population. Basic farm biosecurity such as good farm hygiene and good husbandry practices, good water quality management, proper handling of fish to avoid stress, regular monitoring of health status, good record keeping (gross and environmental observations and stocking records including movement records of fish in and out of aquaculture facilities). Following these good general practices helps maintain healthy fish.

Use of a hazard analysis and critical control points (HACCP) approach to fish pond management focussing on water supply, fish fry, fish feed and pond conditions will help to eliminate contamination of ponds with parasite eggs and snail vectors.

Irradiation of fish to control infectivity of metacercariae may be considered but economic cost and consumer acceptance may be limiting factors.

A number of farm management measures can minimise or prevent the spread of trematode infections. These include:

- Control of molluscan intermediate hosts can be carried out through: responsible use of chemical molluscicides, environmental manipulation (*e.g.* 'weed' control) and the use of molluscophagous fish.
- Design the farm in such a way that contamination with human faecal matter is avoided.
- The traditional practice of building latrines above carp ponds with direct droppings of fresh faeces and using night soil as fertilisers should be avoided as these will help to maintain the infection in cultured fish populations.
- Avoid the use of water plants as feed (for herbivorous species) if there is a risk of such plants being contaminated with human faecal matter.
- Consider carefully the use of wild fish as feed and make sure they are prepared properly if fed.

Wildlife

Minimise the contact between human waste and wild animals.

Human

Intensive health education should be carried out to emphasise the need to consume only cooked fish, the risks of eating raw fish and the importance of environmental sanitation.

IMPORTANCE

Effect on wildlife

Whilst most wild animals are host to some endoparasitic organisms such as trematodes, the impact of these parasites is usually minimal. Negative impacts on individual animals are only noticed at high parasite loads and even then population level impacts are generally low.

Effect on Agriculture and Aquaculture

Losses to livestock and fish farmers through mortalities are generally low.

Effect on humans

Significant impact on public health, with about 40 million people reportedly infected with trematodes; high prevalence in South East Asia and Western Pacific Regions.

Economic importance

Infections in farmed fish are usually subclinical. However, subclinical infections may be important economically leading to retarded growth and reduced productivity. Infected animals can also become more susceptible to other infections. In livestock, significant costs are involved in control and treatment of infected animals.

FURTHER INFORMATION

Useful publications and websites

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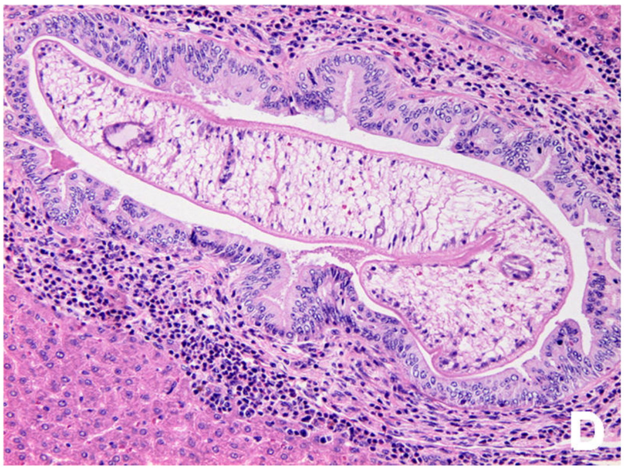
Photos

An adult *Clonorchis sinensis* (measuring 10–25 mm by 3–5 mm): they reside in the small and medium-sized biliary ducts. In addition to humans, carnivorous animals can serve as reservoir hosts (*Sripa* et al*, 2007*).



Photomicrograph of an adult *O. viverrini* worm in bile ducts of experimentally infected hamster (*Sripa, 2008*).

West Nile virus disease

Wetlands inhabited by disease vectors and groups of susceptible animals

Wildlife ✓
Livestock ✓
Human ✓

N

Synonyms: WNVD, West Nile fever (WNF), West Nile fever virus (WNFV), West Nile virus (WNV)

KEY FACTS

What is West Nile virus disease?

A disease that is primarily transmitted between birds, animals and humans by the bite of infected mosquitoes, commonly of the *Culex* and *Aedes* species. Mosquitoes acquire the virus when feeding on infected birds, which are considered the natural hosts of the virus. In birds, it can cause mortality and reduced survival. Other animals, particular horses, may become infected and humans may also contract the virus. In humans the majority of infections will go unnoticed or cause mild disease but in a small proportion of cases the virus can cause severe neurological illness or death. Epidemics are most likely to occur during periods of high mosquito activity.

Causal agent

Virus from the genus *Flavivirus* (arbovirus group B).

Species affected

Many species of bird and some species of terrestrial mammal, including humans. It has also been found, to a lesser extent, in reptiles and amphibians.

Geographic distribution

Originally detected in Africa, the virus appears to be expanding its geographic range into western Asia, the Middle East and Europe, and is now also an important disease in the USA.

Environment

Both temperate and tropical regions inhabited by disease vectors and supporting groups of birds and/or susceptible mammals.

TRANSMISSION AND SPREAD

Vector(s)

Most commonly spread by the bite of an infected mosquito but also by ticks and other insects.

How is the disease transmitted to animals?

Mosquitoes become infected by feeding on infected birds and mammals and then transmit infection when taking the next blood meal. Birds may also become infected after ingesting infected vertebrates and insects.

How does the disease spread between groups of animals?

Birds are the main hosts and once infected, mosquitoes and other biting insects can then spread the disease to other animals and humans. Transmission depends upon the level of virus in the blood, which varies from species to species and stage of infection. Humans and horses are considered ‘dead-end’ hosts, with only low levels of virus in the blood insufficient for efficient mosquito transmission.

How is the disease transmitted to humans?

Most commonly spread by the bite of an infected mosquito but also by ticks and other insects. The disease may also spread to humans through blood transfusions, organ transplants, breast-feeding and from mother to baby during pregnancy but is not spread by person-to-person contact.

IDENTIFICATION AND RESPONSE

Field signs

Unusual bird mortality may signal an outbreak as seen in the USA, but some outbreaks in Europe have not been associated with detectable bird mortality. Commonly reported signs in animals, particularly horses, include weakness, stumbling, trembling, head tremors, reduced mobility, and lack of awareness that allows them to be easily approached and handled.

Humans may suffer from symptoms, also known as West Nile fever, which can include fever, headache, body aches, nausea, vomiting, and sometimes swollen lymph glands or a skin rash on the chest, stomach and back. Symptoms can last for between a few days to several weeks. A small number of people will develop severe symptoms which can include high fever, headache, neck stiffness, stupor, disorientation, coma, tremors, convulsions, muscle weakness, loss of vision, numbness and paralysis. These symptoms can last several weeks and neurological effects may be permanent.

Recommended action if suspected

The disease is notifiable to the OIE so report suspected cases to local and national authorities. Contact and seek assistance from animal and human health professionals immediately if there is any unusual bird mortality or illness in birds, livestock and/or people.

Diagnosis

Detection of the causative agent by health professionals is needed for a definitive diagnosis. For dead birds, fresh organ specimens are required, preferably kidney, brain or heart. Ideally, a variety of species should be tested with emphasis on corvids. In live birds, diagnosis can be made by testing the blood. For other animals and humans, testing usually involves extracting serum and cerebrospinal fluid (CSF).

PREVENTION AND CONTROL IN WETLANDS

Overall

Habitat management.

Encourage mosquito predators and their access to mosquito breeding habitats:

- Connect shallow water habitat (mosquito breeding areas) with deep-water habitat > 0.6m (favoured by larvivorous fish) with steep sides, through meandering channel connections, deep ditches and tidal creeks.
- Include at least some permanent or semi-permanent open water.
- Construct artificial homes or manage for mosquito predators such as bird, bat and fish species.

Reduce mosquito breeding habitat:

- Reduce the number of isolated, stagnant, shallow (2-3 inches deep) areas.
- Cover or empty artificial containers which collect water.
- Manage stormwater retention facilities.
- Strategic manipulation of vegetation.
- Vary water levels.
- Construct a vegetation buffer between the adjacent land and the wetland to filter nutrients and sediments.
- Install fences to keep livestock from entering the wetland to reduce nutrient-loading and sedimentation problems.

In ornamental/more managed ponds:

- Add a waterfall, or install an aerating pump, to keep water moving and reduce mosquito larvae. Natural ponds usually have sufficient surface water movement.

- Keep the surface of the water clear of free-floating vegetation and debris during times of peak mosquito activity.

Vector control (chemical).
It may be necessary to use alternative mosquito control measures if the above measures are not possible or ineffective.
- Use larvicides in standing water sources to target mosquitoes during their aquatic stage. This method is deemed least damaging to non-target wildlife and should be used before adulticides. However, during periods of flooding, the number and extent of breeding sites is usually too high for larvicidal measures to be feasible.
- Use adulticides to spray adult mosquitoes.
- The environmental impact of vector control measures should be evaluated and appropriate approvals should be granted before they are undertaken.

Biosecurity.
Protocols for handling sick or dead wild animals and contaminated equipment can help prevent further spread of disease.
- Wear gloves whilst handling animals and wash hands with disinfectant or soap immediately after contact with each animal.
- Change or disinfect gloves between animals.
- Change needles and syringes between blood collection from different animals.
- Wear different clothing and footwear at each site and disinfect clothing/footwear between sites.
- Disinfect field equipment between animals and sites.

Monitoring and surveillance.
Bird and mosquito surveillance should be prioritised. Animals, particularly horses, are also important sentinels of epizootic activity and human risk in some geographic regions.
- Dead bird surveillance is the most sensitive early detection system. Unusual mortality events should be reported quickly along with prompt submission of selected individual birds for testing. Generally, surveillance should start when local adult mosquito activity begins or should be ongoing if mosquito activity is high all year round.
- Larval and adult mosquito surveillance.
- Horse surveillance, particularly where there have been unusual mortality events, should be reported quickly along with prompt submission of selected samples for testing.

Livestock

- Reduce the chance of animals being bitten by mosquitoes
  - Use insect repellent. Note that this method should not be solely relied upon.
  - Use screened housing with measures to eliminate mosquitoes from inside structures.
  - Use fans to reduce the ability of mosquitoes to feed on animals.
- Vaccination of horses.

Wildlife

A well managed and healthy wetland is the best strategy to prevent or minimise the spread of the virus in the wild. Actions outlined above (►**Overall, Habitat Management**) should be implemented to maximise mosquito predator abundance/diversity and minimise mosquito habitat (accepting that mosquitoes are part of natural diversity of wetlands).

Conduct active dead bird monitoring: unusual bird deaths may signal a West Nile virus disease outbreak and should be quickly reported to local animal health authorities. Because of their susceptibility, the same caution should also be applied to any wild or free-ranging horse populations inhabiting wetlands.

Humans

Medical attention should be sought if WNV is suspected. Milder symptoms usually pass on their own but hospitalisation may be needed in more severe cases for supportive care (there is no human vaccine and no specific treatment for humans).

Measures to reduce the chance of being bitten by mosquitoes:

- Wear light coloured clothing which covers arms and legs.
- Use impregnated mosquito netting when sleeping outdoors or in an open unscreened structure.
- Avoid mosquito-infested areas or stay indoors when mosquitoes are most active.
- Use colognes and perfumes sparingly as these may attract mosquitoes.
- Use mosquito repellent when outdoors. Note that some repellents cause harm to wildlife species, particularly amphibians. Wash hands before handling amphibians.
- Use citronella candles and mosquito coils in well ventilated indoor areas.
- Use mesh screens on all doors and windows.

IMPORTANCE

Effect on wildlife

Causes morbidity and mortality in many species of bird and some species of mammal although its impacts on animal populations are currently unknown. Some infected bird species may also have reduced survival. There are concerns that species vulnerable to fatal infection may be more prone to extinction, although there is no evidence of this currently. The disease can result in negative perception and therefore unnecessary destruction of wildlife.

Effect on livestock

Horses are particularly affected and up to 30% of those showing clinical signs may die. Poultry do not appear to be seriously affected.

As well as affecting birds and humans, horses are susceptible to infection and can suffer high levels of mortality (*Matthew Simpson*).

Effect on humans

Most people (80%) bitten by an infected mosquito show no signs or symptoms. Only around 20% of the people who become infected will develop symptoms, usually West Nile fever. A small number (<1%) will suffer from a severe infection (West Nile encephalitis, West Nile meningitis, or acute flaccid paralysis). People over 50 years old or with suppressed immune systems are most likely to develop severe illness or die.

Economic importance

There is potential for significant economic losses to the equine industry, through death and illness in horses. Illness in humans can result in economic losses due to the time lost from normal activities. Effects on wildlife and in zoological collections can have a significant impact on tourism.

FURTHER INFORMATION

Useful publications and websites

- Centers for Disease Control and Prevention (CDC). **Epidemic/epizootic West Nile virus in the United States; guidelines for surveillance, prevention and control**. (2003). www.cdc.gov/ncidod/dvbid/westnile/resources/wnv-guidelines-aug-2003.pdf. [Accessed March 2012].
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- U.S Geological Survey (USGS) National Wildlife Health Center. **West Nile virus**. www.nwhc.usgs.gov/disease_information/west_nile_virus/index.jsp. [Accessed March 2012].
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Contacts

- **Centers for Disease Control and Prevention (CDC) public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY).**
- **WHO Communicable Diseases Surveillance and Response (CSR)**. zoonotic_alert@who.int fmeslin@who.int and outbreak@who.int
- **FAO Animal Production and Health Division**. www.fao.org/ag/againfo/home/en/who.htm

Chapter 5
Where to go for Further Assistance and Advice

In this chapter you will find:

A list of key contacts.

A bibliography of key resources providing information and guidance on disease management.

Chapter contents

5.1 Key contacts

✉ **Email or post address**
☎ **Phone number**
🖱 **Website**

Centers for Disease Control and Prevention (CDC)

The CDC, based in Atlanta in the US, is responsible for protecting people from public health threats. Within this US federal agency the **National Center for Emerging and zoonotic infectious diseases** (NCEZID) utilises and provides research, health monitoring, training prevention and preparedness strategies to prevent and control infectious zoonotic diseases.

✉ **CDC Headquarters**
1600 Clifton Rd
Atlanta, GA.
30333
USA
✉ cdcinfo@cdc.gov
☎ +1 800 232 4636
TTY (Text telephone): +1 888 232 6348
☎ Public response hotline
+1 888 2462675 (English), +1 888 246 2857 (Español), or +1 866 874-2646 (TTY).
🖱 http://www.bt.cdc.gov/

Within the NCEZID, the **Division of Vector-Borne Diseases** (DVBD) strives to protect the US from bacterial and viral zoonoses transmitted by **mosquitoes**, **ticks** and **fleas** and can be contacted at the address below**:**

✉ **Division of Vector-Borne Diseases**
Centers for Disease Control and Prevention
3150 Rampart Road
Ft. Collins, CO 80521
USA
✉ **Division of Emerging Infections and Surveillance Services** (DEISS)
contact: Helen Perry HPerry@cdc.gov
✉ **Office of Global Health Division of International Health**
contact: Jim Vaughan JVaughan@cdc.gov

☎ **CDC National Center for Environmental Health and Agency for Toxic Substances and Disease Registry**: +1 770-488-7100

Food & Agriculture Organization of the United Nations (FAO's) Animal Production and Health Division (AGA)

The FAO, an intergovernmental organization, has 191 Member Nations and is present in over 130 countries. Operating from the FAOs headquarters in Rome, Italy, the Animal Production and Health Division (AGA) encompasses the Animal Health Service (AGAH). This department is a source of technical expertise on the control of transboundary disease emergencies, working to provide early disease detection, based on disease intelligence and surveillance carried out in conjunction with the World Organisation for Animal Health (OIE) (FAO, 2012).

🖂 **FAO Headquarters**
Viale delle Terme di Caracalla
00153 Rome, Italy
☎ +39 06 57051
Fax: +39 06 570 53152
🖂 FAO-HQ@fao.org
🖰 http://www.fao.org/

🖰 **Animal Production & Health Division Contacts**:
www.fao.org/ag/againfo/home/en/who.htm

🖂 **Emergency Prevention System for Transboundary Animal and Plant Pests and Diseases (EMPRES)**
Jan Slingenbergh
Senior Officer
FAO HQ, Room C-522
Viale delle Terme di Caracalla
Rome 00153, Italy
☎ +39 06 570 54102
🖂 Jan.Slingenbergh@fao.org

World Organisation for Animal Health (OIE)

The OIE is the intergovernmental organisation responsible for improving animal health worldwide. It is recognised as a reference organisation by the World Trade Organization (WTO) and in 2011 had a total of 178 Member Countries. The OIE maintains permanent relations with 45 other international and regional organisations and has regional and sub-regional offices on every continent (OIE, 2012).

🖂 **OIE Headquarters**
12 Rue de Prony
75017 Paris, France
🖂 oie@oie.int
☎ +33 (0) 1 44 15 18 88
🖰 http://www.oie.int

USGS National Wildlife Health Center (NWHC) – for US Enquiries

The National Wildlife Health Center (NWHC) is a science center of the Biological Resources Discipline of the United States Geological Survey. The NWHC was established in 1975 as a biomedical laboratory dedicated to assessing the impact of disease on wildlife and identifying the role of various pathogens in contributing to wildlife losses. The NWHS aims to provide national leadership to safeguard wildlife and ecosystem health through dynamic partnerships and exceptional science (USGS, 2012).

🖂 **USGS Headquarters**
6006 Schroeder Road
Madison
WI 53711-6223
USA
🖂 AskNWHC@usgs.gov
☏ US enquiries: +1 608 270 2400
🖰 http://www.usgs.gov/

World Health Organization (WHO)

The WHO, based in Geneva, Switzerland, is the directing and coordinating authority for health within the United Nations system. It is responsible for providing leadership on global health matters, shaping the health research agenda, setting norms and standards, articulating evidence-based policy options, providing technical support to countries and monitoring and assessing health trends (WHO, 2012).

🖂 World Health Organization
Avenue Appia 20
1211 Geneva 27
Switzerland
☏ + 41 (0) 22 791 21 11
Fax: +41 (0) 22 791 31 11
🖰 www.who.int/

🖂 **WHO Communicable Diseases Surveillance and Response (CSR)**
zoonotic_alert@who.int fmeslin@who.int and outbreak@who.int

5.2 Bibliography of key resources

FAO Corporate Document Repository; Diseases & Pests of Animals and Plants. http://www.fao.org/documents/en/docrep.jsp [Accessed March 2012].

OIE Listed Diseases (2012). http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2011/ [Accessed March 2012].

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2011. (Sixth edition, Volume 1, 2008). http://www.oie.int/manual-of-diagnostic-tests-and-vaccines-for-terrestrial-animals/ [Accessed March 2012].

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OIE (2010). Training Manual on Wildlife Diseases and Surveillance. Workshop for OIE National Focal Points for Wildlife, Paris. http://www.oie.int/fileadmin/Home/eng/Internationa_Standard_Setting/docs/pdf/WGWildlife/A_Training_Manual_Wildlife.pdf [Accessed March 2012].

OIE World Animal Health Information Database (WAHID) Interface. http://web.oie.int/wahis/public.php?page=home [Accessed March 2012].

The Merck Veterinary Manual. http://www.merckvetmanual.com/mvm/index.jsp [Accessed March 2012].

U.S Geological Survey (USGS) Field Manual of Wildlife Disease – General Field Procedures and Diseases of Birds: http://www.nwhc.usgs.gov/publications/field_manual/ [Accessed March 2012].

Wetlands International (2010). Wetlands & Water, Sanitation and Hygiene (WASH) - understanding the linkages. Wetlands International, Ede, The Netherlands. http://www.wetlands.org/WatchRead/Currentpublications/tabid/56/mod/1570/articleType/ArticleView/articleId/2467/Default.aspx [Accessed March 2012].

WHO Global Alert and Response (GAR). http://www.who.int/csr/disease/en/ [Accessed March 2012].

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Appendix II
Key Messages for Wetland Managers and Policy Makers

1. Introduction to diseases in wetlands

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS
▪ The term '**disease**' is used to define any impairment to health resulting in dysfunction. There are **many disease types**, including: infectious, toxic, nutritional, traumatic, immunological, developmental, congenital/genetic and cancers. ▪ **Disease is often viewed as a matter of survival or death when, in fact, effects are often far more subtle**, instead affecting productivity, development, behaviour, ability to compete for resources or evade predation, or susceptibility to other diseases factors which can consequentially influence population status. ▪ **Well functioning wetlands** with well managed livestock, with little interface, with well managed wildlife should provide human wetland dwellers with the **ideal healthy environment in which to thrive.** ▪ **Disease is an integral part of ecosystems** serving an important role in population dynamics. **However, there are anthropogenic threats affecting wetlands** including climate change, substantial habitat modification, pollution, invasive alien species, pathogen pollution, wildlife and domestic animal trade, agricultural intensification and expansion, increasing industrial and human population pressures including the interface between humans and domestic and wild animals within wetlands, all of which may act as drivers for emergence or re-emergence of diseases. ▪ **Wetlands are meeting places** for people, livestock and wildlife and **infectious diseases can be readily transmitted** at these interfaces. ▪ **Stress is often an integral aspect of disease** capable of exacerbating existing disease conditions and increasing susceptibility to infection. **There are a broad range of stressors** including toxins, nutritional stress, disturbance from humans and/or predators, competition, concurrent disease, weather and other environmental perturbations. Stressors can be additive, working together to alter the disease dynamics within an individual host or a population. ▪ **Impacts** of disease on public and livestock health, biodiversity, livelihoods and economies **can be significant**. ▪ **The emergence and re-emergence of diseases has become a wildlife conservation issue** both in terms of the impact of the diseases themselves and of the actions taken to control them. Some diseases may be significant sources of morbidity and mortality of wetland species and in some cases (*e.g.* amphibian chytridiomycosis) can play a role in multiple extinctions of wetland species.

2. Principles of managing diseases in wetlands

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- The term '**disease**' is used to define any impairment to health resulting in dysfunction. There are **many disease types**, including: infectious, toxic, nutritional, traumatic, immunological, developmental, congenital/genetic and cancers.
- **Disease is often viewed as a matter of survival or death when, in fact, effects are often far more subtle**, instead affecting productivity, development, behaviour, ability to compete for resources or evade predation, or susceptibility to other diseases factors which can consequentially influence population status.
- **Well functioning wetlands** with well managed livestock, with little interface, with well managed wildlife should provide human wetland dwellers with the **ideal healthy environment in which to thrive.**
- **Disease is an integral part of ecosystems** serving an important role in population dynamics. **However, there are anthropogenic threats affecting wetlands** including climate change, substantial habitat modification, pollution, invasive alien species, pathogen pollution, wildlife and domestic animal trade, agricultural intensification and expansion, increasing industrial and human population pressures including the interface between humans and domestic and wild animals within wetlands, all of which may act as drivers for emergence or re-emergence of diseases.
- **Wetlands are meeting places** for people, livestock and wildlife and **infectious diseases can be readily transmitted** at these interfaces.
- **Stress is often an integral aspect of disease** capable of exacerbating existing disease conditions and increasing susceptibility to infection. **There are a broad range of stressors** including toxins, nutritional stress, disturbance from humans and/or predators, competition, concurrent disease, weather and other environmental perturbations. Stressors can be additive, working together to alter the disease dynamics within an individual host or a population.
- **Impacts** of disease on public and livestock health, biodiversity, livelihoods and economies **can be significant**.
- **The emergence and re-emergence of diseases has become a wildlife conservation issue** both in terms of the impact of the diseases themselves and of the actions taken to control them. Some diseases may be significant sources of morbidity and mortality of wetland species and in some cases (*e.g.* amphibian chytridiomycosis) can play a role in multiple extinctions of wetland species.

3. Practices of managing diseases in wetlands

3.1 Assessing risk and planning for the future

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- To ensure consideration for **disease prevention and control** is **at the heart of wetland management**, activities need to be **integrated into wetland management plans**. Clearly defined roles and responsibilities are required to ensure effective management which can deliver a range of benefits to stakeholders.
- **Risk assessments are valuable tools for animal health planning** and serve to identify problems/hazards and their likely impact thus **guiding wetland management practices**. From these assessments, risk management and communication actions can be taken. **Good** local, national and regional **surveillance data** are needed **for robust risk assessments**. Risk assessments are living documents which require regular revision.
- **Multidisciplinary advisory groups provide a broad range of benefits** for disease prevention and control. Their role is to review epidemiological and other disease control information, inputting to the activation of agreed contingency plans and advising the appropriate decision makers on future contingency planning. As appropriate, wetland managers can play a key role in these groups.
- **Contingency plans aim to consider possible emergency disease management scenarios** and to integrate **rapid cost effective response actions** that allow the disease to be prevented and/or controlled. It is advisable to develop bespoke contingency plans for specific high-risk/high-priority diseases and also generic standard operating procedures (SOPs) that may be common to many situations. Plans and SOPs should be documented and tested with a broad range of stakeholders in 'peacetime' (*i.e*. outwith any emergency situation), and subjected to periodic review.

3.2 Reducing risk of disease emergence

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- An **understanding by the wetland manager of the uses** of a wetland and its catchment by people, industry, agriculture including livestock, and wildlife, coupled with an appreciation of risk factors for disease emergence, can **provide a sound foundation for disease risk reduction**.
- It is **important that wetland managers identify stressor risks** within their site and the broader catchment/landscape, and understand that these may change over time. Once these factors are identified, **they can be managed and/or their impact mitigated,** as appropriate.
- **Disease zoning** (although challenging in wildlife and/or aquatic systems) **can help control some infectious diseases** through the delineation of infected and uninfected zones defined by sub-populations with different disease status. Buffer zones separating infected and uninfected zones may consist of physical barriers, an absence of hosts, an absence of disease vectors or only immune hosts *e.g*. following ring vaccination. **Appropriate levels of surveillance are required** to accurately define zones and for prevention of disease spread to

occur, the movements of animals between zones, needs to be restricted.

- **The movement of infected animals to new areas and populations represents the most obvious potential route for introduction of new/novel infections**. The risk of transmission and spread of disease can be **minimised by conducting risk assessments and following certain standardised national and international guidelines and regulations** for moving, relocating and/or releasing animals. A disease risk analysis should be conducted for any translocations for conservation purposes.
- **Biosecurity** in wetlands refers to the **precautions taken to minimise the risk of introducing infection (or invasive alien species)** to a previously uninfected site and, therefore, preventing further spread. Infectious animal diseases are spread not only through movement of infected hosts but also their products *e.g*. faeces, saliva *etc*. or *via* human and fomite (inanimate object) contact with animals and their products. **Constructed treatment wetlands can assist greatly in reducing risks** from contaminated wastewaters.
- Where possible, **biosecurity measures should be implemented routinely** as standard practice whether or not an outbreak has been detected. A regional/supra-national approach to biosecurity is important for trans-boundary diseases, particularly those where domestic and international trade are considered as important pathways for disease spread, *e.g*. trans-boundary aquatic animal diseases.
- **If wetland stakeholders understand the principles and value of biosecurity** and what measures to take, this will **encourage the development of an everyday 'culture' of biosecurity** which can help disease prevention and control.
- **Implementing biosecurity measures in the natural environment can be extremely challenging**, particularly in aquatic systems, and although eliminating risk will be impossible, **a substantial reduction in risk may be achievable**, particularly where several complementary measures are employed.

3.3 Detecting, assessing and responding to new disease

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- The **detection** of new, emerging disease, robust **risk assessments**, and effective **disease control** in and around wetlands, **all rely on effective disease surveillance and monitoring**. Surveillance programmes should be well designed with clearly defined aims and objectives. Robust surveillance requires appropriate methods for sample collection, recording, storage and transportation, which in turn depend on well trained personnel and adequate resourcing.
- **Timely and accurate diagnoses and early warning systems for disease emergence are critical for swift responses**, achieving effective disease control and minimising losses and costs. Early warning systems may depend on a comprehensive understanding of a wetland site and catchment, good disease intelligence from a range of stakeholders (including crucially the wetland manager, as well as data from local and national disease surveillance programmes), and clear systems and networks for communication and reporting.
- **Identifying** when a disease presents a '**problem**' is complex and **requires** thorough disease investigation and existing **good long term surveillance** information.
- In the event of a suspected outbreak of disease, **wetland managers are not expected to be**

the final disease diagnostician. However, they should **play a key role in an outbreak investigation** team being ideally placed to provide the crucial contextual epidemiological information about timing of events, the populations at risk, the effects on these, land use and environmental conditions at the time and leading up to the outbreak, and other relevant local information.

3.4 Managing disease

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- **The appropriate approach to disease management will depend on the characteristics of the problem** and, when dealing with an infectious disease, on the correct identification of reservoirs, hosts and vectors of infection. **Management measures may target** the **pathogen**, **host, vector, environmental factors or human activities**. Ultimately, an **integrated approach** involving several complimentary measures is likely to be most **successful** in managing diseases in wetlands.
- **Disinfection and sanitation** procedures target pathogens and can be **very effective at controlling spread of infection** but must be **used with caution in wetland situations** to avoid negative impacts on biodiversity.
- **Animal carcases** represent a significant **potential source of infection** and **require rapid and appropriate collection and disposal**. Disposal options are varied and again need to be used with caution in wetland situations to reduce risks of pollution of water courses or further spread of infection.
- **Targeting vectors in integrated disease control strategies can be effective** and usually take the form of environmental management, biological controls and/or chemical controls, or actions to reduce the contact between susceptible hosts and vectors. To reduce negative impacts on biodiversity **caution must be used when using these measures within wetlands**.
- **Vaccination programmes**, often supplemented by other disease control measures, **can help control** and even eliminate diseases affecting livestock. Vaccination of wildlife is feasible but it is often complex - other management strategies may be of greater value.
- **Habitat modification in wetlands can eliminate or reduce the risk** of disease, by reducing the prevalence of disease-causing agents, vectors and/or hosts and their contact with one another, through the manipulation of wetland hydrology, vegetation and topography and alterations in host distribution and density.
- **Movement restrictions of animals and people**, usually imposed by government authorities, can be an **effective tool** in preventing and controlling disease transmission through avoiding contact between infected and susceptible animals.
- **Complete eradication of a disease requires a thorough understanding of its epidemiology, sufficient political and stakeholder support** and thorough resourcing and is thus rarely achieved! Elimination of disease from an area is a more likely outcome although this depends on measures to prevent re-emergence being taken. ‘Stamping out’ (involving designation of infected zones, quarantine, slaughter of susceptible species, safe disposal of carcases and cleaning and disinfection) is a management practice used for rapidly reducing the prevalence of a disease during an outbreak situation.

3.5 Communication, education, participation and awareness (CEPA)

KEY MESSAGES FOR WETLAND MANAGERS AND POLICY MAKERS

- Well planned, targeted and resourced CEPA programmes for wetland stakeholders are essential for raising awareness and appreciation of wetland diseases and the measures that can be taken to successfully prevent, detect, control and mitigate disease outbreaks. Such programmes should be integrated into all wetland disease management strategies.
- Programmes should aim to inform wetland stakeholders of the basic principles of healthy habitat management, thus reducing the risk of a disease outbreak.
- A ‘culture’ of proactive disease management can only be developed if a broad range of wetland stakeholders participate in CEPA programmes.
- Communication strategies should aim to make stakeholders aware of the nature and potential consequence of animal disease and of the benefits gained from prevention and control measures. They should ultimately encourage people to take the recommended courses of action in preventing and controlling a disease outbreak. Awareness raising campaigns should emphasise the importance of early warning systems and of notifying and seeking help from the nearest government animal and/or human health official as soon as an unusual disease outbreak is suspected.
- Selection of the appropriate message, the messenger and the method of delivery is critical for successful communication.
- A strategy, written in ‘peacetime’ for dealing with the media can increase likelihood of successful outcomes from this relationship maximising potential benefits and minimising potential negative impacts.
- Simulation exercises and testing of contingency plans are a valuable method for training.

Appendix III
Glossary

Abiotic disease: Non-infectious disease caused by non-living environmental agents, such as toxic chemicals, heavy metals, extreme temperatures, UV radiation, nutrient imbalance.

Acaricides: A chemical used to kill mites or ticks.

Accidental host: A host that harbours an organism that is not ordinarily pathogenic in that particular species. Accidental hosts are usually a 'dead end' for a pathogen.

Acute: Meaning either a rapid onset of infection or short in nature.

Agar gel immunodiffusion: Laboratory technique that uses the diffusion of antibodies and antigens across an agar gel to diagnose infections.

Aerosol: Suspension of solid or liquid particles in a gas or droplets of liquid (*e.g.* disease agents in spray).

Aetiology: The study of the causes of diseases.

Anorexia: Poor appetite and/or inability to eat leading to loss of body weight.

Anthelminthic: A substance capable of destroying or expelling parasitic worms.

Anthropogenic: Caused or influenced by human activities.

Antibiotic: Chemical substance produced with the ability to kill or inhibit growth of other microorganisms. Used in the treatment of some infectious diseases.

Antibodies: Serum protein produced by lymphocytes in response to the presence of specific antigens. Detection of specific antibodies is useful for diagnosis.

Anticoagulated: The prevention of coagulation (clotting), usually referring to blood taken into tubes containing an additive *e.g.* heparin.

Antigen: Any substance that is recognised by the body as foreign *e.g.* invading organisms, toxins, non-self tissues. The recognition gives rise to an immune response and antibody production.

Aquaculture: The cultivation of aquatic plants and animals for food.

Arthropod: A member of the phylum Arthropoda. An invertebrate animal with an external skeleton, a segmented body and jointed appendages (*e.g.* insects, arachnids, crustaceans).

Ascitic: An abnormal accumulation of serous fluid (or serum) in the abdominal cavity.

Asymptomatic: Carrying an infection or diseased but showing no symptoms.

Ataxia: Neurological disorders which cause the loss of ability to coordinate muscular movement.

Attenuated vaccine: A vaccine containing a weakened form of the organism that causes the disease. Also called a live vaccine.

Avian influenza (AI): A disease of birds caused by influenza A virus, can refer to either low pathogenic or highly pathogenic forms of the disease (LPAI or HPAI).

Bacteriophage: A virus that parasitises a bacteria.

Biliary duct: A duct that transports bile from the liver to the intestines.

Bioassay: Bioassay (biological assay) is a procedure that determines the concentration of a particular biological constituent of a mixture.

Biochemical: Chemical composition of a particular living system or biological substance.

Biosafety: The precautions taken to prevent exposure to infectious agents.

Biosecurity: The precautions taken to minimise the risk of introducing infection (or invasive alien species) to a previously uninfected site and therefore preventing further spread.

Biotic diseases: Those caused by a living agent, such as a bacterium, virus, fungus or protist.

Bovid: Member of the Bovidae family (including cattle, buffalo and bison).

Buffer zone: An area of land separating two or more different land types (*e.g.* between a disease-infected area and a disease-free area). This zone may consist of physical barriers, an absence of hosts, an absence of disease vectors or only immune hosts e.g. following ring vaccination.

Bushmeat: Hunting and/or consumption of meat from wild animals.

Caecal: Of, or pertaining to, the cæcum, or blind gut.

Carrier (disease): A person or organism infected with an infectious disease agent but displaying no symptoms (asymptomatic).

Caudates: Latin for 'tail', refers to amphibians with tails (*e.g.* salamanders and newts).

Causative: Refers to the agent or cause of a disease.

Cercaria: Parasitic larval stage of a trematode worm.

Cervid: Member of the Cervidae family *i.e.* deer.

Challenge: The physiological, and especially immunological, stress a host is subjected to by a pathogen.

Chemical fixation: Using chemicals to preserve tissues and prevent decay.

Chemotaxis: The characteristic movement or orientation of an organism or cell along a chemical concentration gradient either toward or away from a chemical stimulus.

Chronic: Describing a long-standing disease or lesion which may follow an acute phase, or a disease marked by frequent reoccurrence.

Clinical signs: Observed changes in the course of a disease process.

Cloacal: The common cavity into which the intestinal, genital and urinary tract open in vertebrates such as birds, fish, reptiles and some primitive mammals.

Colostrum: The first secretion from the mammary glands after giving birth, rich in antibodies.

Communicable: Capable of being transmitted from one person/species to another, infectious or contagious in nature.

Convulsions: Uncontrolled shaking of the body as a result of the body muscles rapidly and repeatedly contracting and relaxing.

Counter immune-electrophoresis: A laboratory technique that uses an electrical current to migrate antibodies and antigens across a buffered agar gel. Used to diagnose bacterial infections.

Culling: A reduction in population by selective slaughter. Usually implemented to restrict disease movement.

Culture: The growth and multiplication of biological cells in a controlled nutrient-rich medium.

Cutaneous: Relating to or affecting the skin.

Decontamination: The process of cleansing to remove contamination from substances.

Definitive host: An animal in which a pathogen lives and reproduces.

Diagnosis: Determining the nature and cause of a disease through examination of physical and chemical symptoms.

Diagnostician: Person that performs diagnostics.

Diagnostics: The science and practice of diagnosis.

Disease: A departure from a state of health or any impairment to health resulting in physiological dysfunction.

Disease agent: An organism capable of causing disease *i.e.* viruses, bacteria, fungi, protozoa, helminths or arthropods.

Disease ecology: The interaction of the behaviour and ecology of hosts with the biology of pathogens in relation to the impacts of diseases on populations.

Ecohealth: The concept of health at the level of ecosystem, appreciating the interconnectivity of humans and all living organisms and functions within this and how these linkages are reflected in a population's state of health.

Ecological: The relationship between organisms and their environment.

Ecthyma: A contagious viral disease of sheep and goats marked by lesions on the lips.

Ectoparasite: An external parasite.

ELISA: Enzyme-linked immunosorbant assay. A diagnostic test that uses disease specific proteins (antigens or antibodies) to detect antibodies (or antigens), and therefore disease.

Emerging disease: A disease that has appeared in a population for the first time, or that may have existed previously but is rapidly increasing in incidence or geographic range, or has recently evolved from another disease.

Encyst: Enclose or become enclosed in a cyst.

Endemic: Native to a population, or a disease characteristic of a particular area.

Endogenous: Originating from within an organism.

Endoparasite: An internal parasite.

Enteric: Relating to or being within the intestines.

Entomopathogens: Pathogens that infect insects.

Epidemic: A disease affecting many organisms at the same time, spreading rapidly within a population where the disease is not usually prevalent.

Epidemiology: The study of the distribution and determinants of health-related states and its application to the control of diseases.

Equid: Member of the Equidae family *i.e.* wild or domestic horses.

Eradicate: To exterminate an infectious agent so no further cases of a specific disease arise.

Exotic: Non-native species introduced to areas where they do not naturally occur.

Flukes: Also known as trematodes, a class of parasitic flatworm.

Fomites: Inanimate objects on which disease agents may be transported (*e.g.* bedding or faeces).

Gastroenteritis: Inflammation, infection or irritation of the digestive tract, particularly the stomach and intestine.

Genetic resistance: Genetically determined resistance to specified infectious agents.

Haemorrhage: Profuse bleeding from ruptured blood vessels.

Health: A positive state of physical and mental well-being.

Helminth: Parasitic worm.

Herbivorous: Plant eating animals.

Heterozygosity: The state of being heterozygous *i.e.* having dissimilar alleles at corresponding chromosomal loci **–** having genetic diversity.

Histopathology: Diagnosis and study of disease by expert interpretation of cells and tissue samples.

Horizontal transmission: Transmission of an infectious agent between members of the same species.

Host: An organism in which another, usually parasitic, organism is nourished and harboured.

Host range: The range of host species which a particular pathogen is able to infect.

HPAI H5N1: Highly pathogenic avian influenza. H5N1 refers to the combination of haemagglutinin (H) and neuraminidase (N) proteins on the surface of the virus coat protein.

Hydrology: Pertains to the movement, distribution and quality of water.

Hyphae: Long, branching filamentous structures of a fungus.

Iodophore: A solution that contains iodine and a surface-active agent, it releases iodine gradually to act as a disinfectant.

Immunity: The condition of being immune refers to a state in which a host is not susceptible to infection or disease from invasive pathogens.

Immunocompetence: The ability of the body to resist disease and distinguish between alien and endogenous bodies.

Immunocompromise: Having an impaired immune system and therefore a reduced ability to mount an immune response and fight infection.

Immunofluorescence: A laboratory technique used to detect the presence of an antigen or antibody in a sample by coupling a specific interactive antigen or antibody with a fluorescent compound.

Immunohisto-chemistry: The application of immunological techniques to the chemical analysis of cells and tissues.

Immunologically naïve: Pertaining to an immune system not previously exposed to stimuli from pathogens.

Immunosuppression: The inhibition of the normal immune response because of disease, administration of drugs or surgery.

Incidence: The number of individual cases of disease in relation to the population at risk.

Infection: Occurs when one living organism (the host) is invaded by another living organism. This may be clinically inapparent or result in only local cellular damage. The infection may remain localised, subclinical and temporary if the immune system is effective. A local infection may persist and spread to become an acute, subacute or chronic clinical infection or disease state when micro-organisms gain access to lymphatic or vascular systems.

Infectious: Capable of being transmitted between host organisms.

Intermediate host: An animal in which a parasite lives in a non-sexual, larval stage.

Intervention: The act of intervening in a disease or epidemiological sequence.

Intraspecific: Arising or occurring within a species.

Invasive alien species: Species that have been introduced outside their natural distribution area.

Keystone species: A species with a disproportionate impact *i.e.* positive function, on its ecosystem relative to biomass and abundance.

Larvicide: An insecticide specifically targeted against the larval life stage of an insect to halt the life cycle.

Lesion: A region in an organ or tissue that has suffered damage through injury or disease.

Live vaccine: A vaccine containing a weakened form of the organism that causes the disease. Also known as an attenuated vaccine.

Maintenance host: An animal which is capable of acting as natural source of infection for other individuals of the same species (see reservoir host).

Meningeal: Of or affecting the meninges (the membrane system that envelope the central nervous system).

Metacercaria: The encysted larva of a trematode in an intermediate host.

Metamorphs: Amphibian life-cycle stage between larvae and adult.

Microbial: Pertaining to microorganisms.

Microorganisms: Microscopic organism such as bacteria, virus, fungi or protozoa.

Microprogagated: The production of a large number of individual plants from a piece of plant tissue cultured in a nutrient medium.

Miracirdium: Free-living, ciliated, first larva stage of the trematode lifecycle.

Mitigate: To moderate or alleviate a condition.

Morbidity: Incidence of clinical cases of a disease in a given population.

Mortality: The incidence of death in a given population in a given period. The incidence of death among animals affected by a particular disease or condition.

Mustelids: Member of the Mustelidae family (includes weasel, ferret, mink, otter and skunk).

Necrophagy: Eating dead bodies or carrion.

Necropsy: *Post mortem* examination.

Non-infectious: Not capable of being transmitted between host organisms.

Non-zoonotic: Disease or infection which cannot be naturally transmitted from vertebrate animals to humans (noun: non-zoonosis). *See 'zoonosis'.*

Notifiable: A disease which must be reported to the relevant national and international authority (*i.e.* OIE).

Novel disease: A novel disease is one that has not previously been exposed to a given species or population. This may be due to mutation of the organism that is responsible for the disease.

Obligate: Used to describe an organism which is bound or restricted to a certain habitat, host or environment.

Oomycetes: Marine, freshwater and soil living non-photosynthetic algae-like fungi, includes water moulds and downy mildews.

Oropharyngeal: Of, or relating to, the oropharynx, which is the part of the pharynx, between the soft palate and the epiglottis.

Ovid: Member of the Ovidae family *i.e.* wild or domestic sheep.

Oviposition: Process of depositing or laying eggs.

Paralysis: Temporary or permanent loss of muscle function in the body.

Parasite: Used in this context to be synonymous with *pathogen*. However, the *microparasites* (viruses, bacteria, fungi and protozoa) tend to be referred to as *pathogens*. *Macroparasites* refer to helminths and arthropods and are often referred to simply as *parasites*.

Parasite load: A measure of the degree of which a host is burdened by parasites.

Pathogen: An organism capable of causing infection and disease *i.e.* viruses, bacteria, fungi, protozoa, helminths or arthropods.

Pathogenic: Causing disease or capable of doing so.

Pathogenicity: A pathogen's ability to cause disease. Similar to the term 'virulence' but broader because it is more of a qualitative term.

Pathology: The study of the structural and functional changes in the fluids, cells, tissues and organs of the body.

PCR: Polymerase chain reaction (PCR): a technique in molecular biology to amplify a single or a few copies of a piece of DNA or RNA across several orders of magnitude, generating thousands to millions of copies of a particular sequence allowing easier detection.

Peacetime: Used in this context to mean before a problem.

Phytosanitary: Relating to the health of plants and the prevention of spreading plant diseases.

***Post mortem*:** After death but often used to refer to medical/veterinary examination of a dead body (short for *post mortem* examination).

Poultry: Term referring to domestic birds bred for meat, eggs and/or feathers. Includes chickens, turkeys, ducks, geese, quail *etc*.

Prevalence: A general term describing the commonality of a disease or condition in a group of animals. Proportion of individuals within a given population with disease at a given time.

Protist: Mostly single-celled, but some multicellular, organisms in the kingdom Protista. Includes protozoans, eukaryotic algae and slime moulds.

Pulmonary: Pertaining to the lungs.

Quarantine: A period of isolation to cover the incubation period of a certain disease which reduces disease transmission to the remaining population.

Redia: The cylindrical larval stage of some trematodes.

Reservoir host: Refers to host organisms that serve as a source of infection by sustaining a population of an infectious pathogen, often with immunity to the disease. Pathogens may 'spillover' from reservoir populations to cause disease in nearby susceptible hosts which may be a different species.

Resilient: Ability to recover quickly or withstand adverse conditions.

Resistant: Ability of an organism to remain uninfected and/or unaffected by agents.

Scrapie: Degenerative brain disease of sheep.

Secretions: Substances secreted from the blood or cells (*e.g.* saliva, mucus, tears, bile, and hormones).

Sentinel herd: Small herd of susceptible hosts located in geographically representative areas used to detect prevalent diseases.

Septicaemia: Blood poisoning, invasion of the bloodstream by virulent microorganisms from a focus of infection.

Serological: Relates to serum and antigen-antibody reactions.

Serovar: A group of closely related microorganisms distinguished by a characteristic set of antigens.

Slurries: Thin, watery mixtures of fine, insoluble material such as clay, cement, soil, or faeces.

Spatial variation: Differences in a landscape usually associated with populations. For example, this may be related to habitat or weather differences.

Spillback: Reverse spillover of infectious agents from wildlife to sympatric populations of susceptible animals (often domesticated species).

Spillover: The transmission of infectious agents from reservoir animal populations (often domesticated species) to sympatric wildlife.

Spore: An infectious body produced within bacteria.

Sporocyst: The larva of a trematode worm that produces redia larvae by asexual reproduction.

Sterile: Free from microorganisms.

Stressors: A chemical or biological agent, an environmental condition, an external stimulus or an event that causes stress to an organism (*e.g.* capture, overcrowding, harassment by humans or other animals).

Subclinical: A mild infection or early stage infection with no detectable symptoms.

Substrate: A surface on which an organism grows.

Suid: Member of the Suidae family *i.e.* wild or domestic pigs (including warthog, babirusa and bush pigs).

Susceptibility: The state of being susceptible *i.e.* readily affected by disease.

Symbiont: An organism in a symbiotic relationship *i.e.* a relationship of mutual benefit or dependence.

Syndromes: The result of the combination of clinical signs or symptoms that collectively indicate or characterise a disease.

Taxa: Plural of taxon. A taxonomic category for the classification of organisms.

Toxin: A type of poisonous substance.

Transmission: Transfer of an infection from one source to another.

Trypanotolerant: Trypanotolerant and trypanotolerance describe the condition of being able to resist trypanosomiasis *e.g.* some endemic breeds of cattle.

Vector: A carrier which transfers an infectious agent from one host to another *e.g.* a tsetse fly carries trypanosomes from animals to humans and other animals.

Vertical transmission: Transmission of an infectious agent between different generations within a population *i.e.* mother to offspring.

Viable: Capable of function under favourable conditions.

Virion: The infective form of a virus.

Virulence: The severity to which a microorganism can cause disease, similar to pathogenicity.

Waterbird: Species of birds ecologically dependent on wetlands for at least part of their annual cycle. Synonymous with 'waterfowl'.

Water-borne disease: A disease caused by pathogenic micro-organisms that are most commonly transmitted in contaminated fresh water.

Zoonosis: Disease or infection which can be naturally transmitted from vertebrate animals to humans (plural: zoonoses; adjective: zoonotic).

Zoosanitary: Relating to the health of animals and the prevention of spreading animal diseases through cleaning and containment practices.

Zoospore: A motile asexual spore that uses a flagellum for locomotion

Appendix IV
OIE Member Countries (as of March 2012)

Afghanistan
Albania
Algeria
Andorra
Angola
Argentina
Armenia
Australia
Austria
Azerbaijan
Bahamas
Bahrain
Bangladesh
Barbados
Belarus
Belgium
Belize
Benin
Bhutan
Bolivia
Bosnia and Herzegovina
Botswana
Brazil
Brunei
Bulgaria
Burkina Faso
Burundi
Cambodia
Cameroon
Canada
Cape Verde
Central African Rep.
Chad
Chile
China (People's Rep. of)
Colombia
Comoros
Congo
Congo (Dem. Rep. of the)
Costa Rica
Cote d'Ivoire
Croatia
Cuba
Cyprus
Czech Republic
Denmark
Djibouti
Dominican (Rep.)
Ecuador
Egypt
El Salvador
Equatorial Guinea
Eritrea
Estonia
Ethiopia
Fiji
Finland
Former Yug. Rep. of Macedonia
France
Gabon
Gambia
Georgia
Germany
Ghana
Greece
Guatemala
Guinea
Guinea Bissau
Guyana
Haiti
Honduras
Hungary
Iceland
India
Indonesia
Iran
Iraq
Ireland
Israel
Italy
Jamaica
Japan
Jordan
Kazakhstan
Kenya
Korea (Dem. People's Rep. of)
Korea (Rep. of)
Kuwait
Kyrgyzstan
Laos
Latvia
Lebanon
Lesotho
Libya
Liechtenstein
Lithuania
Luxembourg
Madagascar
Malawi
Malaysia
Maldives
Mali
Malta
Mauritania
Mauritius
Mexico
Micronesia Federated States of
Moldova
Mongolia
Montenegro
Morocco
Mozambique
Myanmar
Namibia
Nepal
Netherlands
New Caledonia
New Zealand
Nicaragua
Niger
Nigeria
Norway
Oman
Pakistan
Panama
Papua New Guinea
Paraguay
Peru
Philippines
Poland
Portugal
Qatar
Romania
Russia
Rwanda
San Marino
Sao Tome and Principe
Saudi Arabia
Senegal
Serbia
Seychelles
Sierra Leone
Singapore
Slovakia
Slovenia
Somalia
South Africa
Spain
Sri Lanka
Sudan
Suriname
Swaziland
Sweden
Switzerland
Syria
Taipei Chinese
Tajikistan
Tanzania
Thailand
Timor-Leste
Togo
Trinidad and Tobago
Tunisia
Turkey
Turkmenistan
Uganda
Ukraine
United Arab Emirates
United Kingdom
United States of America
Uruguay
Uzbekistan
Vanuatu
Venezuela
Vietnam
Yemen
Zambia
Zimbabwe

Appendix V
OIE Listed Diseases (2012)

Source: http://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2012/ (Accessed March 2012).

Multiple species diseases

- Anthrax
- Aujeszky's disease
- Bluetongue
- Brucellosis (*Brucella abortus*)
- Brucellosis (*Brucella melitensis*)
- Brucellosis (*Brucella suis*)
- Crimean Congo haemorrhagic fever
- Echinococcosis/hydatidosis
- Epizootic haemorrhagic disease
- Equine encephalomyelitis (Eastern)
- Foot and mouth disease
- Heartwater
- Japanese encephalitis
- Leptospirosis
- New world screwworm (*Cochliomyia hominivorax*)
- Old world screwworm (*Chrysomya bezziana*)
- Paratuberculosis
- Q fever
- Rabies
- Rift Valley fever
- Rinderpest
- Surra (*Trypanosoma evansi*)
- Trichinellosis
- Tularemia
- Vesicular stomatitis
- West Nile fever

Cattle diseases

- Bovine anaplasmosis
- Bovine babesiosis
- Bovine genital campylobacteriosis
- Bovine spongiform encephalopathy
- Bovine tuberculosis
- Bovine viral diarrhoea
- Contagious bovine pleuropneumonia
- Enzootic bovine leukosis
- Haemorrhagic septicaemia
- Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis
- Lumpky skin disease
- Theileriosis
- Trichomonosis
- Trypanosomosis (tsetse-transmitted)

Swine diseases

- African swine fever
- Classical swine fever
- Nipah virus encephalitis
- Porcine cysticercosis
- Porcine reproductive and respiratory syndrome
- Swine vesicular disease
- Transmissible gastroenteritis

Sheep and goat diseases

- Caprine arthritis/encephalitis
- Contagious agalactia
- Contagious caprine pleuropneumonia
- Enzootic abortion of ewes (ovine chlamydiosis)
- Maedi-visna
- Nairobi sheep disease
- Ovine epididymitis (*Brucella ovis*)
- Peste des petits ruminants
- Salmonellosis (*S. abortusovis*)
- Scrapie
- Sheep pox and goat pox

Equine diseases

- African horse sickness
- Contagious equine metritis
- Dourine
- Equine encephalomyelitis (Western)
- Equine infectious anaemia
- Equine influenza
- Equine piroplasmosis
- Equine rhinopneumonitis
- Equine viral arteritis
- Glanders
- Venezuelan equine encephalomyelitis

Amphibian diseases

- Infection with *Batrachochytrium dendrobatidis*
- Infection with ranavirus

Mollusc diseases

- Infection with abalone herpes-like virus
- Infection with *Bonamia exitiosa*
- Infection with *Bonamia ostreae*
- Infection with *Marteilia refringens*
- Infection with *Perkinsus marinus*
- Infection with *Perkinsus olseni*
- Infection with *Xenohaliotis californiensis*

Avian diseases

- Avian chlamydiosis
- Avian infectious bronchitis
- Avian infectious laryngotracheitis
- Avian mycoplasmosis (*M. gallisepticum*)
- Avian mycoplasmosis (*M. synoviae*)
- Duck virus hepatitis
- Fowl typhoid
- Highly pathogenic avian influenza and low pathogenic avian influenza in poultry as per Chapter 10.4. of the *Terrestrial Animal Health Code*
- Infectious bursal disease (Gumboro disease)
- Newcastle disease
- Pullorum disease
- Turkey rhinotracheitis

Crustacean diseases

- Crayfish plague (*Aphanomyces astaci*)
- Infectious hypodermal and haematopoietic necrosis
- Infectious myonecrosis
- Necrotising hepatopancreatitis
- Taura syndrome
- White spot disease
- White tail disease
- Yellowhead disease

Bee diseases

- Acarapisosis of honey bees
- American foulbrood of honey bees
- European foulbrood of honey bees
- Small hive beetle infestation (*Aethina tumida*)
- *Tropilaelaps* infestation of honey bees
- Varroosis of honey bees

Fish diseases

- Epizootic haematopoietic necrosis
- Epizootic ulcerative syndrome
- Gyrodactylosis (Infection with *Gyrodactylus salaris*)
- Infectious haematopoietic necrosis
- Infectious salmon anaemia
- Koi herpesvirus disease
- Red sea bream iridoviral disease
- Spring viraemia of carp
- Viral haemorrhagic septicaemia

Lagomorph diseases

- Myxomatosis
- Rabbit haemorrhagic disease

Other diseases

- Camelpox
- Leishmaniosis

Appendix VI
Outputs of disease prioritisation exercise

Disease *Factsheets produced for diseases in **bold***	**Relevance to wetlands**	**Importance to** Wildlife	Livestock	Human health	Livelihoods	**Weighted sum**
Oyster diseases	**5**	5	5	1	5	61
Tick borne diseases	**4**	5	5	5	5	60
Epizootic ulcerative syndrome	**5**	5	5	0	5	60
Crayfish plague	**5**	5	?	0	4	?
Avian influenza	**5**	4	5	4	5	59
Avian cholera	**5**	5	5	0	3	58
Coral diseases	**5**	5	2	0	5	57
Inclusion body disease (fish)	**5**	4	5	0	5	55
Bovine tuberculosis	**3**	5	5	5	5	55
Harmful algal blooms	**5**	4	0	4	5	54
Salmon and trout sea lice	5	4	4	0	4	53
Trematodes (fish)	**5**	3	4	4	4	52
Pesticides	4	5	1	4	2	52
Heavy metals (other than lead)	4	5	1	4	2	52
Lead poisoning	**4**	5	1	4	1	51
Amphibian chytridiomycosis	**5**	5	1	0	0	51
Rift Valley fever	**4**	3	5	5	5	50
African animal trypanosomiasis	**3**	4	5	5	5	50
Ranavirus infection	**5**	5	0	0	0	50
Avian tuberculosis	**4**	4	4	1	4	49
Avian botulism	**5**	4	2	0	2	49
Anthrax	**2**	5	5	4	4	48
Duck virus enteritis	**5**	3	3	0	3	46
West Nile virus disease	**3**	4	4	4	3	46
Peste des petits ruminants	**3**	4	5	0	5	45
Foot and mouth disease	2	5	5	0	5	45
African swine fever	2	5	5	0	5	45
Classical swine fever	2	5	5	0	4	44
Salmonellosis	**4**	2	5	5	3	43
Brucellosis	**3**	3	5	4	4	43
Rabies	2	5	2	5	1	43
Eastern equine encephalitis	3	3	3	4	3	40
Venezuelan equine encephalitis	3	3	3	4	3	40
Newcastle Disease	3	3	5	0	5	40
Rinderpest	1	5	5	0	5	40
Schistosomiasis	**5**	1	0	5	4	39
Western equine encephalitis	3	3	3	3	3	39
***Escherichia coli* poisoning**	**3**	2	5	5	3	38
Campylobacteriosis	**3**	2	5	5	3	38
Trematodes (various)	4	2	4	0	4	38
Vibrio.	3	3	5	1	2	38

Disease *Factsheets produced for diseases in **bold***	**Relevance to wetlands**	**Importance to**				**Weighted sum**
		Wildlife	**Livestock**	**Human health**	**Livelihoods**	
Pasteurellosis	3	3	3	1	4	38
Leptospirosis	**3**	2	4	4	4	37
Blue tongue	3	2	5	0	5	35
Japanese encephalitis	3	1	3	4	3	30
Tularaemia	3	1	3	4	3	30
Avian malaria	3	4	0	0	0	35
Leishmaniasis	3	1	0	4	3	27
Necrotic enteritis	3	3	0	0	0	30
African horse sickness	3	0	4	0	3	22
Yellow fever	3	0	0	3	2	20
Chikungunya	3	?	?	3	2	?
Inclusion body disease (birds)	3	3	?	0	0	?

Appendix VII
Summary of impacts of diseases on wildlife

Disease name	Causative agent						Region	Comments	OIE notifiable disease
African animal trypanosomosis	Protozoan trypanosomes						Endemic in most of Africa. Occurs where the tsetse fly vector exists.	Primarily affects domestic mammals. Most wild mammals are trypanotolerant. Mainly spread by the tsetse fly.	N
Amphibian chytridiomycosis	The fungus *Batrachochytrium dendrobatidis*						All continents except Antarctica.	Affects most species of amphibian and is a major cause of amphibian mortality and morbidity.	N
Anthrax	The bacterium *Bacillus anthracis*						Worldwide. Endemic in southern Europe, parts of Africa, Australia, Asia and North and South America.	Spores may remain dormant and viable for decades. An acute infectious disease, can affect almost all species of mammals, including humans.	N
Avian botulism	The bacterium *Clostridium botulinum*						Worldwide.	Affects birds and some mammals. Caused by ingestion of a toxin produced by *C. botulinum*.	—
Avian cholera	The bacterium *Pasteurella multocida*						Mainly North America. Also occurs in South America, Africa, Asia, Europe and Oceania.	Most commonly affects ducks, geese, swans, shore birds, coots, gulls and crows.	—
Avian influenza	*Influenzavirus* A subtypes						Since 1997, highly pathogenic AI (subtype H5N1) has been reported in S.E. Asia, Europe, Africa and the Middle East.	HPAI H5N1 is the cause of unprecedented AI-related mortality. Has both direct and indirect conservation consequences.	N
Avian tuberculosis	The bacterium *Mycobacterium avium*						Worldwide.	Most commonly reported in wild waterbirds, gregarious birds, raptors and scavengers. Clinical manifestation in mammals is rare.	—
Bovine tuberculosis	The bacterium *Mycobacterium bovis*						Worldwide. Widespread in Africa, parts of Asia and some Middle Eastern countries.	Cattle are considered the true hosts of *M. bovis*; responsible for elevated mortality and morbidity in wild mammals in some protected areas.	N

Disease name	Causative agent						Region	Comments	OIE notifiable disease
Brucellosis	Bacteria of the genus *Brucella*						Worldwide. High risk areas include: the Mediterranean Basin, South and Central America, Eastern Europe, Asia, Africa .	Particularly affects cattle, swine, goats, sheep but also wild bison, elk, deer, other ruminants. Infection can cause reproductive losses.	N
Campylo-bacteriosis	Bacteria in the genus *Campylobacter*						Worldwide.	Infection in wild birds and mammals often inapparent.	—
Coral diseases	Various						Reported in marine ecosystems worldwide.	Responsible for considerable ecological damage, affecting numerous species of coral (primarily the soft corals or true stony corals).	—
Crayfish plague	Oomycete *Aphanomyces astaci*						Widespread in Europe and North America.	All species of freshwater crayfish are considered susceptible to infection, European species have declined due to novel infection.	N
Duck virus enteritis	Herpesvirus						Reported in North America, Asia and several countries in Europe.	Can cause high seasonal mortality in ducks, geese and swans	—
Epizootic ulcerative syndrome (EUS)	Oomycetes *Aphanomyces Invadans/ piscidida*						Worldwide distribution. Affects 25 countries in four continents: southern Africa, Asia, Australia and North America.	Affects wild and farmed, fresh- and brackish-water fish.	N
Escherichia coli poisoning	Strains of the bacterium *Escherichia coli*						Worldwide.	Direct release of raw sewage is a frequent source. Often inapparent in wild animals. Certain strains (O157) can cause severe disease in humans.	—
Harmful algal blooms	Toxic species of algae						Worldwide.	Occur in both saltwater and freshwater environments, particularly where there are high nutrient levels, causing high levels of mortality.	—
Lead poisoning	Toxic lead						Occurs globally and in any wetland where lead is deposited.	Particularly affects waterbirds, birds of prey, and mammals.	—

Disease name	Causative agent						Region	Comments	OIE notifiable disease
Leptospirosis	Bacteria from the genus *Leptospira*						Worldwide. Most common in temperate or tropical climates with high rainfall.	Causes infections in many terrestrial and marine mammals. Commonly affects domestic animals and humans.	N
Oyster diseases	Various						Worldwide.	Can affect wild populations of oysters and also commercial setups. Oysters grown in contaminated areas can cause human disease.	N
Peste des petits ruminants (PPR)	Peste des petits ruminants virus						Considered endemic across North Africa, China and parts of the Far East.	Predominantly affects sheep and goats causing very high mortality, less severe in wildlife.	N
Ranavirus infection	Ranaviruses						Reported in the Americas, Asia, Pacific and Europe.	Significant effects on amphibians (including salamanders, toads and frogs).	N
Rift Valley fever	Rift Valley fever *Phlebovirus*						Endemic in tropical regions of Eastern and Southern Africa. Cases also reported in Saudi Arabia and Yemen.	A vector-borne disease, commonly transmitted by mosquitoes. Affects most terrestrial mammals; predominantly sheep, cattle and wild ruminants.	N
Salmonellosis	Types of *Salmonella* bacteria						Worldwide.	Affects many domestic and wild animals including birds, reptiles, amphibians, fish and invertebrates.	*S. abortus ovis* only
Schistosomiasis	Schistosomes (trematode worms)						Most commonly found in Asia, Africa and South America in areas where the water contains freshwater snails.	Affects many species of wild animals and wildfowl, however, humans and livestock are the most at risk of clinical disease.	—
Tick-borne diseases	Variety of pathogens						As a collective TBDs occur worldwide. Usually in foci with suitable conditions for ticks and with susceptible animal hosts.	Ticks often found in grassy, wooded habitat. TBDs can affect most mammals and birds; primarily livestock, humans and companion animals.	Some TBDs are OIE listed
Trematode Infection of fish	Trematodes (flatworms / flukes)						Worldwide.	Trematodes can parasitise many vertebrate species. Commonly fish, frogs, livestock, domestic animals, humans and some invertebrates.	—

Disease name	Causative agent						Region	Comments	OIE notifiable disease
West Nile virus disease	West Nile *Flavivirus*						Reported in Africa, Europe, the Middle East, west and central Asia, Oceania and most recently, North America.	Spread by insect vectors (primarily mosquito). Affects numerous bird species and some terrestrial mammals (including humans).	N

KEY

Taxa symbols	
	Invertebrates Animals without backbones – all animals except fish, amphibians, reptiles, birds and mammals. Includes corals, molluscs, insects, crustacea *etc*.
	Fish A group of taxa, including hagfish, lampreys, sharks and rays, ray-finned fish, bony fish, coelacanths and lungfish.
	Amphibians and reptiles (together known as herpetafauna) Animals from the classes Amphibia (such as frogs, salamanders and caecilians) and Reptilia (such as crocodiles, lizards and turtles).
	Birds Animals from the class Aves.
	Mammals Animals from the class Mammalia.

Impact colours			
	Severe impact		**Mild impact**
	Moderate impact		**No impact**

Appendix VIII
Technical editors of the disease fact sheets

Disease fact sheet	Technical editor	Affiliation
African animal trypanosomiasis	Richard Kock	The Royal Veterinary College, UK
Amphibian chytridiomycosis	Andrew Cunningham	Institute of Zoology, Zoological Society of London, UK
Anthrax	Richard Kock	Royal Veterinary College, UK
Avian botulism	Paul Duff	Animal Health and Veterinary Laboratories Agency, UK
Avian cholera	Richard Botzler	Humboldt State University, USA
Avian influenza	Richard Irvine	Animal Health and Veterinary Laboratories Agency, UK
Avian tuberculosis	Alexandra Tomlinson	Food and Environment Research Agency, UK
Bovine tuberculosis	Alexandra Tomlinson	Food and Environment Research Agency, UK
Brucellosis	Lindsey McCrickard	Food and Agriculture Organisation of the United Nations, Italy
Campylobacteriosis	Richard Kock	Royal Veterinary College, UK
Coral disease	-	-
Crayfish plague	Birgit Oidtmann	Centre for Environment, Fisheries & Aquaculture Science, UK
Duck virus enteritis	Paul Holmes	Animal Health and Veterinary Laboratories Agency, UK
Epizootic ulcerative syndrome (EUS)	Birgit Oidtmann	Centre for Environment, Fisheries & Aquaculture Science, UK
***Escherichia coli* poisoning**	Richard Kock	Royal Veterinary College, UK
Harmful algal blooms	Henrik Enevoldsen	Intergovernmental Oceanographic Commission of UNESCO, Denmark
Lead poisoning	Paul Holmes	Animal Health and Veterinary Laboratories Agency, UK
Leptospriosis	Alexandra Tomlinson	Food and Environment Research Agency, UK
Oyster diseases	*FAO Aquaculture Service*	
Peste des petits ruminants	Ashley Banyard	Animal Health and Veterinary Laboratories Agency, UK
Ranavirus infection	Andrew Cunningham	Institute of Zoology, Zoological Society of London, UK
Rift Valley fever	Daniel Horton	Animal Health and Veterinary Laboratories Agency, UK
Salmonellosis	Becki Lawson	Institute of Zoology, Zoological Society of London, UK
Schistosomiasis	Paul Phipps	Animal Health and Veterinary Laboratories Agency, UK
Tick-borne diseases	Daniel Horton	Animal Health and Veterinary Laboratories Agency, UK
Trematodes of fish	*FAO Aquaculture Service*	
West Nile virus disease	Daniel Horton	Animal Health and Veterinary Laboratories Agency, UK

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