

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.

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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 managers, 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 schistosomes 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

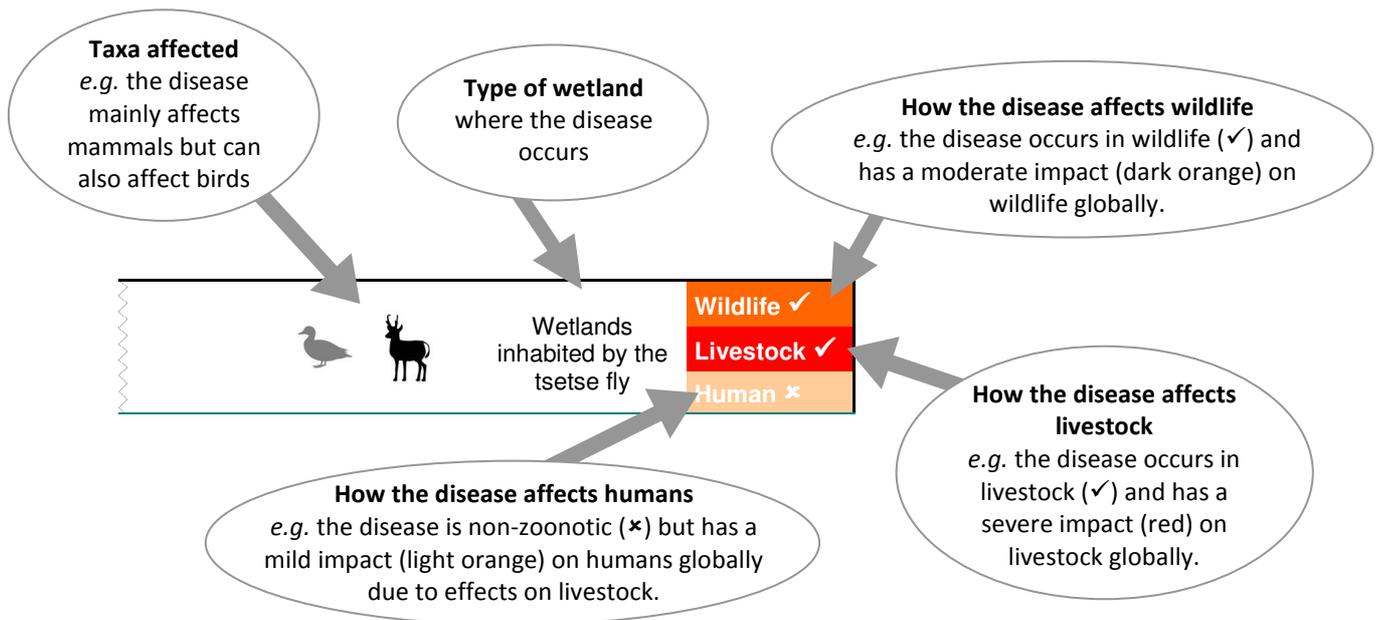
The factsheets are divided into eight sections:

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	
	<p>Invertebrates</p> <p>Animals without backbones – all animals except fish, amphibians, reptiles, birds and mammals. Includes corals, molluscs, insects, crustacea etc.</p>
	<p>Fish</p> <p>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.</p>
	<p>Amphibians and reptiles (together known as herpetofauna)</p> <p>Animals from the classes Amphibia (such as frogs, salamanders and caecilians) and Reptilia (such as crocodiles, lizards and turtles).</p>
	<p>Birds</p> <p>Animals from the class Aves.</p>
	<p>Mammals</p> <p>Animals from the class Mammalia. Includes humans.</p>

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 ✓



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

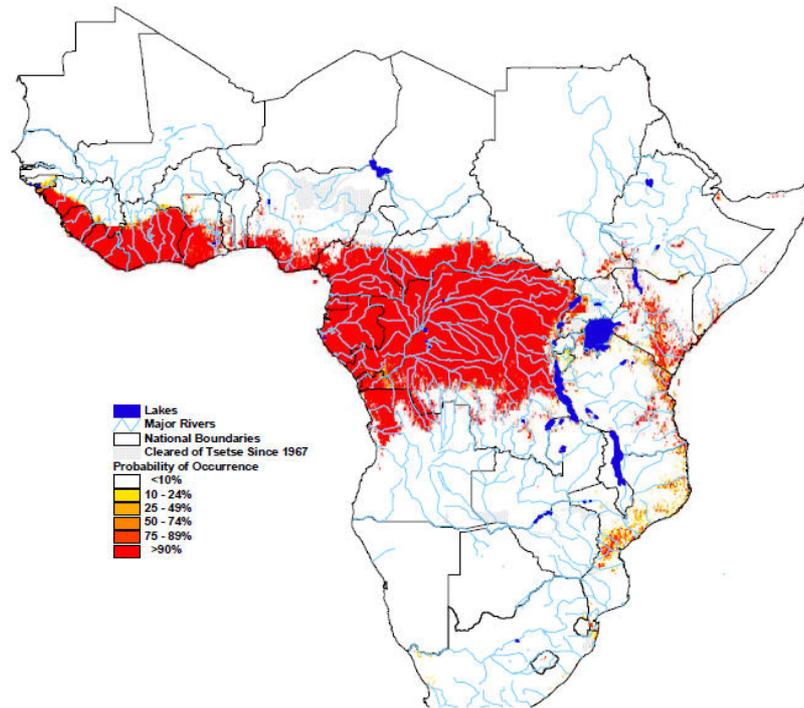
Trypanosomes, protozoan parasites of the genus *Trypanosoma* that live in the blood, lymph and various tissues of vertebrate hosts. The most important species for this disease are *Trypanosoma congolense*, *T. vivax* and *T. brucei subsp. brucei* and *rhodesiensi*.

Species affected

Many species of domestic and wild animals including cattle, swine, camels, goats and sheep. Cattle are preferred 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. fuscica*, 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 carcass 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°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

- ☐ Woodhams, D.C., Bosch, J., Briggs, C.J., Cashins, S., Davis, L.R., Lauer, A., Muths, E., Puschendorf, R., Schmidt, B.R., Sheafor, B. & Voyles, J. (2011). **Mitigating amphibian disease: strategies to maintain wild populations and control chytridiomycosis.** *Frontiers in Zoology*, 8, 8. www.frontiersinzoology.com/content/pdf/1742-9994-8-8.pdf [Accessed March 2012].
- ☐ World Organisation for Animal Health (OIE). **Disease information card – infection with *Batrachochytrium dendrobatidis*.** http://www.oie.int/fileadmin/Home/eng/International_Standard_Setting/docs/pdf/Chytridio_card-final.pdf [Accessed March 2012].
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- ☞ Amphibiaweb. **An overview of chytridiomycosis.** amphibiaweb.org/chytrid/chytridiomycosis.html [Accessed March 2012].
- ☞ European Association of Zoos and Aquaria. **EAZWV transmissible disease fact sheet No. 130.** [http://www.eaza.net/activities/tdfactsheets/130%20Chytridiomycosis%20\(Amphibian\).doc.pdf](http://www.eaza.net/activities/tdfactsheets/130%20Chytridiomycosis%20(Amphibian).doc.pdf) [Accessed March 2012].

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 ✓



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 carcass 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 carcasses contaminated with anthrax bacterial spores.

Wild carnivores and scavengers become infected through the consumption of infected meat.

After feeding on an infected carcass, 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 acquired 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 consciousness 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. Unclothed blood issuing from body orifices, rapid decomposition of the carcass 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 carcasses reduces contamination sources. Burn all anthrax-infected carcasses or bury in deep lime pits. When this is not possible, place the unopened carcasses 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 carcass is effectively sterilised under these conditions. Carcasses 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 carcasses by *e.g.* burning on site.
- Scavengers should be kept away from carcasses by reducing access to carcasses *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 carcasses 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

- ☐ Clegg, S. (2006). Wildlife Anthrax Epizootic Workshop Working Group. **Preparedness for anthrax epizootics in wildlife areas** [conference summary]. *Emerging Infectious Diseases*, 12. http://wwwnc.cdc.gov/eid/article/12/7/06-0458_article.htm [Accessed March 2012].
- ☐ Hugh-Jones, M. E. & de Vos, V. (2002). **Anthrax and wildlife**. *Revue Scientifique et Technique de l'Office International des Epizooties*, 21 (2): 359-383. www.catsg.org/cheetah/05_library/5_3_publications/H/Hugh-Jones_&_de_Vos_2002_Anthrax_in_wildlife.pdf [Accessed March 2012].
- ☐ World Organisation for Animal Health (OIE). **Terrestrial animal health code. Chapter 8.1 : Anthrax**. http://www.oie.int/fileadmin/Home/eng/Health_standards/tahc/2010/en_chapitre_1.8.1.pdf [Accessed March 2012].
- ☐ International Union for Conservation of Nature (IUCN). **Frequently asked questions (FAQs) on anthrax for wildlife managers (2006)**. www.iucn-vsg.org/documents/anthrax.pdf [Accessed March 2012].
- ☐ Center for Food Security & Public Health (CFSPH). **Anthrax**. <http://www.cfsph.iastate.edu/Factsheets/pdfs/anthrax.pdf> [Accessed March 2012].
- ☑ World Health Organization (WHO). **Anthrax**. www.who.int/csr/disease/Anthrax/en/index.html [Accessed March 2012].
- ☑ Center for Biosecurity. **Bacillus anthracis (anthrax)**. www.upmc-biosecurity.org/website/focus/agents_diseases/fact_sheets/anthrax.html [Accessed March 2012].
- ☑ Centre for Disease Control and prevention (CDC). **Anthrax**. www.bt.cdc.gov/agent/anthrax [Accessed March 2012].
- ☑ Food and Agricultural Organisation (FAO). **Anthrax in animals**. www.fao.org/ag/magazine/0112sp.htm [Accessed March 2012].
- ☑ Wildpro. **Anthrax**. <http://wildpro.twycrosszoo.org/S/00dis/Bacterial/anthrax.htm> [Accessed March 2012].

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 <i>Clostridium botulinum</i> . 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 <i>Clostridium botulinum</i> . 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 <i>Clostridium botulinum</i> 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 (<i>e.g.</i> maggots) and birds (see below for details of carcass/maggot cycle) and by transfer of infected carcasses 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 carcass/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 carcasses 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 carcasses coinciding with receding water levels may signal an outbreak. Healthy, sick and dead birds are often found together during an outbreak with carcasses 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 carcasses 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 carcasses**.
- 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 carcasses and their disposal by burial or burning, especially during outbreaks, removes nutrient sources for bacteria.

- Immediately place carcasses into two plastic bags to prevent leakage of fluids. Bags should always be securely closed before they are removed from the area.
- Submit carcasses to disease diagnostic laboratories before being incinerated.
- Take care to avoid contaminating new areas whilst carcasses 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. Carcasses 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 carcasses.
- 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

- ☞ Friend, M. & Franson, J.C. (2001). **Avian botulism**. In: Field manual of wildlife diseases: general field procedures and diseases of birds. E. A. Ciganovich (ed.). pp. 271-281. U.S. Department of the Interior and U.S. Geological Survey, Washington, DC.
www.nwhc.usgs.gov/publications/field_manual/chapter_38.pdf [Accessed March 2012].
- ☞ World Organisation for Animal Health (OIE). **Botulism factsheet**.
www.cfsph.iastate.edu/Factsheets/pdfs/botulism.pdf [Accessed March 2012].
- ☞ World Health Organization (WHO). **Botulism factsheet**.
www.who.int/mediacentre/factsheets/fs270/en/ [Accessed March 2012].
- ☞ Prairie Habitat Joint Venture. **Ecology and management of avian botulism on the Canadian prairies**.
http://www.phjv.ca/pdf/BotulismReport_FINAL_FullReport_Aug2011.pdf [Accessed March 2012].
- ☞ U.S Geological Survey (USGS) National Wildlife Health Center. **Avian botulism**.
www.nwhc.usgs.gov/disease_information/avian_botulism/index.jsp [Accessed March 2012].
- ☞ Wildpro. **Avian botulism**.
<http://wildpro.twycrosszoo.org/S/00dis/toxic/Biotoxin/botulism.htm> [Accessed March 2012].

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 <i>Pasteurella multocida</i> .
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 (<i>e.g.</i> infected carcasses) 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 carcasses or contaminated environments. Bacteria are released into the environment by dead and dying birds, by live birds carrying the disease or from contaminated objects (<i>e.g.</i> 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 (<i>e.g.</i> 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 carcass 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 carcasses 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 carcasses to disease diagnostic laboratories before being

incinerated.

- Remove carcasses 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 carcasses 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 carcasses.
- 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 carcasses 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

- ☐ Blanchong, J.A., Samuel, M.D., Goldberg, D.R., Shaddock, D.J. & Creekmore, L.H. (2006). **Wetland environmental conditions associated with the risk of avian cholera outbreaks and the abundance of *Pasteurella multocida***. *The Journal of Wildlife Management*, 70 (1): 54-60.
- ☐ 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.
- ☐ Friend, M. & Franson, J.C. (2001). **Avian cholera**. In: Field manual of wildlife diseases: general field procedures and diseases of birds. E. A. Ciganovich (ed.). pp. 75-92. U.S. Department of the Interior and U.S. Geological Survey, Washington, DC. http://www.nwhc.usgs.gov/publications/field_manual/chapter_7.pdf [Accessed March 2012].
- ☞ U.S Geological Survey (USGS) National Wildlife Health Center. **Avian cholera**. www.nwhc.usgs.gov/disease_information/avian_cholera/index.jsp [Accessed March 2012].
- ☞ Wildpro. **Avian cholera**. http://wildpro.twycrosszoo.org/S/00dis/Bacterial/Avian_Cholera.htm [Accessed March 2012].

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 productivity.

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.

**Surveillance
for Avian Influenza**

In this area of Scotland, dead birds are being sampled for a survey of the incidence of influenza viruses in wildfowl.

If you see **dead ducks, geese, swans, gulls or waders**, you can report them, to the:

GB Wild Bird Helpline 08459 33 55 77
(choose the 'avian influenza' option)

Please note that some, but not all, birds reported will be collected for testing. You will not be asked to pick up the birds. Thank you for your help with the survey.

More information about the survey, which is being co-ordinated by the **Scottish Government** and **Scottish Natural Heritage** as part of a GB-wide surveillance programme, can be seen on the web:

<http://www.scotland.gov.uk/avianinfluenza>

The survey areas are: Angus, Fife, Clackmannanshire, Dundee City, Falkirk, West Lothian, City of Edinburgh, Midlothian, East Lothian and the Scottish Borders.




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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- 📄 Friend, M. & Franson, J.C. (2001). **Avian influenza.** 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. http://www.nwhc.usgs.gov/publications/field_manual/chapter_22.pdf. [Accessed March 2012].
- 📄 Food and Agriculture Organization (FAO). **Wild bird highly pathogenic avian influenza surveillance.** <http://www.fao.org/docs/eims/upload/218650/a0960e.pdf> [Accessed March 2012].
- 📄 Food and Agriculture Organization (FAO). **Wild birds and avian influenza.** <ftp://ftp.fao.org/docrep/fao/010/a1521e/a1521e.pdf>. [Accessed March 2012].
- 📄 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.

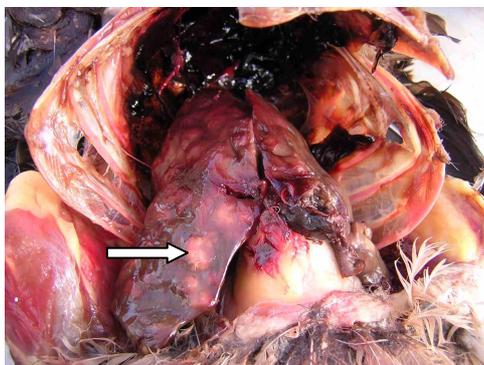
In pigs, there are generally no obvious signs of disease with evidence of infection being found at slaughter in either or both the lymph nodes around the neck or those draining the intestine.

Recommended action if suspected

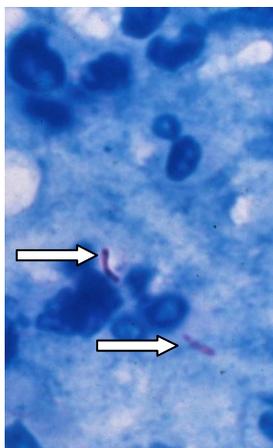
The disease is not notifiable to the OIE but prevention of establishment of the disease is highly desirable as control thereafter is complex and often unsuccessful. ► **Prevention and control in wetlands below.**

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 carcass 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].
- ☐ World Organisation of Animal Health (OIE). **Chapter 2.03.06: Avian tuberculosis**. Manual of diagnostic tests and vaccines for terrestrial animals. www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.03.06_AVIAN_TB.pdf [Accessed March 2012].
- ☐ Tell, L.A., Woods, L. & Cromie R.L. (2001). **Mycobacteriosis in birds**. *Revue Scientifique et Technique de l'Office International des Epizooties*, 20, 180–203.
- ☐ Thorel, M.-F., Huchzermeyer, H.F. & Michel, A.L. (2001). **Mycobacterium avium and Mycobacterium intracellulare infection in mammals**. *Revue Scientifique et Technique de l'Office International des Epizooties*, 20, 204-218.
- ☐ 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**
- ☐ Cousins, D. V & Florisson, N. (2005). **A review of tests available for use in the diagnosis of tuberculosis in non-bovine species.** *Revue Scientifique et Technique de l'Office International des Épizooties*, 24 (3): 1039-1059
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www.oie.int/fileadmin/Home/eng/Media_Center/docs/pdf/Disease_cards/BOVINE-TB-EN.pdf. [Accessed May 2011].
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 - ☐ World Organisation for Animal Health (OIE). **Terrestrial animal health code.**
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 - ☐ Merck & Co. Inc. **The Merck veterinary manual: tuberculosis.**
<http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/204400.htm&word=tuberculosis>. [Accessed May 2011].
 - ☐ Michigan Bovine Tuberculosis Eradication Project.
www.michigan.gov/emergingdiseases/0,1607,7-186-25804-74719--,00.html
 - ☐ Public Health Agency of Canada. **Pathogen safety data sheets.**
www.phac-aspc.gc.ca/msds-ftss/index.html

Brucellosis



Wetlands supporting
groups of susceptible
mammals

Wildlife ✓

Livestock ✓

Human ✓



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 (<i>e.g.</i> 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 <i>Brucella</i> , infections are mainly caused by <i>B. abortus</i> , <i>B. melitensis</i> , <i>B. ovis</i> , <i>B. canis</i> , and <i>B. suis</i> .
Species affected	Many species of terrestrial and marine mammals, particularly cattle, swine, bison, elk <i>Cervus canadensis</i> , 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 susceptible 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 (<i>e.g.</i> 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, <i>e.g.</i> 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 occurred through 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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- 📄 Nicoletti, P. (1984). **The control of brucellosis in tropical and subtropical regions.** *Preventive Veterinary Medicine*, 2, 193-196.
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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 carcasses 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].
- ☐ The Center for Food Security and Public Health (CFSPH)/ World Organisation for Animal Health (OIE). **Factsheet: campylobacteriosis**. <http://www.cfsph.iastate.edu/Factsheets/pdfs/campylobacteriosis.pdf> [Accessed March 2012].
- ☐ Jones, K. (2001). **Campylobacters in water, sewage and the environment**. *Journal of Applied Microbiology*, 90 (6): 68-79. www.bvsde.paho.org/bvsacd/cd29/campylob.pdf. [Accessed March 2012].
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- ☐ 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].
- ☐ World Health Organization (WHO). ***Campylobacter***. www.who.int/topics/campylobacter/en [Accessed March 2012].
- ☐ Centre for Disease Control and Prevention (CDC). ***Campylobacter***. www.cdc.gov/nczved/divisions/dfbmd/diseases/campylobacter [Accessed March 2012].
- ☐ Wildpro. ***Campylobacter* infection**. http://wildpro.twycrosszoo.org/S/00dis/Bacterial/Campylobacter_Infection.htm [Accessed March 2012].
- ✉ **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

Contacts



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 (<i>e.g.</i> 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 (<i>e.g.</i> fish predation, gastropod predation, galls, algal abrasion/overgrowth, Crown of Thorns Starfish predation, sediment damage <i>etc.</i>). Also record lesion type (<i>e.g.</i> 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 <i>e.g.</i> discrete or diffuse). Develop a monitoring programme to help address impacts of disease on coral communities (<i>e.g.</i> 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	<p>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.</p> <p>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.</p> <p>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.</p>

PREVENTION AND CONTROL IN WETLANDS

Environment	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Ensure that divers collecting samples or visiting sites always visit healthy sites before those considered to be diseased.</p> <p>All samples should be placed in double containment and divers should</p>
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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].
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- ☐ 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].
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- ☐ 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 aphanomyiasis, Kraftpest, Krebspest, la peste

KEY FACTS

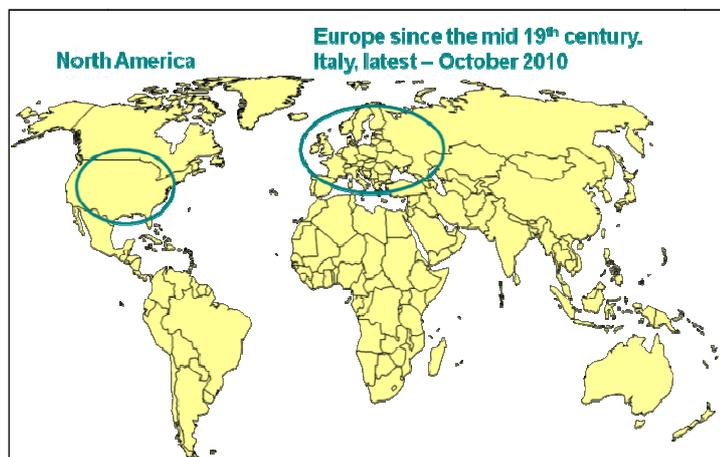
What is Crayfish Plague? Crayfish plague is a disease caused by an oomycete (water mould) that affects wild and farmed freshwater crayfish. The disease can cause large scale mortality.

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 be 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 highly 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].

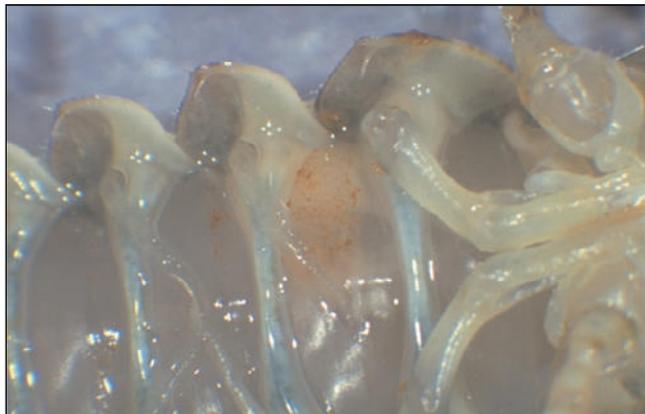
Contacts

- ☒ 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 <i>via</i> exposure to contaminated objects/environments – see below.
How is the disease transmitted to animals?	Spread is by bird to bird contact or <i>via</i> 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 carcasses 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 carcasses should be incinerated and the area used to process the carcasses 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 carcasses** by incineration should be meticulous and systematic. Personnel and equipment associated with carcass 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 carcasses.

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 carcasses** 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.
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- ☐ 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 ✗



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.

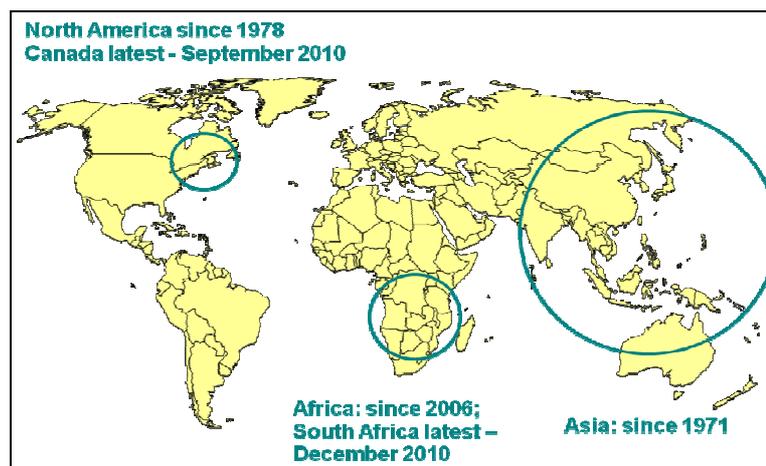
Species affected

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 *Barbus 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	<p>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.</p> <p>A number of simple biosecurity measures can minimise or prevent the spread of EUS. These include:</p> <ul style="list-style-type: none">▪ 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:<ul style="list-style-type: none">- Good farm hygiene (<i>e.g.</i> handwashing between tanks, separation of nets/tanks/stocks, regular and correct disinfection procedures, <i>etc.</i>)- 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, <i>etc.</i>).▪ 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.
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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

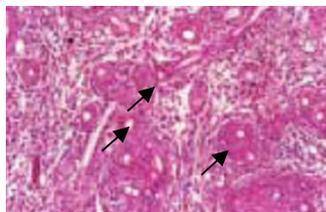
Useful publications and websites

- ☐ 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*, colibacillosis, 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

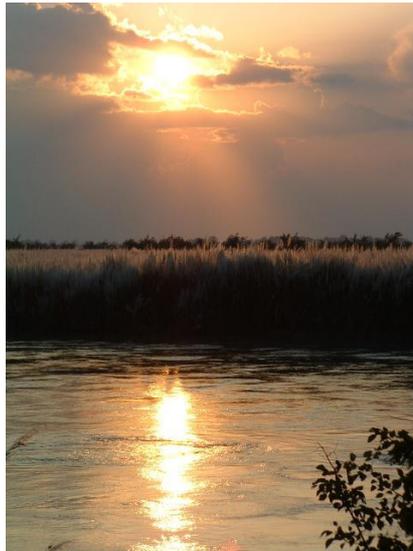
Vector(s)	<i>E. coli</i> is not vector-borne although some mechanical transfer from contaminated areas is possible.
How is the disease transmitted to animals?	Animals (livestock in particular) become infected with <i>E. coli</i> by exposure to items including food, water and inanimate objects (fomites) 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>i.e.</i> 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 <i>E. coli</i> from contaminated food (<i>e.g.</i> undercooked ground beef) or unpasteurised milk or contact with animal faeces from the environment. Animals do not have to be ill to transmit <i>E. coli</i> , including <i>E. coli</i> O157, to humans.

IDENTIFICATION AND RESPONSE

Field signs	<p>Signs of <i>E. coli</i> 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.</p> <p>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.</p>
Recommended action if suspected	Alert the relevant authorities of any suspected cases.
Diagnosis	Many laboratory-based methods for detection of <i>E. coli</i> 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 <i>E. coli</i> 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 <i>E. coli</i> , treatment wetland systems can help treat water running off from agricultural premises and animal holdings.
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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

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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 piscicida*, *Pfiesteria*, *Prorocentrum lima*, *Protoperidinium crassipes*, *Pseudonitzschia* 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 carcasses 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 carcasses 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**
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- 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 Oceanográfico de Vigo, Cabo Estay-Canido, 36390 Vigo, Spain. vigohab@vi.ieo.es ☎ Tel: +34 986492111 ; Fax: +34 986492003.
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 - 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&doclistD=60 [Accessed March 2012].
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 - 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=11&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=15&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.

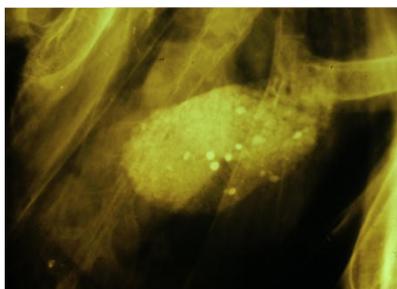
IDENTIFICATION AND RESPONSE

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 carcasses 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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Contacts

National Wildlife Health Center (USGS).

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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. grippityphosa*, *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 *Leptospira* 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].
- 📄 World Health Organization (WHO). Excerpt from: **WHO recommended standards and strategies for surveillance, prevention and control of communicable diseases.** www.who.int/zoonoses/diseases/Leptospirosissurveillance.pdf [Accessed March 2012].
- 📄 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].
- 📄 Centers for Disease Control and Prevention (CDC). **Leptospirosis.** <http://www.cdc.gov/leptospirosis/> [Accessed March 2012].
- 📄 **The Leptospirosis Information Center.** www.leptospirosis.org. [Accessed March 2012].
- 📄 World Health Organisation (WHO). **Leptospirosis.** www.who.int/zoonoses/diseases/leptospirosis/en. [Accessed March 2012].
- ✉ **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

Contacts



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*)

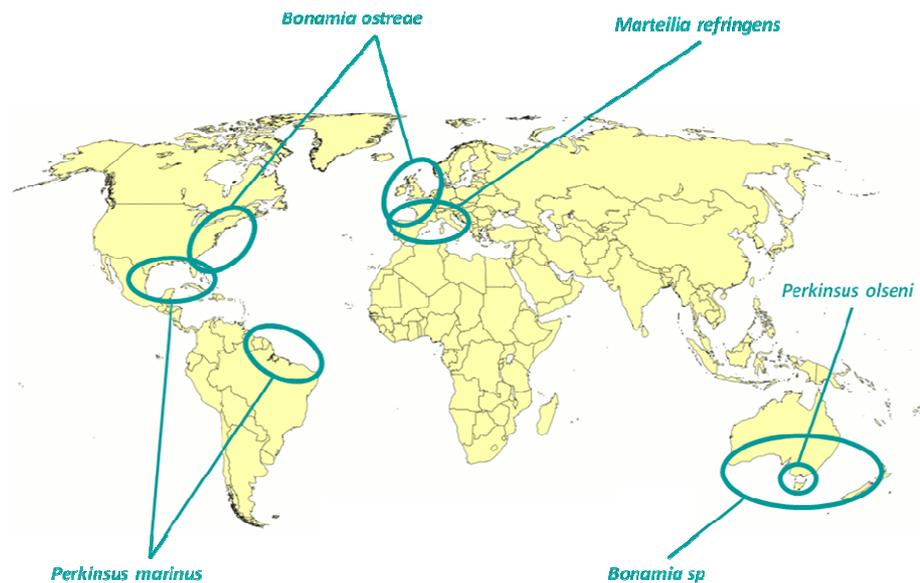
Bacteria of particular concern for human health include *Vibrio parahaemolyticus*, *V. vulnificus* and choleraenic *V. cholera*. Illness in humans is linked to the consumption of raw oysters.

Species affected

Farmed and wild oysters worldwide are affected by diseases and those species known to be susceptible are:

Scientific name	Common name
<i>Ostrea angasi</i>	Australian mud oyster
<i>O. chilensis</i>	Chilean flat oyster
<i>O. edulis</i>	European flat oyster
<i>O. puelchana</i>	Argentinean flat oyster
<i>O. denselammellosa</i>	Asiatic oyster
<i>Crassostrea gigas</i>	Pacific oyster
<i>C. virginica</i>	Eastern oyster
<i>C. ariakensis</i>	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 marsteiliosis 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 cholerae, *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

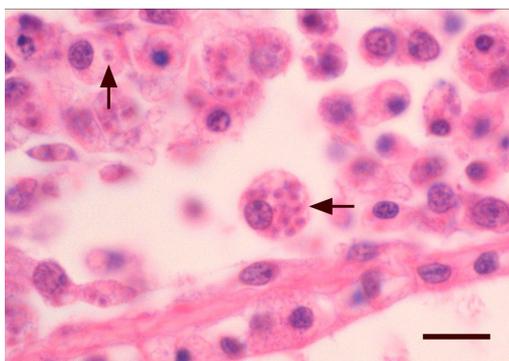
Useful publications and websites

- ▢ 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 ✗



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

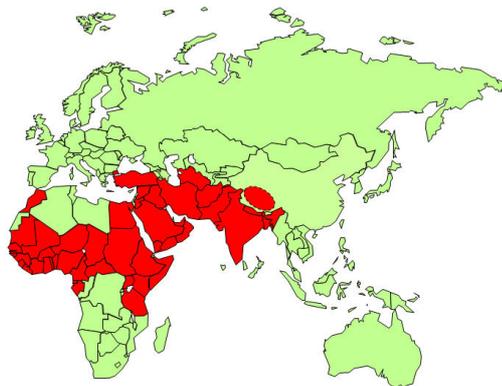
Peste des petits ruminants virus (PPRV), a member of the morbillivirus genus that includes measles virus and rinderpest virus (RPV).

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 carcasses (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 carcasses 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 carcasses 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 petits 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].
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- ☐ 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].
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- ☐ 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 ✗



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 carcasses/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/International_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 ✓



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 <i>Aedes</i> 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 <i>Phlebovirus</i> .
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 (<i>e.g. Aedes, Anopheles, Culex, Eretmapodites</i> and <i>Mansonia</i> 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 <i>via</i> 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 (<i>e.g. Aedes, Anopheles, Culex, Eretmapodites</i> and <i>Mansonia</i> 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 carcasses 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 ✓



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 carcasses should be submitted to a diagnostic laboratory. If the whole carcass 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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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 are most 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 propagates by asexual reproduction and several thousand free-swimming larvae, known as cercariae, 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, diarrhoea 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 desiccation 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 ability 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 susceptibility 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 ✓



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 piroplasmiasis**.
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 in 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 piroplasmiasis. 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.

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Useful publications and websites

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Contacts

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 trematodiasis such as heterophyiasis and echinostomiasis); (ii) nematodiasis (*e.g.* capillariasis, gnathostomiasis, anisakiasis); and (iii) cestodiasis (*e.g.* diphyllbothriasis). 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. felinus* (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 trematodiasis are caused by intestinal trematode parasites belonging to the families Heterophyidae 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.

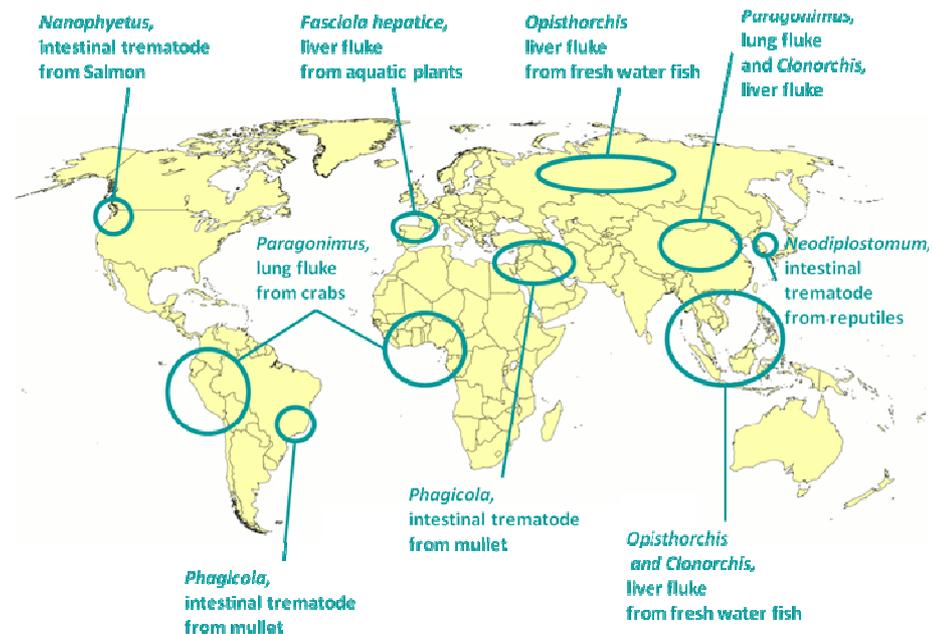
A large number of gastro-intestinal trematode species (paramphistomes) have been described. They are usually thick, short (4-12 mm), fleshy, maggot-like worms. They can infect all ruminants but young calves and lambs are the most susceptible. Not all species are pathogenic but clinical outbreaks of paramphistomiasis 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), herpetofauna (*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

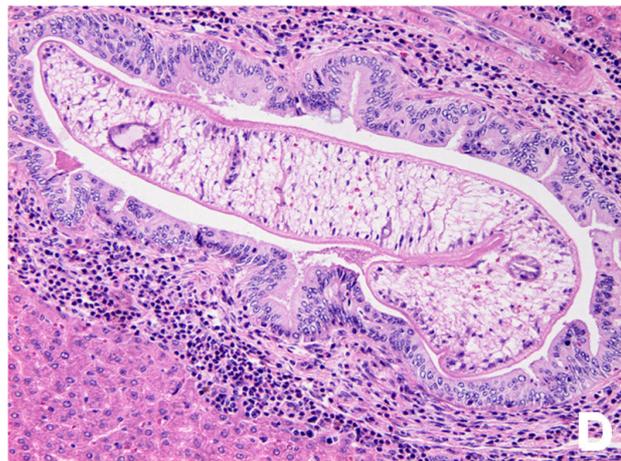
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Photos



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 ✓



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 <i>Culex</i> and <i>Aedes</i> 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 <i>Flavivirus</i> (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].
- ☐ World Organisation for Animal Health (OIE). **Chapter 2.01.20: West Nile disease.** Manual of diagnostic tests and vaccines for terrestrial animals. http://web.oie.int/eng/normes/MANUAL/2008/pdf/2.01.20_WEST_NILE.pdf [Accessed March 2012].
- ☐ European Centre For Disease Prevention and Control (ECDC). **Meeting Report - expert consultation on West Nile virus infection** (2011). http://www.ecdc.europa.eu/en/publications/Publications/1106_MER_WNV_Expert_Consultation.pdf [Accessed March 2012].
- 🔗 Centers for Disease Control and Prevention (CDC). **WNV what you need to know.** www.cdc.gov/ncidod/dvbid/westnile/wnv_factsheet.htm [Accessed March 2012].
- 🔗 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].
- 🔗 Wildpro. **West Nile virus disease.** http://wildpro.twycrosszoo.org/S/00dis/viral/WNV_Infection.htm [Accessed March 2012].

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