Response Policy Briefs

Brief policy statements for emergency animal diseases that are subject to cost sharing between governments and livestock industries but not covered by full AUSVETPLAN disease strategies

Version 3.5, 2013

AUSVETPLAN is a series of technical response plans that describe the proposed Australian approach to an emergency animal disease incident. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and emergency-management plans.

Standing Council on Primary Industries
These response policy briefs form part of:

AUSVETPLAN Edition 3

This manual will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to:
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DISEASE WATCH HOTLINE

1800 675 888

The Disease Watch Hotline is a toll-free telephone number that connects callers to the relevant state or territory officer to report concerns about any potential emergency disease situation. Anyone suspecting an emergency disease outbreak should use this number to get immediate advice and assistance.
Preface

These response policy briefs for the control and eradication of emergency animal diseases (EADs) not otherwise covered by AUSVETPLAN disease strategies are an integral part of the Australian Veterinary Emergency Plan, or AUSVETPLAN (Edition 3). AUSVETPLAN structures and functions are described in the AUSVETPLAN Summary Document.

Diseases that are listed by the World Organisation for Animal Health (OIE) are diseases with the potential for international spread, significant mortality or morbidity within the susceptible species and/or potential for zoonotic spread to humans.1 The principles contained in this document for the control of such EADs conform with the OIE Terrestrial Animal Health Code, where appropriate.2

In Australia, the arrangements for funding the control of EAD outbreaks are set out in the Government and Livestock Industry Cost Sharing Deed In Respect of Emergency Animal Disease Responses (EAD Response Agreement).3 Cost-sharing between governments and industry is determined according to four disease categories (categories 1, 2, 3 and 4) depending on the potential impact for Australia on public health, livestock production and international trade.

This manual provides brief information about the suggested starting policy and guidelines for agencies and organisations involved in a response to any one of these 29 diseases, which are covered under the EAD Response Agreement but not currently covered by individual AUSVETPLAN disease strategies. Further details of the diseases covered in this manual are in Section 1 (Introduction). West Nile virus disease is not currently included in the EAD Response Agreement.

In this manual, the placing of text in square brackets [xxx] indicates that that aspect of the manual remains contentious or is under development; such text is not part of the official manual. The issues will be worked on by experts and relevant text included at a future date.

Guidelines for the field implementation of AUSVETPLAN are contained in the disease strategies, operational procedures manuals, management manuals and wild animal manual. Industry-specific information is given in the relevant enterprise manuals. The full list of AUSVETPLAN manuals that may need to be accessed in an emergency is shown below.

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1 These criteria are described in more detail in Chapter 1.2 of the OIE Terrestrial Animal Health Code (www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.1.2.htm).
2 www.oie.int/en/international-standard-setting/terrestrial-code/access-online/
3 Information about the EAD Response Agreement can be found at www.animalhealthaustralia.com.au/programs/emergency-animal-disease-preparedness/ead-response-agreement
AUSVETPLAN manuals

Disease strategies
- Individual strategies for each of 35 diseases
- Bee diseases and pests
- Response policy briefs (for diseases not covered by individual manuals)

Enterprise manuals
- Artificial breeding centres
- Feedlots
- Meat processing
- Saleyards and transport
- Pig industry
- Poultry industry
- Zoos

Operational procedures manuals
- Decontamination
- Destruction of animals
- Disposal
- Livestock welfare and management
- Public relations
- Valuation and compensation

Management manuals
- Control centres management (Parts 1 and 2)
- Laboratory preparedness

Wild animal response strategy

Summary document

Nationally agreed standard operating procedures

Nationally agreed standard operating procedures have been developed for use by jurisdictions during responses to emergency animal disease incidents and emergencies. These procedures underpin elements of AUSVETPLAN and describe in detail specific actions undertaken during a response to an incident.

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   2.14 Haemorrhagic septicaemia ............................................38
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1 Introduction

The AUSVETPLAN disease strategies are the authoritative reference to the control/eradication policies for a range of emergency animal diseases (EADs) in Australia. Each strategy provides information about:

- the nature of the disease;
- the principles of its control; and
- control policies.

Each strategy provides sufficient information to allow authorities to make informed decisions on the policies and procedures that should be used to control an outbreak of the disease in Australia.

For AUSVETPLAN Edition 2 (1996), full AUSVETPLAN disease strategies were developed for all exotic diseases covered by the 1994 cost-sharing agreement between with the Australian and state/territory governments and industry for sharing the costs of disease control should an outbreak occur. The 1994 agreement covered 12 diseases that were exotic to Australia. AUSVETPLAN Edition 2 manuals published in 1996 also included disease strategies for 12 further exotic animal diseases that were considered important for Australia.

Since 1996, the scope of AUSVETPLAN has been broadened to include further exotic animal diseases not previously included, as well as a number of endemic animal diseases, a serious outbreak of which would also cause significant problems for public health, livestock production or trade. The broadened scope of AUSVETPLAN is reflected in the change of emphasis from ‘exotic animal diseases’ to ‘emergency animal diseases’.

At the time of writing (August 2008), AUSVETPLAN Edition 3 includes 29 disease strategies (see Appendix 2).

Arrangements for funding the control of EAD outbreaks are set out in the Government and Livestock Industry Cost Sharing Deed In Respect of Emergency Animal Disease Responses (EAD Response Agreement). Under this agreement, cost sharing between governments and industry is determined according to four disease categories (Categories 1, 2, 3 and 4), depending on the potential impact of the disease on public health, livestock production and trade.

The EAD Response Agreement covers a total of 59 emergency animal diseases, including the 29 diseases for which there are AUSVETPLAN disease strategies and a further 29 diseases for which there are no disease strategies. This manual provides information about these remaining diseases with Australia’s policy for controlling them should an outbreak occur. One further disease, bovine

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6 Information about the EAD Response Agreement can be found at www.animalhealthaustralia.com.au/programs/emergency-animal-disease-preparedness/ead-response-agreement/
tuberculosis due to *Mycobacterium bovis*, is covered separately and is not included in this manual.

Table 1.1 lists the diseases covered by this manual and shows their status with the OIE, their classification under the EAD Response Agreement and their occurrence in Australia. Table 1.2 gives further details of Australian disease classifications.
Table 1.1  Emergency animal diseases that are subject to cost-sharing arrangements under the EAD Response Agreement, but for which there is no AUSVETPLAN disease strategy

<table>
<thead>
<tr>
<th>Disease</th>
<th>OIE notifiablea</th>
<th>Australian categoryb</th>
<th>Occurrence in Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borna disease</td>
<td>–</td>
<td>4</td>
<td>Unconfirmed isolation of disease agent</td>
</tr>
<tr>
<td>Bovine tuberculosis(^c)</td>
<td>+</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Brucellosis (due to <em>Brucella melitensis</em>)</td>
<td>+</td>
<td>2</td>
<td>Not present in domestic livestock</td>
</tr>
<tr>
<td>Contagious bovine pleuropneumonia</td>
<td>+</td>
<td>3</td>
<td>Last case in 1967; declared free in 1973</td>
</tr>
<tr>
<td>Contagious equine metritis</td>
<td>+</td>
<td>4</td>
<td>Outbreak in 1980</td>
</tr>
<tr>
<td>Dourine</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>East coast fever (theileriosis)</td>
<td>+</td>
<td>4</td>
<td>Disease agent not present</td>
</tr>
<tr>
<td>Encephalitides (tick-borne)</td>
<td>–</td>
<td>3</td>
<td>Never</td>
</tr>
<tr>
<td>Epizootic lymphangitis</td>
<td>–</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Equine babesiosis (equine piroplasmosis)</td>
<td>+</td>
<td>4</td>
<td>Last case in 1976</td>
</tr>
<tr>
<td>Equine encephalomyelitis (eastern, western and Venezuelan)</td>
<td>+</td>
<td>1</td>
<td>Never</td>
</tr>
<tr>
<td>Equine encephalosis</td>
<td>–</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Getah virus disease</td>
<td>–</td>
<td>4</td>
<td>Unconfirmed detection of disease agent in 1960s</td>
</tr>
<tr>
<td>Glanders</td>
<td>+</td>
<td>2</td>
<td>Last case in 1891</td>
</tr>
<tr>
<td>Haemorrhagic septicaemia</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Heartwater</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Hendra virus infection (formerly equine morbillivirus)</td>
<td>–</td>
<td>2</td>
<td>Sporadic outbreaks have occurred regularly (on an almost annual basis) since 1994</td>
</tr>
<tr>
<td>Jembrana disease</td>
<td>–</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Maedi–visna</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Menangle virus (porcine paramyxovirus)</td>
<td>–</td>
<td>3</td>
<td>One outbreak in 1997</td>
</tr>
<tr>
<td>Nairobi sheep disease</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Nipah virus</td>
<td>–</td>
<td>1</td>
<td>Never</td>
</tr>
<tr>
<td>Potomac fever</td>
<td>–</td>
<td>4</td>
<td>Serological evidence of agent; clinical disease has not occurred</td>
</tr>
<tr>
<td>Pulmonary adenomatosi6s</td>
<td>–</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Sheep scab</td>
<td>–</td>
<td>4</td>
<td>Parasite eradicated in 1896</td>
</tr>
<tr>
<td>Swine influenza</td>
<td>–</td>
<td>4</td>
<td>Negative serology in 1977; last case in 2009</td>
</tr>
<tr>
<td>Teschen disease (enterovirus encephalomyelitis)</td>
<td>+</td>
<td>4</td>
<td>Never</td>
</tr>
<tr>
<td>Trichinosis</td>
<td>+</td>
<td>3</td>
<td>Never</td>
</tr>
<tr>
<td>Vesicular exanthema</td>
<td>–</td>
<td>3</td>
<td>Never</td>
</tr>
<tr>
<td>Wesselsbron disease</td>
<td>–</td>
<td>4</td>
<td>Never</td>
</tr>
</tbody>
</table>

\(^a\) World Organisation for Animal Health (OIE) emergency disease lists (see Table 2). Key: – = not notifiable; + = notifiable.

\(^b\) Government and Livestock Industry Cost Sharing Deed In Respect of Emergency Animal Disease Responses (EAD Response Agreement) (see Table 3)
Note: The principles contained in this document for the diagnosis and management of an outbreak of EAD conform, where appropriate, with the OIE Terrestrial Animal Health Code (see Appendix 3).

### Table 1.2 Australian classification of emergency animal diseases

<table>
<thead>
<tr>
<th>EAD category</th>
<th>Definition</th>
<th>Cost sharing (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Emergency animal diseases that predominantly seriously affect human health and/or the environment (depletion of native fauna) but may only have minimal direct consequences to the livestock industries.</td>
<td>Govt: 100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Industry: 0</td>
</tr>
<tr>
<td>2</td>
<td>Emergency animal diseases that have the potential to cause major national socioeconomic consequences through very serious international trade losses, national market disruptions and very severe production losses in the livestock industries that are involved. Category 2 also includes diseases that may have slightly lower national socioeconomic consequences, but also have significant public health and/or environmental consequences.</td>
<td>Govt: 80</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Industry: 20</td>
</tr>
<tr>
<td>3</td>
<td>Emergency animal diseases that have the potential to cause significant (but generally moderate) national socioeconomic consequences through international trade losses, market disruptions involving two or more states and severe production losses to affected industries, but have minimal or no effect on human health or the environment.</td>
<td>Govt: 50</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Industry: 50</td>
</tr>
<tr>
<td>4</td>
<td>Diseases that could be classified as being mainly production loss diseases. While there may be international trade losses and local market disruptions, these would not be of a magnitude that would be expected to significantly affect the national economy. The main beneficiaries of a successful emergency response to an outbreak of such a disease would be the affected livestock industries.</td>
<td>Govt: 20</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Industry: 80</td>
</tr>
</tbody>
</table>

The following subsections provide brief disease information and a policy statement for each of the 29 emergency animal diseases that are subject to cost sharing between governments and livestock industries but are not currently (2008) covered by full AUSVETPLAN disease strategies (see Table 1).

In this section:

- AAHL is the CSIRO–Australian Animal Health Laboratory in Geelong, Victoria
- SCAHLS is the Animal Health Committee’s Subcommittee on Animal Health Laboratory Standards.

Other abbreviations are shown in the Abbreviations list (see Contents for page number).
2.1 Borna disease

Borna disease (BD) is an immune-mediated viral polioencephalomyelitis of horses, sheep and occasionally other animals. Near-east equine encephalomyelitis (NEEE) is caused by a similar virus (see below). NEEE is exotic to Australia.

Causative agent

BD virus is the prototype of a newly recognised virus family, Bornaviridae, within the nonsegmented, negative-sense, single-strand RNA viruses (order Mononegavirales). NEEE is also caused by a bornavirus.

Hosts

Horses and sheep are the main natural hosts of BD virus, but occasional cases occur in other equids, cattle, goats, deer, rabbits and ostriches. Many other species have been experimentally infected, and cats have been found to be serologically positive. The behavioural changes seen in animals, together with worldwide serological and virological evidence that either BD virus, or a variant of it, may infect humans, have led to the hypothesis that BD virus may be responsible for neurological disturbances leading to the behavioural changes seen in some human neuropsychiatric disorders. However, immunological and molecular studies have provided inconsistent evidence of this association, and the involvement of BD virus in psychiatric disease remains unclear.

Distribution

BD first appeared as an epidemic disease of horses in the southern areas of Germany (Borna is a town in Saxony) in the late nineteenth century. Clinical BD in horses and sheep was originally thought to be restricted to the endemic areas of central Europe. NEEE occurs in several countries in the Middle East, and sporadic outbreaks may occur as far afield as Sudan and some areas of the former Soviet Union.

As diagnostic methods have improved and greater interest in the disease has developed, evidence of BD virus infection has been found worldwide in an increasing number of species. Information about its distribution is limited because a reliable diagnostic test is not available.

There have been reports of the isolation of BD virus from horses and cats in Australia, but these have not been confirmed.

Method of spread

The mode of transmission of BD is unknown, but the presence of the virus in saliva, nasal secretions and urine suggests that it is spread mainly by direct contact between animals. Rodents have been suggested as both reservoirs and vectors. NEEE is transmitted by the tick *Hyalomma anatolicum* and occurs seasonally.

Disease management

The epidemiology of BD is unclear. Serological studies indicate that BD virus infections are clinically inapparent in most cases. Sporadic outbreaks, with mortality rates of up to 90%, occur in horses in central Europe. Often, only
individual animals are clinically affected in stables with a high seroprevalence. Cases occur mainly in young horses at any time of the year, but are concentrated in late spring and early summer. Occasionally, BD causes substantial losses in sheep. No effective control processes currently exist.

**Laboratory diagnostic capacity**

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.

<table>
<thead>
<tr>
<th>Australia's policy for Borna disease (BD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BD is not an OIE-listed disease. If BD virus is detected in Australia, the species involved are likely to be horses or cats. Because of the reported association between BD virus infection and human neuropsychiatric disease, there is potential for a significant media and public reaction.</td>
</tr>
<tr>
<td>The policy is to eradicate BD if the infection is identified in a recently introduced animal. If BD virus is found in Australian-born animals and proves to be widespread in the population, eradication will not be attempted. Both approaches will require:</td>
</tr>
<tr>
<td>• serosurveillance (if the tests available are considered reliable) to assess the extent of the virus spread;</td>
</tr>
<tr>
<td>• stamping out of the disease in individual animals and small groups of in-contact animals; and</td>
</tr>
<tr>
<td>• a public awareness campaign to inform the public, including animal owners and consumers, of the known risks associated with the virus.</td>
</tr>
<tr>
<td>BD is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.</td>
</tr>
</tbody>
</table>
2.2 Brucellosis (due to *Brucella melitensis*)

The bacterium *Brucella melitensis* is a serious cause of brucellosis in goats, sheep and humans. The disease affects mainly adult female animals, causing abortion and udder infection.

**Causative agent**

*B. melitensis* is one of several species of the bacterial genus *Brucella*. Another of the genus, *B. abortus*, causes a form of brucellosis in cattle (covered by an AUSVETPLAN disease strategy).

**Hosts**

Sheep and goats are the main livestock species affected by the disease. Cattle are occasionally infected by *B. melitensis* in endemic areas, but such infections are usually subclinical.

*B. melitensis* is the most pathogenic species of *Brucella* for humans. It causes the disease known as Malta fever or undulant fever.

**Distribution**

*B. melitensis* occurs in the Mediterranean and Middle East region, Central Asia, China, southern areas of the former Soviet Union, Southeast Asia, some areas of Europe, Africa and the Indian subcontinent. It has a high prevalence in Central and South America.

*B. melitensis* is not present in domestic livestock in Australia, but cases sometimes occur in people who have contracted the disease while overseas.

**Method of spread**

The disease is spread through live animal contact. Aborted foetuses, placentae and foetal fluids are heavily contaminated. In small ruminants, the excretion of *B. melitensis* in vaginal discharges after abortion is more prolonged than is the case with cows infected with *B. abortus*. Mechanical transmission can occur on the hands of the milker.

Human infection most frequently results from ingestion of contaminated raw milk, other unpasteurised dairy products or uncooked meat.

**Disease management**

Management will require vaccination to decrease prevalence, and a test-and-slaughter program for eradication.
Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.

<table>
<thead>
<tr>
<th>Laboratory diagnostic capacity</th>
</tr>
</thead>
</table>

**Australia’s policy for brucellosis due to *Brucella melitensis***

Brucellosis due to *B. melitensis* is an OIE notifiable disease. If *B. melitensis* were uncontrolled in Australia, it would have the potential to spread widely. It is a serious zoonosis, causes production losses and has the potential to disrupt trade.

Because brucellosis is likely to be restricted to small areas, the policy is to eradicate the disease using:

- stamping out of infected groups; and
- quarantine and movement controls.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- declaration and zoning of affected areas to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation with industry and to reassure consumers.

Brucellosis due to *B. melitensis* is currently included as a Category 2 disease in the EAD Response Agreement. The costs of disease control would be shared 80% by governments and 20% by the relevant industries.
2.3 Contagious bovine pleuropneumonia

Contagious bovine pleuropneumonia (CBPP), or ‘pleuro’, is an acute, subacute or chronic mycoplasmal interstitial pneumonia of cattle, with the potential to cause severe production and economic problems.

Causative agent

CBPP is caused by Mycoplasma mycoides subsp. mycoides (small colony type, bovine biotype).

Hosts

Cattle are the main hosts, but the disease also occurs in water buffalo and yaks.

Distribution

CBPP occurs in most parts of Africa, parts of the Middle East, Europe and Asia.

CPBB last occurred in Australia in 1967; Australia declared freedom from the disease in 1973.

Method of spread

Infection is spread through close animal contact by inhalation of infected respiratory aerosols. Spread of the disease in the past was due to movement of animals and droving, but modern husbandry techniques mitigate spread.

Disease management

Disease management includes quarantine, serological testing, vaccination, movement restrictions, and stamping out (where newly introduced diseased animals are detected among healthy herds). Antibiotics have been used for postvaccinal reactions, but are unlikely to be useful in controlling the disease and may contribute to animals becoming chronically infected. Because CBPP is usually a chronic and possibly subclinical disease, serological testing of susceptible animals for importation is essential.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for contagious bovine pleuropneumonia (CBPP)

CBPP is an OIE notifiable disease. An uncontrolled outbreak of CBPP has the potential for rapid spread within a herd and could spread to other herds. The disease would cause severe production losses to the affected producers, with potential dislocation and financial losses to the cattle industry from effects on exports.

The policy is to eradicate CBPP using:

- destruction of all infected and likely to be infected animals, with sanitary disposal of destroyed animals;
- quarantine and movement controls on animals on infected and suspect premises and within the immediate vicinity to prevent the spread of infection; and
- test and slaughter, which involves regular serological testing of in-contact animals and slaughter of those that test positive.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation from industry.

Vaccination will only be used to support eradication if the disease becomes widespread.

CBPP is currently included as a Category 3 disease in the EAD Response Agreement. The costs of disease control would be shared 50% by governments and 50% by the relevant industries.
2.4 Contagious equine metritis

Contagious equine metritis (CEM) is a sexually transmitted disease of horses that causes endometritis and temporary infertility in mares. It is rarely associated with abortion. Infected stallions are asymptomatic.

Causative agent

CEM is caused by the bacterium *Taylorella equigenitalis*. A number of biologically distinct strains exist, which can differ in pathogenicity.

Hosts and clinical signs

Clinical disease occurs only in mares, and all breeds of horses are susceptible to infection. CEM does not affect humans.

Many primary cases of CEM in mares are subclinical. A characteristic sign is the early, unexpected return to oestrus of multiple mares that have been served by the same stallion. In most natural cases, the incubation period is 1–3 days.

If clinical signs are present, the severity of infection varies. The most obvious clinical sign is a mucopurulent vaginal discharge, resulting from endometritis.

Most affected mares recover uneventfully, but some may become asymptomatic carriers where bacteria persist in the smegma of the clitoral fossa and sinuses and possibly, in a small number of cases, in the uterus.

Stallions show no clinical signs, but the organism may localise in the urethral fossa, the anterior urethra and the internal folds of the prepuce.

Distribution

CEM is present or suspected in Japan, Turkey, most of the European Union member states and countries in eastern Europe. It has occurred in Australia, the United States and Switzerland; it has never been recorded in New Zealand or South Africa. The last Australian case was recorded in 1980, and Australia was declared CEM-free in 1985.

Methods of spread

CEM is usually transmitted sexually through natural breeding or artificial insemination. Infection can also occur from other genital-to-genital, or nose-to-genital, contact between stallions/teasers and mares. Mechanical transmission may occur via contaminated equipment used during genital examinations or mating.

The most likely way by which CEM could be introduced into Australia is by the importation of a carrier stallion or brood mare.

Diagnosis

A definitive diagnosis of CEM relies on the isolation of *T. equigenitalis* from swabs. However, the collection, culture and identification of *T. equigenitalis* is a technically difficult procedure because the organism is shed intermittently, does not survive well during transport and requires prolonged incubation on special media. It is
essential that standardised collection methods be used and specimens for culture only be sent to approved laboratories.

A real-time polymerase chain reaction (PCR) test is available and is used for screening horses in countries where the disease is endemic.

Managing the risk
Managing the risk of CEM relies on the following basic principles:

- preventing the exposure of susceptible horses to CEM-infected horses and contaminated semen;
- stopping mechanical spread of CEM bacteria; and
- eliminating CEM bacteria from infected horses.

Meticulous tracing of the breeding history of infected horses will be necessary. Good hygienic gynaecological practice will be essential to prevent inadvertent spread of infection between mares. Compulsory slaughter of infected animals will not be appropriate or necessary as part of a control program.

Australia’s policy for contagious equine metritis (CEM)

CEM is an OIE listed disease with potential to spread rapidly and cause epidemic infertility. It has important implications for the international movement of horses, particularly thoroughbred horses involved in the breeding industry.

The policy is to eradicate CEM where practicable. If the index case(s) is detected early, eradication may be feasible. However, if infection becomes well established before detection, the insidious nature of the disease and the national mobility of breeding horses could make eradication difficult and not economically viable.

The overall policy is to control and then eradicate CEM by:

- cessation of breeding activities on the infected properties until the extent of spread has been clarified;
- quarantine and movement controls on infected and exposed horses, and fomites, to minimise the spread of infection;
- decontamination of facilities and fomites to eliminate the causative agent from infected premises;
- tracing and surveillance to determine the source and extent of infection, and to provide proof of freedom from the disease;
- testing and treatment of infected horses until all susceptible horses on infected premises are confirmed to be free of infection; and
- a public awareness campaign to facilitate cooperation from the industry and the community.

CEM is an Animal Health Australia Category 4 disease under the EAD Response Agreement for cost-sharing arrangements. Category 4 diseases are those for which costs will be shared 20% by government and 80% by industry.
2.5 Dourine

Dourine is a venereally transmitted acute or chronic trypanosomal disease of horses, and is characterised by swelling of the genitalia, nervous disorders and emaciation.

Causative agent

Dourine is caused by the protozoan, *Trypanosoma equiperdum*, one of the salivarian trypanosomes.

Hosts

The disease occurs mainly in horses. Mild or subclinical infection can occur in donkeys and mules.

Distribution

Dourine occurs in parts of the former Soviet Union, South America, Asia, and northern and southern Africa. Because dourine has been eradicated in many countries, it not as widespread as it once was.

This disease has never occurred in Australia.

Method of spread

Dourine is sexually transmitted. Foals born to infected mares may be infected. The incubation period is highly variable, and disease may not appear for several years.

Disease management

Clinical symptoms can be suppressed by chemotherapeutic drugs, but animals remain carriers of the parasite even after drug therapy. Destruction may be used in eradication programs, but may not be necessary if infected animals are castrated or ovariotomised and strict precautions are taken to isolate affected animals. Care needs to be taken when importing from countries with dourine that animals have not recently been exposed and that blood tests are negative. Ordinarily, an infected animal will have already bred by the time the disease is diagnosed and its future breeding value will be negligible. Therefore, the infected animal should be sacrificed; in limited circumstances, stallions may be castrated.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for dourine

Dourine is an OIE notifiable disease. An uncontrolled outbreak in Australia would have important socioeconomic consequences for the horse industry, causing restrictions and losses, especially by disrupting breeding activities. Under OIE regulations, a minimum of two years after the last clinical case of the disease would be required before Australia could be declared free from the disease.

The policy is to eradicate dourine by:

- serological identification of infected animals, which would then be destroyed or neutered to prevent further disease transmission;
- quarantine of all equids in premises where dourine has been detected and premises to which breeding animals have been moved; and
- cessation of equid breeding for two months in the designated infected premises while testing is carried out.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation from industry.

Dourine is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.6 East coast fever

East coast fever (ECF), or theileriosis, is a severe tick-transmitted protozoal disease of cattle and buffalo, characterised by high fever and lymphadenopathy.

Causative agent

ECF is caused by the protozoan *Theileria parva parva*. Related bovine theilerioses are caused by other members of the *T. parva* complex. *T. parva* alternates between cattle and ticks in its lifecycle.

Hosts

ECF occurs in cattle, African buffalo and water buffalo. However, indigenous breeds of *Bos indicus* cattle in Africa are comparatively resistant to the disease.

Distribution

ECF occurs in eastern, central and southern parts of the African continent.

*T. parva* is not present in Australia. However, nonpathogenic *Theileria* (e.g. *T. buffeli*) do occur here.

Method of spread

*Theileria* spp are transmitted by ticks. The most important vector is the three-host tick *Rhipicephalus appendiculatus*, which requires a different host for every instar; the tick drops off each animal after engorging and moults on the ground. *Rhipicephalus sanguineus* (the brown dog tick) is the only tick from this genus present in Australia. ECF is unlikely to occur in Australia unless the tick vector is introduced, but alternative vectors may already be present.

Disease management

An outbreak of ECF in Australia would be localised to areas with suitable vectors.

A number of drugs (such as menocitone, parvaquone, buparvaquone and halofuginone) can be used to treat clinical symptoms. Drug therapy varies in expense and efficacy.

An ‘infection and treatment’ immunisation method can be applied. Other preventive measures include isolation of susceptible cattle, tick control and destocking.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia’s policy for east coast fever (ECF)

ECF is an OIE notifiable disease (described as theileriosis), as the disease can cause high mortalities in cattle breeds that are not indigenous to the endemic area. An uncontrolled outbreak in Australia would cause severe production losses to the affected producers, with potential dislocation and financial losses to the cattle industry from effects on exports.

The policy is to eradicate ECF, where it has been found in a limited distribution, by:

- treating animals to ensure freedom from ticks;
- therapeutic treatment with effective drugs to eliminate the causative organism; and
- a tick vector eradication campaign.

These strategies will be supported by:

- quarantine and movement controls on animals in the designated infected area to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

Vaccination will not be used to support eradication.

ECF is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.7 Encephalitides (tick-borne)

This term refers to a group of diseases (syndromes) caused by neurotropic viruses that are transmitted via the bite of ticks.

The most important animal disease of the group is louping ill, an encephalitis affecting mainly sheep, whose occurrence is closely related to the distribution of the primary tick vector, *Ixodes ricinus*. Louping ill is characterised by fever and uncoordinated movement, leading to animals becoming prostrate and comatose.

In Europe, other diseases caused by these viruses include Russian spring–summer encephalitis, central European encephalitis and related disorders. Powassan encephalitis, caused by the Powassan virus, occurs in North America and Russia.

**Causative agent**

Tick-borne encephalitides are caused by viruses among 14 antigenically related viruses of the *Flavivirus* genus of the family Togaviridae.

**Hosts**

Louping ill affects mainly sheep, but occasional cases occur in cattle, horses, pigs and deer, and rare cases occur in humans.

**Distribution**

Tick-borne encephalitides occur in Europe and the former Soviet Union. The diseases have not occurred in Australia.

**Method of spread**

Several tick species are vectors for this disease group, including the genera *Ixodes*, *Dermacentor* and *Haemaphysalis*. *Ixodes ricinus* (the ‘castor bean’ tick) is considered the natural vector of the virus causing louping ill. The virus is unlikely to spread without a vector. *I. ricinus* is common in Europe and some other countries; it is frequently found on dogs but also on other domestic animals and on wild mammals. The tick transmits several other diseases, including *Babesia divergens* and *Babesia bovis* (redwater of cattle), *Anaplasma marginale*, rickettsial tick-borne fever of sheep, *Coxiella burnetii*, Bukhovinian haemorrhagic fever and Lyme disease.

**Disease management**

Acaricide treatment and vaccination are two tools for control.

**Laboratory diagnostic capacity**

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
**Australia's policy for tick-borne encephalitides**

Louping ill is not an OIE-listed disease. However, it is an FAO listed disease that can cause serious disease in sheep and is transmissible to humans. An uncontrolled outbreak of louping ill would cause production losses to the affected producers, with potential dislocation and financial losses to the sheep industry. The occurrence of the tick *Ixodes ricinus* would be of concern because of its host range and the number of diseases with which it is associated.

The policy is to eradicate louping ill by:
- treating animals to eliminate ticks; and
- considering application of a vaccination program.

These strategies will be supported by:
- movement controls on animals in the designated infected area to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

Louping ill is currently included as a Category 3 disease in the EAD Response Agreement. The costs of disease control would be shared 50% by governments and 50% by the relevant industries.
2.8 Epizootic lymphangitis

Epizootic lymphangitis is a chronic fungal disease associated with ulcers and granulomatous disease of the skin, lymph vessels and lymph nodes on the necks and legs of horses.

Causative agent
Epizootic lymphangitis is caused by the dimorphic fungus, *Histoplasma capsulatum* var *farciminosum*.

Hosts
The disease affects horses and mules and, less commonly, donkeys and camels. It may very rarely occur in humans.

Distribution
Epizootic lymphangitis occurs in parts of Africa, the Middle East, Asia and Central America.

This disease has never occurred in Australia.

Method of spread
Epizootic lymphangitis is spread by live animal contact, mostly through contamination of skin wounds or abrasions by flies or by dirty grooming or harness equipment. The fungus has a saprophytic phase in soil and can persist for many months in warm, moist conditions.

Disease management
Epizootic lymphangitis has been reported to respond to iodide treatment. Treatment by local surgery is only successful when it is performed early.

Prevention consists of isolation of infected animals, disinfection of contaminated properties and proper sanitary measures. A vaccine is not commercially available, but recovered animals are immune to reinfection. Entry into Australia could occur through live horses or contaminated materials.

Laboratory diagnostic capacity
Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
**Australia’s policy for epizootic lymphangitis**

Epizootic lymphangitis is not an OIE notifiable disease. However, it can cause serious disease in horses and donkeys. An uncontrolled outbreak would cause serious disruption to the horse industry.

The policy is to eradicate epizootic lymphangitis by:

- destruction of infected horses;
- quarantine of the infected premises, which will remain destocked for 12 months; and
- strict hygiene, including destruction of contaminated bedding and equipment.

These strategies will be supported by:

- movement controls on animals in the designated infected area to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

Epizootic lymphangitis is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.9 Equine babesiosis

Equine babesiosis, or equine piroplasmosis, is a tick-borne intra-erythrocytic protozoal disease of horses, mules, donkeys and zebras. The disease is characterised by fever, progressive anaemia and abortion.

Causative agent

Two protozoa cause the disease: Babesia equi and Babesia caballi. B. equi is the more pathogenic of the two.

Hosts

Equine babesiosis occurs in horses, donkeys, mules and zebras, but horses are the most susceptible.

Distribution

Equine babesiosis is present in regions of Europe, the former Soviet Union, Africa, the Middle East, India, Indonesia, North and South America, and the Caribbean.

Australia is free of the disease; the last case occurred in 1976.

Method of spread

The tick vectors of equine babesiosis are members of the Dermacentor, Hyalomma and Rhipicephalus genera. The disease can also be spread iatrogenically by intravenous equipment.

Disease management

Several drug therapies of varying efficacy are available. On the evidence, no existing drugs appear to satisfactorily sterilise B. equi infections, but a few are useful in sterilising B. caballi. Chemosterilisation of Babesia infections is rarely recommended, but can be used when moving an infected animal to an area free of the disease. There are no effective vaccines against equine babesiosis.

In intensively managed systems, it is possible to control contact between tick vectors and equid hosts by appropriate use of acaricides. In free-ranging systems, this is more difficult.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia's policy for equine babesiosis

Equine babesiosis (equine piroplasmosis) is an OIE notifiable disease that is a major constraint on the international movement of horses from known infected to uninfected countries. B. equi has entered Australia more than once, but has died for want of a suitable biological vector. The introduction of ticks known to be suitable vectors would be of concern.

The policy is to eradicate equine babesiosis by eradicating the tick vector through:

- treating animals with chemical acaricides; and
- sanitation procedures to remove the vectors or potential vectors.

These strategies will be supported by:

- prevention of iatrogenic transmission by using sterile equipment in intravenous procedures;
- quarantine and movement controls on animals in the designated infected area to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

Equine babesiosis is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.10 Equine encephalomyelitis (eastern, western and Venezuelan)

Eastern, western and Venezuelan equine encephalomyelitis (EEE, WEE and VEE, respectively) are arthropod-borne viral diseases of horses. They also affect humans and poultry. Infection can cause fever, uncoordinated movement, paralysis, coma and death.

Causative agent

The viruses responsible for these diseases are members of the \textit{Alphavirus} genus of the family Togaviridae.

Hosts

Of the species that display clinical disease, horses and humans are the most important natural hosts for the viruses. Donkeys and mules are as susceptible as horses. Two of the viruses (eastern and western) have also caused mortalities among birds, including domestic fowls and emus. Other mammalian and bird species are susceptible to infection, but such infections are usually subclinical.

Distribution

EEE is present in the eastern half of the United States, southern Canada, Central America, the Caribbean and limited areas of Ecuador, Colombia, Venezuela, Guyana, Brazil and Argentina.

WEE is present in the western half of the United States, southwestern Canada, Mexico and most of South America east of the Andes.

VEE is present in northern South America and periodically spreads as an epidemic into Central America.

Equine encephalomyelitis (EEE, WEE or VEE) has never occurred in Australia.

Method of spread

Several mosquito vectors transmit the viruses between a complex array of natural host species.

Disease management

During outbreaks, the most effective way to prevent further spread of disease is to quarantine infected equines. Controlling mosquito populations with insecticides and eliminating mosquito breeding sites will also improve disease control.

Vaccines are available as a preventive measure.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia's policy for equine encephalomyelitis

Equine encephalomyelitis is an OIE notifiable disease. While none of the equine encephalomyelitides has ever occurred in Australia, suitable mosquito vectors probably exist throughout the country. It is extremely unlikely that these diseases could be eradicated once established.

The policy is to attempt eradication of an initial outbreak of equine encephalomyelitis by:

- quarantine and movement controls of infected animals;
- possible destruction of infected animals for humane reasons or of an imported animal found to be infected with equine encephalomyelitis; and
- vector abatement to reduce mosquito vectors to a minimum.

These strategies will be supported by:

- assessment of wild bird populations in the outbreak area to provide information on which to base management decisions;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- prevention of iatrogenic transmission by using sterile equipment in intravenous procedures;
- insect-proof housing for animals that may otherwise be exposed to infected vectors; and
- a public awareness campaign to encourage cooperation from industry.

Equine encephalomyelitis is currently included as a Category 1 disease in the EAD Response Agreement. The costs of disease control would be borne 100% by governments.
2.11 Equine encephalosis

Equine encephalosis is an insect-borne viral disease of horses that can cause a peracute illness with fluctuating fever, nervous signs or cardiac failure and death.

Causative agent

The disease is caused by a virus belonging to the Orbivirus genus of the family Reoviridae.

Hosts

Only horses are known to be affected.

Distribution

Equine encephalosis was identified in South Africa in 1967. It is possible and likely that it occurs in other parts of Africa.

The disease has never occurred in Australia.

Method of spread

The virus has been isolated from the midge Culicoides imicola, which is assumed to be the major insect vector. The Australian species of Culicoides that are competent vectors of bluetongue would most likely be competent vectors of equine encephalosis.

Disease management

Stabling of horses during the peak activity time of Culicoides midges reduces the incidence of infection. Chemical repellents and physical barriers are used to reduce the midges’ access to horses. Grazing horses with sheep and cattle may also act to decrease the incidence of bites to horses. If the disease occurs in an area where vectors do not normally occur, or are present only seasonally, the disease might be self-limiting and disappear as winter sets in.

Nonsteroidal anti-inflammatory drugs can be used to combat the fever, and other drug therapies can be used to alleviate other symptoms.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
**Australia’s policy for equine encephalosis**

Equine encephalosis is not listed as an OIE notifiable disease. While equine encephalosis has never occurred in Australia, the *Culicoides* midge vectors of bluetongue virus that exist in Australia would probably be competent vectors of equine encephalosis. It is unlikely that this disease could be eradicated if it becomes established in an area where vectors are present all year round.

The policy is to consider eradication of equine encephalosis by:

- quarantine and movement controls of infected horses;
- assessment of vector competence; and
- using insect repellents and physical barriers to reduce contact between biting midges and horses.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning in line with vector distribution;
- prevention of iatrogenic transmission by using sterile equipment in intravenous procedures; and
- a public awareness campaign to encourage cooperation from industry.

Equine encephalosis is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.12 Getah virus disease

Getah virus disease is an arthropod–borne viral disease of horses that causes fever and skin rash. Animals normally recover in a week, with very few deaths.

Causative agent

Getah virus is a member of the Semliki Forest antigenic complex of the Alphavirus genus of the family Togaviridae. It is weakly related to the Ross River virus, which is present in Australia.

Hosts

Clinical disease only occurs in horses and possibly neonatal pigs, but a wide range of species are subclinically infected, including humans, cattle, goats, dogs, domestic fowl and night herons.

Distribution

Getah virus disease was first isolated in Malaysia in 1955, and has since been detected in most parts of East Asia. Epidemics of the disease have occurred in horses in Japan.

There was one unconfirmed identification of Getah virus by an Australian laboratory in the 1960s, but the virus has not been detected since.

Method of spread

The virus has been isolated from nine species of mosquitoes, with most isolations occurring from Culex tritaeniorynchus and Aedes vexans nipponi. Natural transmission is between mosquitoes and horses, but pigs may also be an amplifying agent.

Disease management

Adequate import protocols and inspection should minimise the risk of an outbreak.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for Getah virus disease

Getah virus disease is not an OIE-listed disease. An outbreak in Australia would result in significant economic loss to the thoroughbred industry through constraints placed on the movement of horses. No effective control is practised in any other part of the world.

The Getah virus could enter Australia through an introduced mosquito, a natural host or an infected animal. If the virus enters through the mosquito vector, there can be no effective response. If it enters through the import of an infected animal, the policy is to eradicate the disease using:

- vector abatement;
- quarantine and movement controls;
- serological testing, tracing and surveillance to determine the source and extent of infection; and
- an awareness campaign to encourage cooperation of industry and the community.

Getah virus disease is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.13 Glanders

Glanders is a serious bacterial disease affecting mainly equids. Cutaneous, nasal and pulmonary forms of the disease occur.

Causative agent

Glanders is a zoonotic infection caused by *Burkholderia mallei*, a gram-negative, nonmotile, non-encapsulated and non-spore forming bacillus in the bacterial family Burkholderiaceae.

Hosts

The main hosts are horses, mules and donkeys, with the acute disease occurring mainly in the latter two species. Occasional cases, which can be lethal, occur in humans through contact with sick animals. Carnivores, including cats and dogs, are also susceptible.

Distribution

Glanders occurs in parts of the Middle East, Africa, the Indian subcontinent and Southeast Asia.

The disease has not occurred in Australia since 1891.

Method of spread

Animals usually become infected through ingesting contaminated food and water from troughs and through highly infectious nasal discharges. The pathogen can also be transferred through contamination of skin abrasions by dirty harness equipment and grooming tools.

Human infection usually occurs from contact of infected animal discharges with skin cuts and abrasions, while small carnivores are infected by eating infected meat.

Disease management

Treatment with antimicrobials alone or in combination with formalin-treated preparations of *B. mallei* has sometimes been successful. A test-and-slaughter strategy can be effective, but must be accompanied by quarantine of infected and surrounding premises. *B. mallei* is quite sensitive to heat, desiccation and common disinfectants.

Horses may recover from disease, but their subsequent immunity is incomplete. Therefore, immunisation has never successfully controlled the disease.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia's policy for glanders

Glanders is an OIE notifiable disease that can have serious impacts in horses and other equines. An uncontrolled outbreak of glanders in Australia would cause serious disruption to the horse industry.

The policy is to eradicate glanders by:

- identification of infected animals by allergenic or serological tests, and destruction of reactors;
- thorough disinfection of installations and equipment, including destruction of contaminated bedding and foodstuffs; and
- quarantine of the infected premises.

These strategies will be supported by:

- targeted movement controls on animals that may have been exposed, to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

Glanders is currently included as a Category 2 disease in the EAD Response Agreement. The costs of disease control would be shared 80% by governments and 20% by the relevant industries.
2.14 Haemorrhagic septicaemia

Haemorrhagic septicaemia is a specific form of acute pasteurellosis of cattle and buffalo that causes high mortality in infected animals. The disease has the potential to cause severe economic losses.

Causative agent

Haemorrhagic septicaemia is caused by serotype B:2 or E:2 of the bacterium Pasteurella multocida, which is a gram-negative, nonmotile rod.

Hosts

Water buffalo are the most susceptible species, followed by cattle. A haemorrhagic septicaemia–like disease, also caused by type B:2 or E:2 of the bacterium, has also been reported in pigs and elephants in contact with diseased cattle.

Distribution

Haemorrhagic septicaemia occurs in South and Southeast Asia (where it is regarded as one of the most serious diseases of large ruminants), the Middle East and most of Africa. It is associated with distinct wet–dry seasonal cycles, in which changes to climatic, dietary and physical conditions can subject animals to stress.

The disease has never been reported in Australia. Some strains of P. multocida are present, but not the strains that cause haemorrhagic septicaemia.

Method of spread

The pathogen is transmitted by direct contact between animals or through contaminated feedstuffs and water. The organism does not persist in the environment beyond a couple of days.

Disease management

The acute nature of most cases of the disease limits the efficacy of antimicrobial therapy of sick animals. An outbreak may be effectively controlled by administering a sulfonamide or other antibiotic to healthy animals that show a febrile reaction.

Immunity may be actively acquired through natural exposure or vaccination. Newborns can acquire immunity by ingestion of colostrum from immune dams. Long-lasting immunity is conferred on animals that recover from the natural disease. Vaccination can decrease the incidence of the disease, but usually has to be administered repeatedly through the life of the animal.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia’s policy for haemorrhagic septicaemia

Haemorrhagic septicaemia is an OIE notifiable disease. An uncontrolled outbreak in Australia would cause severe production losses in the cattle industry and loss of export markets.

The policy is to eradicate haemorrhagic septicaemia using:

- quarantine and movement controls;
- identification of infected animals by culture of blood and identification of the isolate as *P. multocida* serotype B:2 or E:2; and
- antibiotic treatment of animals showing a febrile reaction.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas;
- a public awareness campaign to encourage cooperation with industry and to reassure consumers; and
- development and assessment of an appropriate vaccine.

Haemorrhagic septicaemia is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.15 Heartwater

Heartwater, or cowdriosis, is an acute, tick-borne rickettsial disease of ruminants, with the potential to cause production and economic market loss. Peracute cases involving high fever, convulsions and death have been recorded, but acute cases are more common.

Causative agent

The disease is caused by *Ehrlichia ruminantium*, part of the Rickettsia group. The organism was previously named *Cowdria ruminantium*, but genetic analysis has regrouped *Cowdria* into the *Ehrlichia* genus.

Hosts

Cattle, water buffalo, sheep, goats and many wild ruminants are natural hosts.

Distribution

Heartwater occurs in Africa south of the Sahara and in the Caribbean. In November 1999, quarantine and eradication procedures were put in place in Florida in the United States after inspectors found 15 *Amblyomma sparsum* ticks on leopard tortoises imported from Africa. The ticks tested positive for *Ehrlichia ruminantium*.

Heartwater has never occurred in Australia.

Method of spread

The pathogen is carried by the tick genus *Amblyomma*, with *A. hebraeum* being the main vector. These ticks prefer wooded or brushy country rather than grasslands. Many of the world’s tropical and subtropical areas have competent tick vectors. Australian native fauna carry two indigenous *Amblyomma* species that very occasionally infest cattle. Wild animals may act as reservoirs.

Disease management

Heartwater can be treated in the early stages with tetracycline, sulphonamide and rifamycin antibiotics, and anti-inflammatory agents. Diuretics can also be given to clinically affected animals to counteract oedema formation.

Vaccines are available, but all contain live virulent organisms, and are hazardous because deaths can occur even with treatment. Tick control can limit the exposure of livestock to potential vectors. Chemoprophylaxis (with a series of oxytetracycline injections) can be used to protect susceptible animals that may have been exposed to the disease.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia's policy for heartwater

Heartwater is an OIE notifiable disease that affects cattle, water buffalo, sheep and goats. An outbreak of the disease in Australia would cause serious production losses and loss of export markets.

The policy is to eradicate heartwater using:
- quarantine and movement controls;
- acaricides or similar treatments to eliminate ticks on animals; and
- vaccination and antibiotic treatment.

These strategies will be supported by:
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation with industry and to reassure consumers.

Heartwater is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.16 Jembrana disease

Jembrana disease is a viral disease of Bali cattle that causes fever, lethargy and anorexia.

Causative agent

The disease is caused by a lentivirus from the family Retroviridae. Jembrana disease virus can be easily and consistently reproduced and is present only in cattle in areas where the disease is present. Virus has been detected by in situ hybridisation in proliferating lymphoid and macrophage cells in lesions of affected animals. Comparison of the Jembrana disease virus genome with those of other lentiviruses has shown it is most closely related to the ‘bovine immunodeficiency virus’.

Jembrana disease is unusual for a lentivirus disease. Most lentiviruses produce a slowly progressive disease syndrome after a prolonged incubation period of months to years. In contrast, Jembrana disease is acute and occurs after a short incubation period of 12 days or less.

Hosts

Indonesian ‘Bali’ (*Bos javanicus*) cattle are the main host. Experimental infection of crossbred (*Bos indicus* and *Bos taurus*) cattle produces only a mild or subclinical infection.

Distribution

Jembrana disease occurs in parts of Indonesia. Although the disease has not occurred in Australia, feral Bali cattle are present in Australia in the Northern Territory, particularly in the Coburg Peninsula.

Method of spread

Jembrana disease is thought to spread through the mechanical transmission of blood, either through biting arthropods or mass vaccination programs. There is evidence of transmission of the disease from acutely affected animals to susceptible cattle in close contact.

Disease management

After initial outbreaks with high rates of mortality in particular areas, the disease has become endemic and the case fatality rate has settled at about 20%. There is no recurrence of any clinical syndrome in animals that recover.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
**Australia's policy for Jembrana disease**

Jembrana disease is not an OIE-listed disease. This disease is likely to be of little significance to Australia, as it would affect only the small population of Bali cattle in the Northern Territory.

The policy is to eradicate Jembrana disease from Bali cattle in Australia, and to carry out surveillance of cattle and buffalo herds to establish that they are not involved, using the following methods.

In Bali cattle:

- segregation of cattle groups, where feasible, with separation greater than the flying range of insect vectors; and
- clinical and serological surveillance.

In cattle and buffalo:

- clinical and serological surveillance.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- zoning to define infected and disease-free areas.

Jembrana disease is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.17 Maedi–visna

Maedi and visna are slowly progressive viral diseases. Maedi affects the respiratory system and visna the central nervous system. They are separate clinical manifestations of infection by the same virus.

Causative agent
Maedi–visna virus is a member of the lentivirus group of the family Retroviridae.

Hosts
Sheep and, to a lesser extent, goats are susceptible.

Distribution
Maedi–visna was first recognised in Iceland, where it caused the most dramatic losses of livestock. It occurs in most of Europe, parts of Africa, the Middle East, the former Soviet Union, India, Asia and the Americas.

The disease has never occurred in Australia.

Method of spread
Maedi–visna is spread by direct contact between animals, presumably by the respiratory route. The incubation period of the disease is usually more than two years.

Disease management
Except for symptomatic medication, there is no specific treatment for the disease, and the outcome is invariably fatal. Prompt killing of infected animals is the only viable control measure.

Laboratory diagnostic capacity
Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for maedi–visna

Maedi-visna is an OIE notifiable disease that causes chronic respiratory and nervous system problems in sheep and goats. An uncontrolled outbreak of maedi-visna in Australia has the potential to cause loss of export markets and long-term production losses to the sheep industry. However, risk of entry of this disease into Australia in extremely small.

The policy is to eradicate maedi–visna using:

- identification and eradication of infected flocks; and
- quarantine and movement controls.

These strategies will be supported by:

- tracing and serological surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to reassure consumers.

Maedi-visna is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.18 Menangle virus (porcine paramyxovirus)

Menangle virus causes a reproductive disorder in pigs, with foetal death. It has caused mild, flu-like symptoms in humans in contact with affected pigs.

Causative agent

Menangle virus is an unclassified virus within the family Paramyxoviridae.

Hosts

Disease occurs in pigs and humans. The natural hosts of the virus appear to be pteropid bats (flying foxes).

Distribution

The disease has occurred only once, near Menangle in New South Wales, but the virus may be widespread in flying foxes. A large serological survey after the outbreak failed to find any evidence of infection in other Australian pigs.

Method of spread

Close contact between pigs and flying foxes is required. Routes of transmission are unknown.

Disease management

The virus can spread within intensive piggeries. Personal protective equipment and adequate protocols are needed to protect humans working in the immediate proximity. Infection can be eliminated by the separation of breeding, weaning and grower classes to limit vertical transmission while thorough decontamination of buildings and facilities is being undertaken.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for Menangle virus

Menangle virus is not an OIE-listed disease. The disease is self-limiting in pigs, but movement of pigs from or within infected or exposed sheds should be avoided. It is unlikely to spread widely but would have serious effects on affected piggeries and cause some disruption to trade.

The policy is to eliminate infection from affected piggeries while limiting human exposure, using:

- quarantine or a moratorium on movement of pigs from infected farms to prevent disease spread, occupational hazards to workers and perceived health risks to consumers;
- where shed capacity is exceeded, the destruction and sanitary disposal of pigs that would otherwise be turned off; and
- serological testing to monitor the progression of the disease and to indicate elimination of the infection.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- management strategies to eliminate vertical transmission;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to reassure consumers.

Menangle virus is currently included as a Category 3 disease in the EAD Response Agreement. The costs of disease control would be shared 50% by governments and 50% by the relevant industries.
2.19 Nairobi sheep disease

Nairobi sheep disease (NSD) is a tick-borne viral disease of small ruminants that is characterised by a haemorrhagic gastroenteritis.

Causative agent

NSD virus is an RNA virus of the Nairovirus genus in the Bunyaviridae family.

Hosts

The disease occurs in sheep and sometimes in goats.

Distribution

NSD occurs in parts of the African continent. It has never occurred in Australia.

Method of spread

The main vector for the virus is the tick *Rhipicephalus appendiculatus* (not present in Australia), but other species of the *Rhipicephalus* and *Amblyomma* ticks occasionally act as vectors. Transmission by contact does not occur.

Disease management

Because NSD is a tick-transmitted disease, vector control and vaccination can be employed. *Rhipicephalus appendiculatus* is also the principal vector of east coast fever (see Section 2.5). Vaccination can be carried out, but is probably best applied to individual animals or flocks moving from ‘clean’ to endemically affected areas.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia's policy for Nairobi sheep disease

Nairobi sheep disease is an OIE notifiable disease. An uncontrolled outbreak of NSD in Australia would cause serious disruption to the sheep industry.

The policy is to eradicate NSD by:

- treatment with acaricides or by other means to eliminate ticks from affected sheep;
- conduct of a tick eradication campaign;
- strict hygienic precautions, including destruction of contaminated bedding and equipment; and
- movement controls on animals moving into known infected areas, unless their immune status is established.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- a public awareness campaign to encourage cooperation from industry.

NSD is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.20 Nipah virus

Nipah virus disease is a serious viral disease of pigs and humans, with a high case fatality rate. The disease is capable of causing severe economic impacts.

Causative agent

The International Committee for the Taxonomy of Viruses has agreed to name the genus that contains the Hendra and Nipah viruses, *Henipavirus*. The genus is in the Paramyxoviridae family.

Hosts

The species that appears most affected is the pig. The disease also seriously affects humans. Other animals in Malaysia, including horses, cats, dogs and goats, have been infected with the Nipah virus. Flying foxes are the natural hosts.

Distribution

Nipah virus has occurred in peninsular Malaysia. The disease occurred in abattoir workers in Singapore who had been exposed to Malaysian pigs.

Nipah virus has not been detected in Australia.

Method of spread

Nipah virus appears to be easily transmitted between pigs by aerosol, and may be transmitted from pigs to other animals. Means of spread from the natural host to pigs is unknown.

Disease management

Nipah virus is a serious zoonotic disease and is capable of causing severe economic impacts on pig production. It can spread rapidly between pigs and between pig-growing areas by stock movement.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia's policy for Nipah virus infection

Nipah virus disease is an OIE-listed disease. An outbreak in Australia could have serious public health implications and cause disruption to trade.

The policy is to eradicate Nipah virus using:

- destruction and sanitary disposal of all affected and exposed pigs, and all other infected animals;
- quarantine and movement controls on affected piggeries and piggeries in the immediate vicinity;
- protection of humans from infection; and
- decontamination of piggeries.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease
- zoning to define infected and disease-free areas
- a public awareness campaign to encourage cooperation with industry and to reassure consumers.

Nipah virus is currently included as a Category 1 disease in the EAD Response Agreement. The costs of disease control would be borne 100% by governments.
2.21 Potomac fever

Potomac fever is an acute ehrlichial disease, primarily of horses, causing fever, anorexia and depression. The disease is often fatal.

Causative agent

The causal organism of the disease is Ehrlichia risticii. Other Ehrlichia also cause disease, including E. equi, E. sennetsu and E. canis.

Hosts

Horses are the main host, but cats, pigs and goats can be infected.

Distribution

Potomac fever occurs in North America and parts of Europe.

There is some serological evidence of E. risticii in Western Australia and Queensland. However, positive serology has not been linked with clinical disease, and it has not been established whether the positive serology is a result of infection with E. risticii or infection with a related organism.

Method of spread

The disease, which develops in random animals, does not appear to be contagious. Because of this and the seasonal occurrence of the disease, an insect vector such as a fly or tick is suspected of spreading the disease. Horses may remain carriers for at least 40 days.

Disease management

Vaccination of horses is an option for disease management, but a number of disease cases have been reported in vaccinated animals. Vaccination has been reported to protect up to 78% of horses from developing symptoms more severe than fever. Protection is relatively shortlived, so vaccination at four-month intervals is recommended.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia's policy for Potomac fever

Potomac fever is not an OIE-listed disease. If clinical cases were to occur in Australia, they would disrupt the horse industry and cause increased costs for treatment and prevention.

The policy is to eradicate Potomac fever using:

- destruction of infected horses, or isolation of infected horses in a tick-free environment for three months, with release from isolation only after a demonstrated reduction in antibody titre; and
- reduction of vector populations by fogging of the immediate area and by regular treatment of animals in the area to reduce exposure to arthropods.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas;
- use of sterile equipment for intravenous procedures to mitigate the risk of iatrogenic transmission; and
- a public awareness campaign to encourage cooperation from industry and to reassure consumers.

Potomac fever is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.22 Pulmonary adenomatosis

Pulmonary adenomatosis, or Jaagsiekte, is a slowly progressive, neoplastic lung disease of sheep and goats.

Causative agent

The disease is caused by a virus belonging to the family Retroviridae. Ovine herpesviruses had been suggested as possible causes of the disease, but these seem only to be passenger viruses.

Hosts

Pulmonary adenomatosis occurs mainly in sheep and, to a much lesser extent, goats.

Distribution

Pulmonary adenomatosis is present in Europe, Africa, the Middle East, India, China and parts of the Americas.

Ovine pulmonary adenomatosis has never occurred in Australia.

Method of spread

Infection spreads by direct contact between sheep, presumably by means of aerosols or droplets. The disease remains endemic in infected flocks for very long periods. There is a prolonged incubation period from nine months to three years.

Disease management

The disease was eradicated from Iceland through the slaughter of almost all of the sheep population. However, in the absence of a reliable diagnostic test, eradication is unlikely to be economically feasible in other countries. Another option is the establishment of closed flocks free from infection, although the lack of techniques for determining infection hampers this approach.

Susceptibility to infection decreases rapidly after birth. If the lambs of infected ewes are eliminated along with their dams, the prevalence of the disease can be further reduced.

Good management practices can decrease the probability of direct transfer of droplets/aerosols, as the retrovirus is relatively unstable in dry, warm environments.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
Australia’s policy for pulmonary adenomatosis

Ovine pulmonary adenomatosis is not an OIE listed disease. If uncontrolled, the disease usually causes initial heavy losses, but subsequently may cause losses of only 1–3% per year.

The policy is to eradicate pulmonary adenomatosis using:

- slaughter of all sheep on the initially infected property;
- long-term surveillance based on investigation of clinical signs and pathology; and
- if the disease appears to be more widespread, quarantine of affected properties and destruction of clinically affected animals and maternal offspring.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation from industry and to reassure consumers.

Pulmonary adenomatosis is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.23 Sheep scab

Sheep scab is a parasitic skin infestation of sheep that causes papules or pustules and lesions, leading to serious production losses through fleece damage.

Causative agent
Sheep scab is caused by Psoroptes ovis, a small, white mite just visible to the naked eye.

Hosts
The parasite infests sheep and possibly cattle.

Distribution
P. ovis is present in most countries of Europe, Africa, the Middle East, Asia and Central and South America.

The parasite was eradicated from Australia by 1896.

Method of spread
All stages of the lifecycle of P. ovis are completed on the host. The mite is spread by direct contact between animals or by contamination of tufts of wool, fences or soil. It may also be mechanically transmitted by birds. All stages can live away from the host for a period; adult mites can live independently for up to three weeks.

Disease management
Treatment is primarily based on plunge-dipping with organophosphates. An alternative is the use of injectable ivermectin.

Laboratory diagnostic capacity
Samples should be submitted to AAHL, which will arrange their transport to an entomological reference laboratory for definitive laboratory diagnosis.
Australia’s policy for sheep scab

Sheep scab is not an OIE-listed disease. If it occurs in Australia, it will cause considerable disruption to the wool industry and will be expensive to treat.

The policy is to eradicate sheep scab using:

- clinical surveillance, identification and quarantine of infested flocks;
- compulsory treatment of infested flocks using ivermectin-related or other approved products; and
- movement controls.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- zoning to define infected and disease-free areas; and
- a public awareness campaign to encourage cooperation with industry.

Sheep scab is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.24 Swine influenza

Swine influenza is a highly contagious viral disease of the respiratory tract of pigs caused by a type A influenza virus.

Case definition: For the purposes of this Response Policy Brief, swine influenza (SI) is defined as an outbreak of influenza in pigs caused by a type A influenza virus. SI is classically associated with high morbidity and low mortality, but the clinical picture may vary depending on the strain of influenza virus involved (there may be only mild to very mild respiratory signs) and by the presence of other infectious agents.

Causative agents

SI is caused by type A influenza viruses of the family Orthomyxoviridae. Influenza A viruses are respiratory pathogens that can infect a wide variety of animal species.

The three subtypes usually involved in SI are H1N1, H1N2 and H3N2, although influenza viruses are constantly evolving genetically and have the ability to create new subtypes, possibly changing characteristics such as virulence, infectivity and host range. Multiple strains are recognised within these subtypes, and pigs may also be infected by influenza viruses from other species, particularly humans and birds. A recent example is the novel H1N1 virus (called pandemic (H1N1) 2009) that was declared a pandemic in the human population in June 2009. This H1N1 subtype appears to be a genetic reassortment of the established ‘triple reassortant’ SI virus that has been circulating in North America, with two gene segments typical of Eurasian SI viruses (Irvine and Brown 2009). Pigs are susceptible to infection with this subtype.

The zoonotic potential of SI viruses should be kept in mind when planning and implementing the response to an outbreak.

Geographic distribution

SI viruses are endemic in pig populations in most of the world; New Zealand remains free of this disease. A survey of Australian pigs was undertaken in 1976–77 with negative results and a survey conducted by the Northern Australia Quarantine Strategy in 1997–99 found no evidence of SI. In 2009, Australia reported cases of infection with pandemic (H1N1) 2009 virus in 3 piggeries in New South Wales, Victoria and Queensland, most likely due to ‘spill-over’ from the human population.

The disease was first described in the USA in 1918. Prior to the mid-late 1970s, there was little evidence of SI outbreaks in other parts of the world. SI is now endemic in North America and Europe, and outbreaks have been reported in Africa, Asia and South America. Before 1998, SI in the United States was caused by the H1N1 subtype. In late 1998, for the first time, H3N2 viruses caused severe disease in North American pigs; these viruses were triple reassortants of avian, human and swine viruses distinct from earlier strains and European strains. Since then, three distinct genotypes of H3N2 viruses, as well as H1N2 and H4N6 subtypes, have emerged among pigs in the United States and Canada. The H1N2 virus isolates represent a reassortment of the recently emerged H3N2 and the
classical H1N1. The current H3N2 viruses are closely related to human H3N2 viruses.

In Europe, recent monitoring has shown that subtypes H1N1, H3N2 and H1N2 co-circulate in pig populations. Differences in prevalence exist among countries in Europe.

Strains within the various subtypes differ in their ability to cause disease.

**Hosts**

Pigs are the main hosts for SI viruses, but there is increasing evidence of interchange of influenza viruses among pigs and other mammalian and avian hosts, either directly or after a process of genetic reassortment. This may be significant in the emergence of new strains pathogenic to humans.

Human influenza virus genes and avian influenza virus genes are known to reassort with SI virus genes and enrich the pool of viruses circulating in birds, pigs and humans. Through their susceptibility to both avian and mammalian type influenza viruses, pigs may act as a ‘mixing vessel’ for genetic reassortment of RNA from human, swine and avian viruses (Ma et al 2009), and as a result, genetic diversity of SI viruses has increased over time. In addition, the exchange of viral RNA can occur in both directions, with human influenza virus transmitted to pigs. Similarly, influenza virus can be transmitted from poultry to pigs as well as from pigs to poultry.

The incubation period for SI in pigs is usually 1 to 3 days. Pigs begin excreting the virus within 24 hours of infection, and may shed the virus for 7 to 10 days (OIE 2009, AVMA 2009), although peak shedding occurs around 48–72 hours. In naive herds, SI typically presents as an acute to chronic respiratory disease and infected pigs may show fever, anorexia, weight loss, coughing, sneezing, nasal discharge, and respiratory difficulty. Infection can also occur with no clinical syndrome evident. Reports have indicated that an early sign in commercial piggeries is a fall in feed consumption. Swine influenza may cause reproductive problems in sows, depending on the stage of gestation at infection. While all pigs in a herd may become sick, case mortality rates are generally low (1–3%); in the absence of complications, most affected pigs will recover within five to seven days. Severe bronchopneumonia may develop as a complication and is a high risk factor for mortality.

In the endemic form, there may be intermittent bouts of disease and infertility, and different strains of virus may sequentially infect the herd. Pigs naturally infected with SI virus develop protective immunity to challenge from the same subtype (Gramer 2009), but immunity to influenza viruses is often short lived (6 months) and the immunity profile in a breeding herd may vary considerably over time. There is little evidence of a true long-term carrier state in pigs (OIE 2009).

It is highly unlikely that Australian pigs would have any significant immunity to influenza viruses. In the small number of piggeries in Australia on which infection with the pandemic (H1N1) 2009 virus has been confirmed, the disease has spread quickly and pigs have generally exhibited mild respiratory clinical signs. In a study conducted in the UK, pigs shed the virus mainly via the nasopharyngeal route from 1 to 9 days post-inoculation; peak shedding occurred between 3 and 5 days post-inoculation (VLA 2009). Oral and ocular shedding was detected...
intermittently, but neither faecal shedding nor viraemia was detected; all shedding appeared to have ceased by 10 days post-inoculation. Mortality was not a feature and infected animals were able to transmit the virus to naive contact pigs successively for at least 3 cycles of transmission, suggesting that the virus could become established in susceptible pig populations if introduced.

SI viruses occasionally infect humans and, until the H1N1 2009 pandemic, most influenza in humans caused by swine subtypes was associated with occupational exposure. Most patients recovered swiftly and there were few fatalities. The symptoms found in humans infected with SI viruses resemble seasonal influenza; ie fever, lethargy, lack of appetite and coughing (CDC 2009). It would be beneficial for a strategy for maintaining seasonal influenza vaccination status in humans at risk (such as piggery and abattoir workers, and transporters) to be agreed with public health authorities.

Method of spread

SI is primarily transmitted among pigs in close contact through nasal discharges and aerosols from sneezing and coughing.

The main method of spread of SI is through the movement of infected pigs. While influenza viruses are associated with viraemia and faecal shedding in some species, this has not been seen in SI due to natural infection. The inactivation period for some swine viruses in slurry kept at different temperatures has been investigated (Bøtner 1990); the inactivation time for SI virus was 9 weeks at 5°C, 2 weeks at 20°C, and more than 24 hours at 35°C. Therefore, the virus could survive in slurry for a significant period of time, particularly at cool temperatures.

In pigs, the influenza virus is not found outside the respiratory tract and associated lymph nodes, and there are no reports of the agent being found in semen (AQIS 2000, VLA 2009). Therefore, it would be highly unlikely that known influenza viruses could be transmitted in pork or pork products. This is in line with the Joint FAO/WHO/OIE Statement of 7 May 2009 on pandemic (H1N1) 2009 and the safety of pork.7

Humans and birds may also be a source of infection for pigs, and pigs for humans and birds. For example, seasonal strains of human influenza A viruses can infect pigs but usually don’t spread widely, and pandemic (H1N1) 2009 disease in one Canadian commercial piggery in April–May 2009 and one Argentinean piggery in June 2009 may have been introduced by infected humans. Transmission by infected piggery workers was also the most likely cause of the Australian outbreaks. The risk of pandemic (H1N1) 2009 being introduced to other piggeries in Australia is likely to be strongly associated with the prevalence of infection in the human population.

Whether swine influenza becomes endemic within a swine herd will depend on agent infectivity, the number and susceptibility of naive pigs, pig density etc. Farrow to finish operations are likely to be of higher risk, as infection is maintained due to the regular farrowing of sows within a herd, maintaining a population of

7 www.oie.int/eng/press/en_090507_bis.htm
naive animals. ‘Open’ herds (where live pigs are purchased from external sources), those populations with a lot of movement between groups and a higher rate of contact, and those with animals at higher stocking rates are more likely to maintain infection within them. Commercial herds are most at risk for this, especially larger herd operations. Large herds, with separation of animal buildings and separate airspaces, are likely to create pockets of infected and naive animals, further adding to the risk of persistence. Annual outbreaks are likely to occur within affected herds during the colder months.

Mammalian influenza viruses seem to be relatively labile, but can persist for several hours in dried mucus. They can be inactivated by heating at 56°C for a minimum of 60 minutes (or higher temperatures for shorter periods) and by low pH (pH 2). Influenza viruses are susceptible to a wide variety of disinfectants including sodium hypochlorite, 70% ethanol, oxidizing agents, quaternary ammonium compounds, aldehydes (formalin, glutaraldehyde, formaldehyde), phenols, acids, povidone-iodine and lipid solvents.

**Diagnostic tests**

For index cases, samples should be submitted to the state laboratory for exclusion testing and to AAHL for confirmation.

Appropriate samples should include nasal, oral or respiratory swabs from active clinical cases (preferably within 48 hours of the development of clinical signs)\(^8\) and serum samples from recovered cases.

**Managing the risk**

The following factors are relevant in managing the disease risks associated with SI (including zoonotic risks):

- prior registration of all piggeries with the relevant agricultural authority;
- quality of surveillance on piggeries, especially smallholdings;
- presence of biosecurity programs on piggeries (including non-commercial pig holdings), using guidelines developed by an appropriate industry body;
- importance of effective biosecurity to address highly transmissible nature of pathogen;
- lack of awareness of risk factors among smallholders;
- difficulty of determining quickly the likely source of the pathogen;
- the strain of SI virus involved, and whether other respiratory pathogens are present in the herd;
- ability to prevent exposure of susceptible pigs to infected domestic or feral pigs, or to infected birds or humans (from influenza viruses endemic in these populations), through on-farm biosecurity and/or movement controls;
- farm biosecurity programs on poultry premises in the vicinity of outbreaks in pigs, using guidelines developed by an appropriate industry body;

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\(^8\) [www.oie.int/eng/normes/mmanual/2008/pdf/2.08.08_SWINE_INFLUENZA.pdf](http://www.oie.int/eng/normes/mmanual/2008/pdf/2.08.08_SWINE_INFLUENZA.pdf)
• knowledge of and ability to manage feral pig populations;
• the need to humanely destroy healthy pigs on welfare grounds to address commercial problems such as markets or abattoirs refusing to accept pigs from certain premises;
• availability of vaccines early in a response; inactivated vaccines for some SI strains are registered for use in pigs overseas but not in Australia, and may not prevent infection or viral shedding; effective strain-specific human and pig vaccines are unlikely to be available at the beginning of an outbreak, but could be developed as part of the response;
• absence of risk from pork and pork products; when handled in accordance with routine hygienic practices, these are not a source of infection;
• the need for various strategies to be applied sequentially or in parallel, and over a period of months, in order to contain the outbreak and achieve eradication (if feasible);
• communication and cooperation with public health authorities;
• risk to humans and birds of infection from some SI strains; addressing human health risks during a response (such as through vaccination of piggery workers and issuing of PPE) would need to be coordinated by local public health authorities;
• appropriate management of staff ill with influenza-like symptoms;
• use of effective personal and environmental hygiene and sanitation practices to inactivate influenza viruses and/or remove them from the environment; influenza viruses are quickly destroyed by soap and detergents.

When the relevant chief veterinary officer determines that an influenza outbreak in pigs is caused by a type A influenza virus, an assessment of the risks to animal and public health should be carried out, taking into account the factors above, the virus subtype, the clinical status of the pigs and their proximity to commercial or other pig establishments and populations, and to public amenity areas and poultry operations.

Piggery and poultry farm managers and their veterinarians are responsible for instituting and maintaining farm biosecurity measures to minimise the likelihood of influenza viruses entering the premises and to help the relevant authorities deal with an influenza outbreak if it occurs. Any influenza-specific measures should be integrated into routine farm biosecurity programs.9 For example, farm/piggery workers should be urged not to go to work if they have any signs of respiratory disease or fever, and pig handlers and veterinarians should wear protective clothing to minimise the risk of being infected and of transmitting infection. Such persons may need to be directed to receive influenza vaccination as part of a program to reduce the likelihood of outbreaks of influenza in pigs or as part of a response to an outbreak, as coordinated by public health authorities.

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Australia’s policy for swine influenza

This response policy addresses an outbreak in pigs of swine influenza (SI) caused by a type A influenza virus.

SI is not an OIE-listed disease but it is a notifiable disease in Australia; it can cause serious disease in pigs and illness in humans. An uncontrolled outbreak is likely to cause acute respiratory disease and exacerbate other respiratory diseases in pigs, and cause significant economic loss. It would also raise human health concerns.

Swine influenza is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.

The response policy with regard to an outbreak of SI will be determined by how early the outbreak is detected, the extent of the outbreak, the location of affected premises, the strain of SI virus involved, and whether other respiratory pathogens that could complicate the clinical picture are present.

The default policy is to contain the spread of SI, until the outbreak(s) dies out or has been eradicated. This policy would be implemented by a combination of strategies such as:

- early recognition and laboratory confirmation of cases, to clarify the situation with regard to virus subtype, pathogenicity, transmissibility and likely host susceptibility;
- tracing and surveillance (based on an epidemiological assessment) to determine the source and extent of infection, including in humans, birds and feral pigs if justified by risk assessment;
- enhanced biosecurity at pig and poultry premises in the vicinity;
- movement controls over pigs, poultry, people and fomites (eg facilities, vehicles, equipment, feed) on affected premises, to minimise the spread of infection; on closed pig premises, movement controls will continue until the premises is determined to be free according to an agreed protocol appropriate to the epidemiological situation (this may be a combination of the elapse of a minimum time period after the last clinical signs, the use of sentinel animals, and laboratory testing); movement controls over people will aim to minimise virus transmission between humans and pigs;
- targeted management of feral pig populations in the vicinity, if justified by risk assessment;
- zoning/compartmentalisation to define infected and disease-free areas/premises;
- process slaughter of pigs free from clinical signs (at least 7 days after the final appearance of any clinical signs in the group of pigs to be slaughtered);
- if necessary, destruction of some or all pigs for animal welfare reasons as a result of markets or abattoirs not accepting pigs from certain premises;
- decontamination of fomites to eliminate the pathogen;
- spelling of any depopulated premises to eliminate the pathogen;
in certain circumstances, vaccination of pigs to help control the spread of infection within and between premises;

- enhanced personal and environmental hygiene and sanitation practices;

- *industry support* to enhance understanding of the issues, to facilitate cooperation, and to address on-farm biosecurity and animal welfare issues; and

- a *public awareness campaign* to explain the control measures adopted, publicise the zoonotic aspects and their management, encourage community participation in personal disease control measures, and promote the safety of pork products.

In a situation in which SI is considered not able to be contained or eradicated within an acceptable time period, the policy for long-term management of the disease will be determined following consultation between the government and the pig industry. The policy adopted may involve enhanced biosecurity, surveillance, vaccination and long-term compartmentalisation under an industry program. For example, in a situation where there is a reservoir in the human population and little effect on pigs (eg pandemic (H1N1) 2009), the policy will be one of minimal intervention, consistent with animal health, public health, and industry and marketing requirements.

Surveillance, PPE, antiviral prophylaxis and/or vaccination for people (such as piggery and abattoir workers) with occupational exposure to pigs likely to be infected, to address the zoonotic aspects of swine influenza, may be appropriate to the response. This would be the responsibility of public health authorities to determine.

**Further information**

2.25 Teschen disease

Teschen disease is caused by a highly virulent strain of porcine enterovirus. The virus causes a porcine polioencephalomyelitis with high morbidity and high mortality. Polioencephalomyelitis caused by this strain of the virus is covered by the EAD Response Agreement.

Other porcine enteroviruses also cause forms of porcine polioencephalomyelitis, including Talfan disease and benign enzootic paresis. However, disease in these cases is milder, more sporadic and less contagious, and is not covered by the EAD Response Agreement.

Diseases caused by porcine enteroviruses have recently been grouped under the general disease name of ‘enterovirus encephalomyelitis’.

Causative agent

The viruses that cause porcine polioencephalomyelitis belong to the *Enterovirus* genus of the family Picornaviridae. There are eleven serotypes of porcine enteroviruses. Teschen disease virus belongs to serotype 1 (PEV1).

Hosts

Pigs are the natural hosts.

Distribution

Teschen disease is found in parts of Europe and in Madagascar.

The disease has never been reported in Australia, but the milder Talfan disease, which is caused by a less virulent serotype 1 porcine enterovirus, is present.

Method of spread

The disease is highly contagious, with infected pigs excreting virus in their faeces and oral secretions. The virus can survive in the environment for 3–4 weeks, and infection can be spread by direct or indirect contact. Swill feeding is also a means of spreading the virus.

Disease management

Attenuated and inactivated vaccines can be used to immunise pigs.

Laboratory diagnostic capacity

Samples should be submitted to AAHL or the relevant SCAHLS-endorsed state/territory reference laboratory for definitive laboratory diagnosis.
Australia’s policy for Teschen disease

Enterovirus encephalomyelitis (including Teschen disease) is not an OIE listed disease. It causes high morbidity and mortality in pigs. Teschen disease is highly contagious, and uncontrolled outbreaks could cause very severe production losses in affected herds. However, the disease usually disappears of its own accord.

The policy is to eradicate Teschen disease using:
- identification and eradication of infected herds;
- quarantine and movement controls;
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease; and
- vaccination of infected herds in certain circumstances.

Teschen disease is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.26 **Trichinellosis**

Trichinellosis is a helminth disease of mammals causing gastrointestinal symptoms, fever, muscle pains, weakness and respiratory symptoms. The disease has human health implications.

**Causative agent**

The disease is caused by the nematode parasite *Trichinella spiralis*.

**Hosts**

All mammals are susceptible, but infestation is more common in omnivores and carnivores. Among livestock species, pigs are the most important host, followed by dogs, cats and horses. Trichinellosis is primarily a public health problem. It is not recognised as a clinical disease in pigs, and is usually only diagnosed at slaughter.

**Distribution**

The nematode is found in temperate areas of the world, including North and South America, eastern Europe, Spain, the former Soviet Union, parts of the Middle East, Central and Southeast Asia, Africa and the North Island of New Zealand.

*Trichinella spiralis* has never been diagnosed in animals in Australia.

**Method of spread**

Encysted larvae in muscle tissue are ingested by new hosts. Feeding of livestock with materials contaminated by rat or mouse carcasses allows for transmission. In pigs, transmission is also possible via larvae excreted in the faeces.

**Disease management**

The incidence of trichinellosis in pigs in most countries has declined sharply with the introduction of modern intensive husbandry systems (which have removed sources of contamination introduced with rodents). Several effective drugs are available to treat trichinellosis, such as mebendazole, but are rarely used in animals. Partial immunity may develop from previous infection.

**Laboratory diagnostic capacity**

Samples should be submitted to AAHL, which will arrange their transport to an appropriate reference laboratory for definitive laboratory diagnosis.
Australia's policy for trichinellosis

Trichinellosis is an OIE notifiable disease. While *Trichinella spiralis* has never been diagnosed in animals in Australia, a form of trichinellosis due to *Trichinella pseudospiralis* has been detected in wildlife in Tasmania. This form of the disease in wild pigs in Thailand and France has recently been reported to be pathogenic for humans.

The policy is to eradicate trichinellosis by:
- destruction of infected and potentially infected carcasses;
- quarantine of infected herds so that the location of infected animals is known at all times; and
- rodent control on infected and suspect piggeries.

These strategies will be supported by:
- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- surveys of wildlife within affected areas;
- meat inspection to examine for *Trichinella* from infected and suspect piggeries;
- abattoir surveys of culled sows; and
- a public awareness campaign to encourage cooperation from industry and restore consumer confidence.

Trichinellosis is currently included as a Category 3 disease in the EAD Response Agreement. The costs of disease control would be shared 50% by governments and 50% by the relevant industries.
2.27 Vesicular exanthema

Vesicular exanthema (VE) is an acute disease of pigs characterised by the formation of vesicles that are clinically indistinguishable from those caused by foot-and-mouth disease (FMD).

Causative agent

VE is caused by a virus belonging to the family Caliciviridae, which contains animal and human pathogens.

Hosts and clinical signs

Pigs are the only domestic animals in which natural outbreaks of clinical disease have occurred. A similar disease occurs in pinniped marine mammals such as sea lions, fur seals and elephant seals, which are probably the natural reservoir of VE viruses. Human cases of VE have not been reported.

VE virus enters the host through damaged epithelia, usually the skin of the feet or snout, or the oral mucosa. In the field, clinical signs of VE are very similar to those of FMD, vesicular stomatitis and swine vesicular disease. Morbidity in pigs is high but the case mortality rate is very low except in young piglets. The earliest clinical sign of VE is a marked fever, usually within 1–3 days of infection, with the pigs being lethargic, not eating and unwilling to stand. Sows may abort and lactating sows may stop producing milk. The disease may not be noticed in a herd until lameness is obvious and vesicles (up to 30 mm in diameter) are seen on the snout and in the mouth (on the lips, gum or tongue, causing slobbering and chomping); on the soles, the skin between the toes, cuticle and claws; and occasionally on the teats or udder. In some outbreaks, the foot lesions may predominate and in other outbreaks they may be insignificant. Many pigs recover quickly and uneventfully. In other cases, complications may occur as a result of secondary bacterial infection.

Distribution

The disease first appeared in California in 1932 and was eradicated from the United States in 1956. The occurrence of VE outside mainland USA has been reported on two occasions: in pigs being transported to Hawaii in 1946–47 and on a US military base in Iceland in 1955. On both occasions the animals were promptly destroyed. While related marine caliciviruses have been identified along the Pacific seaboard of North America, VE has not been reported recently in pigs anywhere in the world.

VE has never been identified in pigs in Australia.

Method of spread

The feeding of swill contaminated with material from infected marine mammals or pigs is the principal means of spread. Movement of infected pigs is a major cause of secondary spread of the disease. The most likely way the disease could be introduced into Australia is via uncooked swill, the feeding of infected imported fishmeal to pigs, or perhaps by feral pigs scavenging dead marine animals on the seashore. The virus is fairly resistant to environmental inactivation.
The incubation period in natural outbreaks is usually 1–3 days, although extremes of 12 hours to 12 days have been observed.

**Diagnosis**

Virus is easily isolated during the early acute phase of disease when vesicles are still present.

Specimens required include:

- from live animals — vesicular fluid, epithelial coverings or flaps from vesicular lesions, whole blood, sera; and
- from recently dead animals — fresh and formalised samples of several tissues, including brain.

**Managing the risk**

Managing the risks associated with VE relies on the following factors:

- rapid diagnosis to differentiate it from FMD;
- registration of all piggeries and mandatory biosecurity procedures (including for non-commercial pig holdings); and
- preventing exposure of susceptible pigs to infected pigs, and potentially contaminated pig and marine mammal products.

The action taken, at least initially, will depend on the circumstances, including the number and size of premises affected, the presence of feral pigs in the immediate area, the likelihood of other industries being affected by concerns over the accuracy of the diagnosis, and the design and operation of the affected premises with respect to biosecurity.

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**Australia’s policy for vesicular exanthema**

This policy applies to commercial piggeries, non-commercial pig holdings and, when relevant, feral pig populations.

VE is not an OIE-listed disease, but is considered an important disease of pigs because it can be confused in the field with FMD. A delay in the definitive diagnosis of VE would have a major effect on international trade for a range of animal products, especially beef, until FMD has been excluded. If VE becomes established, ongoing recurrent outbreaks would result in periodic disruption to our international markets.

VE is an Animal Health Australia Category 3 disease under the EAD Response Agreement for cost-sharing arrangements. Category 3 diseases are those for which costs will be shared 50% by government and 50% by industry.

The initial policy is to limit the spread of VE until a definitive diagnosis is made. This would be supported by a combination of strategies including:

- early recognition and laboratory confirmation of cases to differentiate it from FMD;
• quarantine and movement controls over pigs, people, fomites (vehicles, equipment, feed) and pig products, on affected premises to minimise the spread of infection; and

• tracing and surveillance (based on an epidemiological assessment) to determine the source and extent of infection including in feral pigs.

Once a definitive diagnosis of VE has been made, *stamping out* would be the preferred option if only small numbers of pigs are involved. This would be supported by a combination of strategies including:

• quarantine and movement controls over pigs, people, fomites (vehicles, equipment, feed) and pig products, on affected premises to minimise the spread of infection;

• decontamination of facilities and fomites to eliminate the virus on infected premises and to minimise spread;

• sanitary disposal of destroyed pigs and contaminated pig products, to reduce the source of infection; and

• a public awareness campaign to facilitate cooperation from industry and the community.

If large numbers of pigs are involved and effective movement controls can be maintained, the policy would be to closely monitor the pigs for signs of disease or seroconversion supported by the following strategies:

• quarantine and movement controls over pigs, people, fomites (vehicles, equipment, feed) and pig products, on affected premises to minimise the spread of infection;

• tracing and surveillance (based on an epidemiological assessment) to determine the source and extent of infection;

• zoning/compartmentalisation to define infected and disease-free areas / premises;

• process slaughter of animals free from clinical signs; and

• a public awareness campaign to facilitate cooperation from industry and the community.
2.28 Wesselsbron disease

Wesselsbron disease is an acute mosquito-borne viral disease that causes, among other things, relatively high mortality in newborn lambs and kids and flu-like symptoms in humans.

Causative agent

Wesselsbron disease virus is a member of the Flavivirus genus of the Togaviridae family.

Hosts

The natural disease has been reported only in sheep, goats and humans.

Distribution

Wesselsbron disease has been reported only in South Africa, but the disease virus is present in most of sub-Saharan Africa.

The disease has never occurred in Australia.

Method of spread

The virus has been isolated from several species of mosquito, but Aedes spp. appear to be the main vectors in South Africa. Most, if not all, human cases of the disease have been laboratory acquired.

Disease management

Immunisation of livestock with attenuated vaccine is the only effective method of control.

Laboratory diagnostic capacity

Samples should be submitted to AAHL, which will arrange their transport to an overseas reference laboratory for definitive laboratory diagnosis.
### Australia’s policy for Wesselsbron disease

Wesselsbron disease is not an OIE-listed disease. While the disease has never occurred in Australia, it is highly probable that suitable mosquito vectors exist throughout the country. It is extremely unlikely that this disease could be eradicated once established.

The policy is to control Wesselsbron disease by:

- vaccination of at-risk animals;
- quarantine and movement controls of infected animals; and
- vector abatement to reduce mosquito populations.

These strategies will be supported by:

- tracing and surveillance to determine the source and extent of infection and to provide proof of freedom from the disease;
- prevention of iatrogenic transmission by using sterile equipment in intravenous procedures; and
- a public awareness campaign to encourage cooperation from industry.

Wesselsbron disease is currently included as a Category 4 disease in the EAD Response Agreement. The costs of disease control would be shared 20% by governments and 80% by the relevant industries.
2.29 West Nile virus disease

Causative agent

West Nile virus (WNV) is the causative agent of WNV fever/encephalitis. WNV is a mosquito-borne arbovirus in the genus *Flavivirus*, family *Flaviviridae*.

Hosts

Wild birds are the reservoir hosts for WNV. In regions with endemic disease, WNV is maintained in an enzootic cycle between culicine mosquitoes and birds. When environmental conditions favour high viral amplification, significant numbers of mosquitoes that feed on both birds and mammals can spread the virus to humans and other incidental hosts. Migratory birds may carry WNV into new areas. Infected humans and horses are considered to be ‘dead-end’ hosts, since viral titres in blood are generally too low to allow transmission to vectors. The incubation period in horses and humans is estimated to be 3–15 days, and for avian species around 7–10 days.

Many birds carry the virus without clinical signs, but high levels of mortality have been seen in corvids (which includes crows, ravens, jays and magpies) in the Northern Hemisphere. Affected wild birds are usually found dead, and myocarditis and encephalitis may be found on postmortem examination. Prior infection of avian species with closely related flaviviruses may provide some cross-protection.

Among mammals, neurologic disease has been reported in humans and horses, as well as in a range of other domestic and wild animals, including cats, dogs, rabbits and deer.

A vireaemia of low virus titre precedes clinical onset in horses. Clinical signs associated with eventual death or euthanasia include excitability, ataxia, falling down, recumbancy, abnormal gait, muscle fasciculations, lip droop, head pressing, lethargy, sweating, seizures and hyperesthesia. Cases may also be mild, with only incoordination or mild muscle fasciculations observed. Fever is not often observed. Many cases are asymptomatic.

In humans, most cases of WNV disease are not associated with clinical illness, but approximately 20% of those infected develop disease, including mild flu-like symptoms with fever, weakness, and head and body aches. An erythematous skin rash occasionally develops. Most uncomplicated infections resolve in 3–6 days. In more severe cases, there may be encephalitis and/or meningitis, a high fever, disorientation, convulsions, severe muscle weakness, ataxia and coma. In some outbreaks, myocarditis, pancreatitis and hepatitis occur. Neurologic disease is more likely to develop in people older than 50 years. The case mortality rate in outbreaks ranges from 4 to 14%, with a higher rate among older patients.

Distribution

WNV was first identified in Uganda in 1937. Outbreaks were later detected in Eastern Europe, the Mediterranean region, west and central Asia, and the Middle East. Avian mortalities were not associated with these outbreaks until an outbreak occurred in 1998 in domestic geese in Israel. WNV has since caused widespread
clinical disease in avian species, humans and horses in North America, and sporadic cases of disease in horses in Europe and the Middle East. Despite evidence of WNV transmission in southern Africa and Central and South America, clinical disease does not appear to be a major concern there.

Kunjin virus, which is now classified as a subtype of WNV, is endemic to Australia. However, the WNV strains currently causing disease in the eastern and western hemispheres have not been detected in Australia.

Routine surveillance is not sensitive for low levels of WNV transmission. WNV may be present continuously or sporadically in many parts of the world, and detection of the virus would be difficult, if not impossible, in the absence of outbreaks of disease in animals or humans.

**Method of spread**

WNV is transmitted by mosquitoes. *Culex* spp. appear to be the most important vectors in other countries, and studies are continuing on likely Australian vectors. Since Kunjin, Japanese encephalitis and Murray Valley encephalitis viruses are present in Australia, it is likely that suitable mosquito vectors for WNV exist throughout the country.

**Diagnostic tests**

Samples should be submitted to AAHL or the relevant state/territory reference laboratory for definitive laboratory diagnosis. Tests available include virus isolation, molecular testing, serology, immunofluorescent staining, immunohistochemistry, in situ hybridisation, and antigen capture enzyme-linked immunosorbent assay (ELISA).

Success in recovery of virus by isolation techniques will depend on the collection of appropriate tissues coincident with higher levels of virus replication. In general, recovery of virus is more common from affected birds than from affected horses. Brain and spinal cord are the preferred tissues for virus isolation from horses. In birds, kidney, heart, brain or intestine can yield virus. Tissues should also be tested for viral RNA using reverse-transcription polymerase chain reaction (RT-PCR). Nucleic acid sequencing should be used to confirm the identity of virus isolates and products of RT-PCR positive tissues.

Direct detection of antigen by capture ELISA or by immunofluorescent/immunoperoxidase staining may be useful under certain circumstances.

Demonstration of virus-specific IgM antibodies in serum by ELISA (IgM antibody capture or MAC ELISA) is a useful serological marker for recent infection in horses. However IgM antibodies may persist for several months and do not indicate an active infection. Tests that detect antibody by competitive ELISA or plaque reduction neutralisation (PRN) are more commonly used for identifying antibody in avian sera.

Closely related flaviviruses may exhibit serological cross-reactivity. Therefore, these viruses should be ruled out when conducting diagnostic tests for WNV.
Disease management

Broad-scale animal movement controls and environmental vector control have not proven effective in other geographic regions.

Reducing the exposure of human, mammal and avian species to mosquito vectors is the primary method of disease management. Although highly effective vaccines are available for equine species, WNV vaccines for use in humans and avian species have not yet reached the market.

Efforts to control the mosquito populations involved in WNV transmission may reduce exposure locally; however, broad-scale larvacide and adulticide treatments have proven ineffective and extremely costly in most cases.

Treatment for humans and horses exhibiting clinical signs associated with WNV is primarily supportive.

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**Australia’s policy for West Nile virus (WNV) disease**

WNV is an OIE-listed disease. Kunjin virus, which is now classified as a subtype of WNV, is endemic to Australia. The WNV strains currently causing disease in the eastern and western hemispheres, however, have not been detected in Australia. Since Kunjin, Japanese encephalitis and Murray Valley encephalitis viruses are present in Australia, it is likely that suitable mosquito vectors for WNV exist throughout the country.

Due to the wild bird reservoir and the transmission of WNV by mosquito vectors, eradication is extremely unlikely to succeed if the virus becomes established in an enzootic cycle in Australia.

If an outbreak of WNV disease is detected in an imported animal(s) or bird(s) and the virus is not considered to have become established, the policy is to consider eradication by:

- movement controls over the infected imported animal(s) or bird(s); and
- vector abatement to minimise mosquito numbers on the premises.

If the virus is considered to be established when detected (for example, in the case of an outbreak in wild birds), eradication would not be feasible, and efforts would be directed to surveillance and control strategies, including:

- surveillance of wild bird populations (particularly of sick and dead birds) in the outbreak area, to determine the source and geographic extent of infection;
- surveillance of mosquito vector populations;
- vector avoidance measures, including insect-proof housing for animals that may otherwise be exposed to infected vectors;
- vaccination of high-risk groups such as horses; and
- a public awareness campaign, in conjunction with human health authorities, to help prevent human infections.

WNV disease is not currently included in the EAD Response Agreement.
Appendix 1 Guidelines for classifying declared areas

Premises

Infected premises (IP)

An IP is a premises on which an emergency animal disease (EAD) meeting the case definition exists, or the causative agent of the disease exists, or there is reasonable suspicion that either exists. An IP will be subject to quarantine served by notice and to eradication and control procedures.

Dangerous contact premises (DCP)

A DCP is a premises that, based on a risk assessment, is considered highly likely to contain an animal(s) infected with an EAD or contaminated animal products, wastes or things. In most cases, the restricted area (RA) would be drawn around DCPs. The risk assessment would consider factors such as the stage of the response, the epidemiology of the disease, the animal(s) present and the local situation. Although the susceptible animal(s) on such premises are not showing clinical signs, they are considered to have been significantly exposed to the disease agent — this might be via an infected animal(s) or contaminated animal products, wastes or things.

Since a DCP presents an unacceptable risk to the response if the risk is not addressed, such premises are a high priority for investigation and action. An investigation of a DCP may produce the following outcomes:

- If the presence of an infected animal or contaminated animal products, wastes or things is confirmed, the premises would be designated as an IP.
- If their presence is not confirmed but the likelihood is considered to remain high, the premises would continue to be designated as a DCP.
- If, over the course of the response, it is considered unlikely that an infected animal or contaminated animal products, wastes or things are present, the premises would receive the qualifier assessed negative (AN). However, if it is located in the RA, it would be designated as an at-risk premises (ARP).

When the required control measures for a DCP have been completed, the premises would be designated as a resolved premises (RP) or one with a vaccination qualifier. Such premises are still subject to certain procedures if they are located in a declared area; the procedures would be appropriate to the declared area (RA or CA) in which the premises is located.

Suspect premises (SP)

SP is a temporary designation applied to premises that contain a susceptible animal(s) not known to have been exposed to the disease agent but showing clinical signs that require investigation. The RA should contain as many SPs as practical. The investigation may produce the following outcomes:

- If the case definition is confirmed, the premises would be designated as an IP.
• If the case definition is not confirmed but suspicion remains, the premises would continue to be designated as an SP.

• If the case definition is ruled out, the premises would receive the qualifier AN. However, if it is located in the RA, it would be designated as an ARP. If it is located in the CA, it would be designated as a POR.

**Trace premises (TP)**

TP is a temporary designation applied to premises that contain a susceptible animal(s) that tracing indicates may have been exposed to an infected animal(s) or contaminated animal products, wastes or things, and that requires investigation. Exposure may be via aerosol, especially if the premises is contiguous with an IP, or via fomites. The investigation may produce the following outcomes:

• If the case definition is met, the premises would be designated as an IP.

• If it appears highly likely, as a result of an assessment of the risk that the disease is present in the specific epidemiological situation, that the TP contains an infected animal(s) or contaminated animal products, wastes or things, it would be designated as a DCP.

• If the trace proves to be insignificant, the premises would receive the qualifier AN. However, if it is located in the RA, it would be designated as an ARP. If it is located in the CA, it would be designated as a POR.

**At-risk premises (ARP)**

An ARP is a premises in an RA that contains a susceptible animal(s) but is considered at the time of designation not to be an IP, DCP, SP or TP. The animal(s) on such premise(s) are subject to procedures such as heightened surveillance and movement restrictions. This designation provides authorities with power over such premises, facilitates tracking and serves as a communication tool for reporting nationally and internationally on progress in the response.

**Premises of relevance (POR)**

A POR is a premises in a CA that contains a susceptible animal(s) but is considered at the time of designation not to be an SP or a TP. The animal(s) on such premises are subject to the procedures, such as heightened surveillance and movement restrictions, that are applicable in the CA.

**Resolved premises (RP)**

An RP is an IP or a DCP that has completed the required control measures and is subject to the procedures and restrictions appropriate to the area in which it is located.

**Unknown status premises (UP)**

A UP is a premises that has been identified as having an unknown animal status.

**Zero susceptible stock premises (ZP)**

A ZP is a premises that contains no susceptible animals.
Areas

Restricted area (RA)

An RA will be a relatively small declared area (compared with a control area, or CA) around IPs that is subject to intense surveillance and movement controls. Movement out of the area will be prohibited except under permit. Multiple RAs may exist within one CA.

The RA does not need to be circular but can have an irregular perimeter provided the boundary is initially an appropriate distance from the nearest IP, DCP or SP. This distance will vary with the size and nature of the potential source of disease agent, but will be approximately 1–5 km around the IP, depending on the density of premises. The boundary could be the perimeter fence of the IP if the IP is in an isolated location. The boundary in a densely populated area will take into account the distribution of susceptible animals, traffic patterns to markets, service areas and abattoirs, and areas that constitute natural barriers to movement.

Control area (CA)

The CA will be a larger declared area around one or more RAs and, initially, possibly as large as a state or territory, in which restrictions will reduce the risk of disease spreading from the RAs. The boundary of the CA will be adjusted as confidence about the extent of the outbreak increases but must remain consistent with the OIE Terrestrial Code chapters on surveillance and zoning (Chapters 1.3.5 and 1.3.6; see Appendix 3). In general, surveillance and movement controls in the CA will be less intense, and animals and products may be permitted to move under permit from the area.

The declaration of a CA also helps to control the spread of the outbreak from within the RA. The CA is a buffer zone between the RA and the rest of the industry. The boundary does not have to be circular or parallel to that of the RA but should be 2–10 km from the boundary of the RA. In general, the movement of possibly contaminated items and materials within the CA is allowed but movement out of the CA is prohibited without CVO approval. This type of control area allows reasonable commercial activities to continue.

Outside area (OA)

The OA is not a declared area but is used to describe the rest of Australia outside the declared areas. The OA will be subject to surveillance. As it is highly desirable to maintain the OA as ‘disease free’, the movement of animals and commodities from the RA and CA into the OA will generally be controlled.
# Appendix 2 Emergency animal diseases covered by AUSVETPLAN disease manuals

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>Notifiable to OIE</th>
<th>EAD category</th>
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<tbody>
<tr>
<td>African horse sickness</td>
<td>+</td>
<td>3</td>
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<tr>
<td>African swine fever</td>
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<td>3</td>
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<tr>
<td>Anthrax</td>
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<td>Aujeszky's disease</td>
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<td>Bee diseases and pests</td>
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<td>Brucellosis (caused by <em>Brucella abortus</em>)</td>
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<td>Classical swine fever</td>
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<tr>
<td>Transmissible gastroenteritis</td>
<td>+</td>
<td>4</td>
</tr>
<tr>
<td>Vesicular stomatitis</td>
<td>+</td>
<td>2</td>
</tr>
</tbody>
</table>

*a* + = notifiable disease; – = not notifiable (OIE)

Appendix 3  OIE animal health code and diagnostic manual for terrestrial animals

OIE Terrestrial Code

The objective of the OIE Terrestrial Animal Health Code is to prevent the spread of animal diseases, while facilitating international trade in live animals, semen, embryos and animal products. This annually updated volume is a reference document for use by veterinary departments, import/export services, epidemiologists and all those involved in international trade.

The OIE Terrestrial Code is amended in May each year. The current edition is published on the OIE website at:

www.oie.int/international-standard-setting/terrestrial-code/access-online

Chapters of particular relevance are:

Chapter 1.1.2 Notification and epidemiological information

Chapter 1.3.5 Zoning and compartmentalisation

OIE Terrestrial Manual

The purpose of the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals is to contribute to the international harmonisation of methods for the surveillance and control of the most important animal diseases. Standards are described for laboratory diagnostic tests and the production and control of biological products (principally vaccines) for veterinary use across the globe.

The OIE Terrestrial Manual is updated approximately every four years. The current edition is available on the OIE website at:

www.oie.int/international-standard-setting/terrestrial-manual/access-online
<table>
<thead>
<tr>
<th><strong>Glossary</strong></th>
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<tbody>
<tr>
<td><strong>Animal byproducts</strong></td>
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<td><strong>Animal Health Committee</strong></td>
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<td><strong>Animal products</strong></td>
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<td><strong>At-risk premises (ARP)</strong></td>
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<tr>
<td><strong>Australian Chief Veterinary Officer</strong></td>
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<tr>
<td><strong>AUSVETPLAN</strong></td>
</tr>
<tr>
<td><strong>Chief veterinary officer (CVO)</strong></td>
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<tr>
<td>Term</td>
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<td>-------------------------------------------</td>
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<tr>
<td>Compensation</td>
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<tr>
<td>Consultative Committee on Emergency Animal Diseases (CCEAD)</td>
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<tr>
<td>Control area</td>
</tr>
<tr>
<td>Dangerous contact animal</td>
</tr>
<tr>
<td>Dangerous contact premises (DCP)</td>
</tr>
<tr>
<td>Declared area</td>
</tr>
<tr>
<td>Decontamination</td>
</tr>
<tr>
<td>Depopulation</td>
</tr>
<tr>
<td>Destroy (animals)</td>
</tr>
<tr>
<td>Disease agent</td>
</tr>
<tr>
<td>Disease Watch Hotline</td>
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<tr>
<td>Disinfectant</td>
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<tr>
<td>Term</td>
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<tr>
<td>-------------------------------------------</td>
</tr>
<tr>
<td>Disinfection</td>
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<tr>
<td>Disposal</td>
</tr>
<tr>
<td>Emergency animal disease</td>
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<tr>
<td>Emergency Animal Disease Response Agreement</td>
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<tr>
<td>Endemic animal disease</td>
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<tr>
<td>Enterprise</td>
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<td>Epidemiological investigation</td>
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<td>Exotic animal disease</td>
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<tr>
<td>Exotic fauna/feral animals</td>
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<td>Fomites</td>
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<tr>
<td>In-contact animals</td>
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<tr>
<td>Incubation period</td>
</tr>
</tbody>
</table>
Index case
The first or original case of the disease to be diagnosed in a disease outbreak on the index property.

Index property
The property on which the first or original case (index case) in a disease outbreak is found to have occurred.

Infected premises (IP)
A defined area (which may be all or part of a property) in which an emergency disease meeting the case definition exists or is believed to exist, or in which the causative agent of that emergency disease exists or is believed to exist.
See Appendix 1 for further details

Local control centre
An emergency operations centre responsible for the command and control of field operations in a defined area.

Monitoring
Routine collection of data for assessing the health status of a population.
See also Surveillance

Movement control
Restrictions placed on the movement of animals, people and other things to prevent the spread of disease.

National management group (NMG)
A group established to approve (or not approve) the invoking of cost sharing under the Emergency Animal Disease Response Agreement. NMG members are the Secretary of the Australian Government Department of Agriculture, Fisheries and Forestry as chair, the chief executive officers of the state and territory government parties, and the president (or analogous officer) of each of the relevant industry parties.

Native wildlife
See Wild animals

OIE Terrestrial Code
OIE Terrestrial Animal Health Code. Reviewed annually at the OIE meeting in May and published on the internet at: www.oie.int/international-standard-setting/terrestrial-code/access-online
See Appendix 3 for further details

OIE Terrestrial Manual
See Appendix 3 for further details

Operational procedures
Detailed instructions for carrying out specific disease control activities, such as disposal, destruction, decontamination and valuation.

Outside area (OA)
The rest of Australia outside the declared (control and restricted) areas.
Owner

Person responsible for a premises (includes an agent of the owner, such as a manager or other controlling officer).

Premises

A tract of land, including its buildings, or a separate farm or facility that is maintained by a single set of services and personnel.

Premises of relevance (POR)

A premises in a control area that contains a susceptible animal(s) but is not considered at the time of designation to be an infected premises, dangerous contact premises, suspect premises or trace premises. The animal(s) on such a premises are subject to procedures such as heightened surveillance and movement restrictions that are applicable in the control area.

See Appendix 1 for further details

Prevalence

The proportion (or percentage) of animals in a particular population affected by a particular disease (or infection or positive antibody titre) at a given point in time.

Quarantine

Legal restrictions imposed on a place or a tract of land by the serving of a notice limiting access or egress of specified animals, persons or things.

Resolved premises (RP)

An infected premises, dangerous contact premises or dangerous contact processing facility that has completed the required control measures and is subject to the procedures and restrictions appropriate to the area in which it is located.

See Appendix 1 for further details

Restricted area (RA)

A relatively small declared area (compared with a control area) around an infected premises that is subject to intense surveillance and movement controls.

See Appendix 1 for further details

Risk enterprise

A defined livestock or related enterprise, which is potentially a major source of infection for many other premises. Includes intensive piggeries, feedlots, abattoirs, knackeries, saleyards, calf scales, milk factories, tanneries, skin sheds, game meat establishments, cold stores, AI centres, veterinary laboratories and hospitals, road and rail freight depots, showgrounds, field days, weighbridges, garbage depots.

Sensitivity

The proportion of affected individuals in the tested population that are correctly identified as positive by a diagnostic test (true positive rate).

See also Specificity

Sentinel animal

Animal of known health status that is monitored to detect the presence of a specific disease agent.

Serotype

A subgroup of microorganisms identified by the antigens carried (as determined by a serology test).
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specificity</td>
<td>The proportion of nonaffected individuals in the tested population that are correctly identified as negative by a diagnostic test (true negative rate).  See also Sensitivity</td>
</tr>
<tr>
<td>Stamping out</td>
<td>Disease eradication strategy based on the quarantine and slaughter of all susceptible animals that are infected or exposed to the disease.</td>
</tr>
<tr>
<td>Standing Council on Primary Industries (SCoPI)</td>
<td>The council of Australian national, state and territory and New Zealand ministers of agriculture that sets Australian and New Zealand agricultural policy (formerly the Primary Industries Ministerial Council). See also Animal Health Committee</td>
</tr>
<tr>
<td>State or territory control headquarters</td>
<td>The emergency operations centre that directs the disease control operations to be undertaken in that state or territory.</td>
</tr>
<tr>
<td>Surveillance</td>
<td>A systematic program of investigation designed to establish the presence, extent or absence of a disease, or of infection or contamination with the causative organism. It includes the examination of animals for clinical signs, antibodies or the causative organism.</td>
</tr>
<tr>
<td>Susceptible animals</td>
<td>Animals that can be infected with a particular disease</td>
</tr>
<tr>
<td>Suspect animal</td>
<td>An animal that may have been exposed to an emergency disease such that its quarantine and intensive surveillance, but not pre-emptive slaughter, is warranted.  or  An animal not known to have been exposed to a disease agent but showing clinical signs requiring differential diagnosis.</td>
</tr>
<tr>
<td>Suspect premises (SP)</td>
<td>Temporary classification of a premises that contains a susceptible animal(s) not known to have been exposed to the disease agent but showing clinical signs that require investigation. See Appendix 1 for further details</td>
</tr>
<tr>
<td>Trace premises (TP)</td>
<td>Temporary classification of a premises that contains susceptible animal(s) that tracing indicates may have been exposed to an infected animal(s), or contaminated animal products, wastes or things, and that requires investigation. See Appendix 1 for further details</td>
</tr>
<tr>
<td>Tracing</td>
<td>The process of locating animals, persons or other items that may be implicated in the spread of disease, so that appropriate action can be taken.</td>
</tr>
<tr>
<td>Unknown status premises (UP)</td>
<td>A premises that has been identified as having an unknown animal status. See Appendix 1 for further details</td>
</tr>
</tbody>
</table>
Vaccination

Inoculation of individuals with a vaccine to provide active immunity.

Vaccine

A substance used to stimulate immunity against one or several disease-causing agents to provide protection or to reduce the effects of the disease. A vaccine is prepared from the causative agent of a disease, its products, or a synthetic substitute, which is treated to act as an antigen without inducing the disease.

- **attenuated**
  A vaccine prepared from infective or ‘live’ microbes that have lost their virulence but have retained their ability to induce protective immunity.

- **inactivated**
  A vaccine prepared from a virus that has been inactivated (‘killed’) by chemical or physical treatment.

Vector

A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. A **biological** vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A **mechanical** vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.

Veterinary investigation

An investigation of the diagnosis, pathology and epidemiology of the disease. See also Epidemiological investigation

Wild animals

- **native wildlife**
  Animals that are indigenous to Australia and may be susceptible to emergency animal diseases (eg bats, dingoes, marsupials).

- **feral animals**
  Domestic animals that have become wild (eg cats, horses, pigs).

- **exotic fauna**
  Nondomestic animal species that are not indigenous to Australia (eg foxes).

Zero susceptible stock premises (ZP)

A premises that contains no susceptible animals.

Zoning

The process of defining disease-free and infected areas in accord with OIE guidelines, based on geopolitical boundaries and surveillance, in order to facilitate trade.

Zoonosis

A disease of animals that can be transmitted to humans.
## Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>AAHL</td>
<td>CSIRO Australian Animal Health Laboratory (Geelong, Victoria)</td>
</tr>
<tr>
<td>AUSVETPLAN</td>
<td>Australian Veterinary Emergency Plan</td>
</tr>
<tr>
<td>BD</td>
<td>Borna disease</td>
</tr>
<tr>
<td>CA</td>
<td>control area</td>
</tr>
<tr>
<td>CBPP</td>
<td>contagious bovine pleuropneumonia</td>
</tr>
<tr>
<td>CCEAD</td>
<td>Consultative Committee on Emergency Animal Diseases</td>
</tr>
<tr>
<td>CSIRO</td>
<td>Commonwealth Scientific and Industrial Research Organisation</td>
</tr>
<tr>
<td>CVO</td>
<td>chief veterinary officer</td>
</tr>
<tr>
<td>DAFF</td>
<td>Australian Government Department of Agriculture, Fisheries and Forestry</td>
</tr>
<tr>
<td>DCP</td>
<td>dangerous contact premises</td>
</tr>
<tr>
<td>EAD</td>
<td>emergency animal disease</td>
</tr>
<tr>
<td>ECF</td>
<td>east coast fever</td>
</tr>
<tr>
<td>EEE</td>
<td>eastern equine encephalomyelitis</td>
</tr>
<tr>
<td>FAO</td>
<td>Food and Agriculture Organization of the United Nations</td>
</tr>
<tr>
<td>IP</td>
<td>infected premises</td>
</tr>
<tr>
<td>NSD</td>
<td>Nairobi sheep disease</td>
</tr>
<tr>
<td>OIE</td>
<td>World Organisation for Animal Health</td>
</tr>
<tr>
<td>RA</td>
<td>restricted area</td>
</tr>
<tr>
<td>SCAHLS</td>
<td>Subcommittee on Animal Health Laboratory Standards</td>
</tr>
<tr>
<td>SP</td>
<td>suspect premises</td>
</tr>
<tr>
<td>VEE</td>
<td>Venezuelan equine encephalomyelitis</td>
</tr>
<tr>
<td>WEE</td>
<td>western equine encephalomyelitis</td>
</tr>
</tbody>
</table>
References


CDC (Centers for Disease Control and Prevention) (2009). 2009 H1N1 flu (swine flu). CDC. www.cdc.gov/h1n1flu


