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

Agriculture and Resource Management Council of Australia and New Zealand
This Disease Strategy forms part of:
AUSVETPLAN Edition 2.0, 1996
[AUSVETPLAN Edition 1.0, was published in 1991]

This strategy will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to the AUSVETPLAN Coordinator (see Preface).

Record of amendments to this manual:
There are occasional minor differences in the page breaks between the paper version and this electronic version which we can unfortunately not avoid.

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PREFACE

This **Disease Strategy** for the control and eradication of **Aujeszky's disease**, is an integral part of the **Australian Veterinary Emergency Plan**, or AUSVETPLAN (Edition 2.0). AUSVETPLAN structures and functions are described in the **Summary Document**.

This strategy sets out the disease control principles that were approved by the Australian Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ) out-of-session in January 1996, for use in a veterinary emergency caused by the introduction of Aujeszky's disease to Australia.

Aujeszky's disease is designated as a List B disease by the Office International des Epizooties (OIE). List B diseases are, ‘Communicable diseases which are considered to be of socioeconomic and/or public health importance within countries and which are significant in the international trade of animals and animal products. The principles contained in this document for the diagnosis and management of an outbreak of Aujeszky's disease conform with the **OIE International Animal Health Code 1992** (OIE Code; see Appendix 3).

Aujeszky's disease is not included in the Commonwealth/States cost-sharing agreement for the eradication of certain exotic diseases.

Detailed instructions for the field implementation of the strategies are contained in the AUSVETPLAN **Operational Procedures Manuals** and **Management Manuals**. Cross reference to strategies, manuals and other AUSVETPLAN documents are expressed in the form:

Document Name, Section no.

For example, **Decontamination Manual, Section 3**.

In addition, **Exotic Diseases of Animals: A Field Guide for Australian Veterinarians** by W.A. Geering, A.J. Forman and M.J. Nunn, Australian Government Publishing Service, Canberra, 1995 (**Exotic Diseases Field Guide**) is a source for some of the information about the aetiology, diagnosis and epidemiology of the disease and should be read in conjunction with this strategy.

This strategy will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to:

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1 NATURE OF THE DISEASE

Aujeszky's disease (AD) is a viral disease that causes encephalomyelitis and respiratory infections in domestic and wild animals. It is of greatest economic consequence in pigs. Internationally the change in pig management systems has created an environment that facilitates the maintenance and spread of virus within pig herds.

1.1 Aetiology

Aujeszky's disease is caused by a virus belonging to the Herpesviridae. There is only one serotype, but strains vary in pathogenicity, minimum infective dose and tissue tropisms. Virulence of virus strains appears to be related to their ability to cause the formation of large cells with many nuclei in tissue culture.

1.2 Susceptible species

The pig is the only natural host for Aujeszky's disease virus. Sporadic cases occur in cattle, sheep, goats, dogs, cats, mink, foxes, deer, rabbits, mice and rats. The disease is highly fatal in these other species.

There have been no substantiated reports of human infection.

1.3 World distribution and occurrence in Australia

Aujeszky's disease occurs in most countries of Europe and the United States, Mexico, Cuba, Brazil, Venezuela, Japan, Taiwan, Korea, Malaysia, Thailand, Vietnam, The Philippines, Hong Kong, New Zealand (North Island) and Samoa. Over recent years the disease has increased in incidence and severity in many intensive pig farming regions in which it is endemic. Canada, Norway, Finland and Luxembourg are free of the disease. England eradicated it during the 1980s, Denmark has periodic incursions of AD from Germany and eradicates the disease when it occurs (Kluge et al 1992). Singapore closed all its piggeries in the mid-1980s and consequently has not had any outbreaks of Aujeszky's disease since 1989.

AD has never been diagnosed in Australia.

1.4 Diagnostic criteria

[See Glossary for terms not defined in the text]

Presumptive diagnosis may be initially based on histopathology (ie non-suppurative encephalomyelitis, see below) and confirmed by virus isolation and/or antigen detection in tissue or serum.

AD may be diagnosed first in other species that are in close contact with pigs (eg cats, rodents), when sudden deaths with or without itching (pruritus) occur.
1.4.1 Clinical signs and lesions

Pigs
In pigs Aujeszky's disease may be signalled by any acute central nervous system disease in newborn piglets that causes high mortality rates. However, clinical signs may be unremarkable. The onset of the disease may be preceded by abortions, mummified foetuses and stillbirths. In newly-infected herds, the virus usually spreads rapidly through the whole herd. Clinical signs are most likely to be seen in both newborn pigs and breeding sows. The disease pattern is strongly age-dependent and the most severe disease occurs in young animals. The clinical signs are primarily dependent on the strain and dose of virus and the age of pigs affected. The disease mainly affects respiratory and nervous tissue.

- **Newborn.** For piglets less than two-weeks old, the mortality rate may approach 100% and death occurs within hours of the onset of sickness. Prostration is often the only clinical sign. Slightly older piglets show fever and variable signs of loss of appetite, vomiting, depression with central nervous system (CNS) and respiratory involvement. The CNS signs consist of incoordination, abnormal 'goose-stepping' gait, drowsiness, muscular twitching, convulsions, involuntary eye movements and paralysis. Itching is rare in pigs. The mortality rate ranges from 20 to 100%. Deaths occur up to one week after the onset of signs but may be seen as early as 24 hours after the onset of clinical disease.

- **Weaner pigs.** The mortality rate for weaner pigs is generally of the order of 5–10%. The clinical signs already described are present, but respiratory signs may be more prominent. These include coughing, sneezing, laboured breathing and conjunctivitis.

- **Grower and finisher pigs.** Respiratory disease is the most common clinical sign and may be mild but spreads rapidly. Morbidity rates may be very high and approach 100% but if the infection is uncomplicated mortality rates are low (1–2%).

- **Adults.** Infection of adult pigs is often mild or inapparent, however, severe outbreaks have been reported. The virus can cross the placenta. If sows are infected earlier than the 13th day of pregnancy there is likely to be embryonic resorption. At later stages of pregnancy there may be abortion, mummified foetuses, stillbirths or the birth of weak, trembling pigs.

The first sign of infection in a herd may be reproductive failure followed shortly after by neonatal disease. Retrospective analysis of herd records to detect changes in farrowing rates, or numbers weaned per litter and numbers sold per sow per year, may give an indication of the time of infection. In New Zealand (MacDiarmid 1992), Singapore and the United Kingdom, the clinical signs were, for many years, unremarkable. However, a retrospective serological survey in New Zealand after the first AD diagnosis indicated that the virus had been present for at least three years without causing recognisable clinical disease (Oliver 1989).

Cattle and sheep
The disease is almost invariably fatal in these species. The most striking clinical feature is intense itching of a localised area or areas of skin, innervated by one or more spinal nerves. This leads to licking, rubbing or gnawing so severe as to lead to self-mutilation. After a day or so the animal is prostrate but is still capable of rising and walking unsteadily. The animal becomes progressively weaker over the next 12–24 hours and
develops rhythmic convulsions, bellowing, grinding of the teeth, pharyngeal paralysis, rapid shallow breathing and cardiac irregularities. Consciousness is maintained until near death, which usually occurs about two days after the onset of signs.

**Dogs and cats**
The clinical signs are similar to those in ruminants. There is intense itching and self-mutilation. The animal may emit plaintive whimperings and howls. Paralysis of the pharynx and profuse salivation may simulate rabies, hence the alternative name, pseudorabies. There may be rhythmic convulsions. Death occurs within 24–48 hours in dogs and often more rapidly in cats. In Singapore, diagnosis of AD was often preceded by deaths of cats in piggeries.

**Rodents**
Rats and mice are also dead-end hosts because the disease is fatal in these species. On farms where the disease is present increased numbers of dead rodents are frequently evident.

1.4.2 **Pathology**

**Gross lesions**
At postmortem in pigs, gross lesions are often minimal or absent. There may be purulent inflammation of the nasal lining, pharyngitis, tonsillitis and areas of fluid retention, congestion or consolidation in the lungs. The meninges may be congested. Lymph nodes may be mildly congested and contain some tiny, flat red or purple haemorrhages. Occasionally there are small white-to-yellow necrotic foci in the liver and spleen of affected animals or aborted foetuses.

In all species other than pigs, the predominant and sometimes the only nervous system lesions are found in the spinal cord. Lesions consist of oedema, congestion and haemorrhage. These lesions are most severe in the section of the dorsal horn and dorsal root ganglia that innervate the area of skin affected by itching. There is nerve cell degeneration and moderate cellular infiltration. Central nervous system lesions are similar to those in pigs, but are much milder.

**Microscopic lesions (histopathology)**
There is a diffuse, non-suppurative (not involving pus) inflammation of the brain, spinal cord and spinal nerves. Brain lesions are most common in the cerebral and cerebellar cortices, but they also occur in the pons, thalamus and medulla. Grey and white matter are both affected. There is marked perivascular cuffing (white cell accumulation), glial cell proliferation, and varying degrees of nerve cell necrosis. Cowdry type A intranuclear inclusion bodies occur in glial cells, but are by no means plentiful. There are areas of meningitis, particularly adjacent to lesions. In the lungs there may be oedema and interstitial pneumonia. Necrotic foci may be present in the tonsils, liver, kidneys, spleen and associated lymph nodes.

1.4.3 **Laboratory tests**
Animal specimens should initially be sent to the State or Territory diagnostic laboratory from where they will be forwarded to the Australian Animal Health Laboratory (AAHL), Geelong for exotic disease testing after obtaining the necessary clearance from the chief veterinary officer (CVO) of the State or Territory of the disease outbreak and informing the CVO of Victoria (for transport of the specimens to Geelong).
Specimens required

**Fresh specimens.** From all species collect one half of the brain aseptically after longitudinal section. From all species *except* pigs, the skin and subcutaneous tissue at the site of itching should be collected. From pigs, samples of lung, spleen, pharyngeal mucosa and tonsil should also be collected aseptically. From live pigs, nasal swabs should be collected and submitted in virus transport medium.

**Preserved specimens.** The other half of the brain together with cervical, thoracic and lumbar segments of the spinal cord should be fixed in neutral-buffered formalin. Specimens of tonsil, mesenteric lymph nodes, spleen, liver and kidney should also be collected in neutral-buffered formalin. If circumstances permit, a whole pig should be submitted to the laboratory.

Blood samples for serum (about 10 mL) should be collected from convalescent and recovered animals. Serum antibodies are detected 7–10 days post infection.

Transport of specimens

Unpreserved tissue specimens should be chilled and forwarded, through the relevant State laboratory, on ice or frozen gel packs to AAHL. However, if transit is likely to take more than 24 hours, glycerol buffer (pH 7.4) should be added to the specimens. Alternatively, the specimens may be frozen and forwarded on dry ice. For further information see the *Laboratory Preparedness Manual, Section 6 and Appendix 3*.

Laboratory diagnosis

**AAHL tests.** Table 1 shows the tests available at AAHL for the diagnosis of Aujeszky's disease. Diagnosis is based on the direct detection of antigen in tissues, virus isolation in tissue culture and/or the measurement of antibody in serum. Immunoperoxidase tests on formalin-fixed tissue are also available and very effective in detecting AD virus and they are particularly useful as a retrospective diagnostic test.

Serum neutralisation and latex agglutination are used for the detection of antibody in serum, the latter being the test of choice because of the speed with which a result is obtained.

**Table 1** Diagnostic tests currently available at AAHL for Aujeszky's disease

<table>
<thead>
<tr>
<th>Test</th>
<th>Specimen required</th>
<th>Test detects</th>
<th>Time taken to obtain result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Virus isolation in tissue culture and identification</td>
<td>fresh tissue samples</td>
<td>virus</td>
<td>3–5 days</td>
</tr>
<tr>
<td>Latex agglutination</td>
<td>serum</td>
<td>viral antibody</td>
<td>1 day</td>
</tr>
<tr>
<td>Serum neutralisation</td>
<td>serum</td>
<td>viral antibody</td>
<td>2–3 days</td>
</tr>
<tr>
<td>Immunoperoxidase staining</td>
<td>formalin-fixed tissue samples</td>
<td>viral antigen</td>
<td>3 days</td>
</tr>
<tr>
<td>Histopathology</td>
<td>formalin-fixed tissue samples</td>
<td>microscopic changes and presence of virus</td>
<td>2–3 days</td>
</tr>
<tr>
<td>Pathogenicity testing</td>
<td>virus isolate</td>
<td>virus</td>
<td>1 week</td>
</tr>
</tbody>
</table>

Source: Information provided by AAHL, 1995 [refer to AAHL for most up-to-date information].

**Other tests.** Viral antigen can be detected in tissues from affected pigs. Tonsil, pharyngeal mucosa, brain stem and cerebrum are preferred. Direct immunofluorescence tests are
usually applied, and these may be more sensitive than virus isolation for tissues collected some time previously. DNA/RNA hybridisation has also been used to demonstrate viral nucleic acid in the tissues of latently-infected pigs. An ELISA test has been developed and is considered to be highly sensitive and useful in the initial screening of sera (Morrison 1992), although it is not as specific as the serum neutralisation or latex agglutination tests (see above).

Virus can also be isolated from the trigeminal ganglia of latently-infected pigs by tissue culture co-cultivation techniques or by detection of viral genome by polymerase chain reaction.

1.4.4 Differential diagnosis

The following diseases must be considered in the differential diagnosis:

*Pigs — exotic diseases:*
- classical and African swine fever
- porcine reproductive and respiratory syndrome
- enterovirus encephalomyelitis (Teschen disease)

*Pigs — endemic diseases:*
- streptococcal meningoencephalitis
- hypoglycaemia
- haemagglutinating encephalomyelitis virus (HEV)
- encephalomyocarditis (EMC)
- organic arsenic and mercury poisoning
- salt poisoning
- other respiratory diseases (actinobacillosis, enzootic pneumonia and pasteurellosis)
- porcine enterovirus
- other diseases causing abortion

*Other species:*
- rabies
- scrapie (sheep and goats)
- bovine spongiform encephalopathy (BSE)
- any other conditions causing signs of persistent itching

1.5 Resistance and immunity

1.5.1 Innate and passive immunity

Immunity is age related. Piglets sucking immune sows are protected by colostral immunity for about 6–8 weeks depending on the level of sow immunity. Maternally-derived antibody can persist for about 4 months.

1.5.2 Active immunity

Pigs which have recovered from infection are immune to further exposure. During periods of stress recovered asymptomatic pigs may excrete virus.
1.5.3 Vaccination

Attenuated virus, inactivated and gene-deleted vaccines have been developed. Attenuated ('live') vaccines are considered to be better than inactivated or killed vaccines (Vannier et al 1991). Vaccines effectively protect pigs against clinical disease, significantly reduce the quantity and duration of viral shedding, but do not prevent latent infections. Transmission of AD virus may occur from latently-affected, vaccinated pigs within a herd. A disadvantage with inactivated or attenuated vaccines is that the serological response following their use is indistinguishable from natural infection.

Gene-deleted vaccines have been genetically engineered to remove some non-essential glycoprotein genes and the absence of these surface proteins (e.g., gG, gE, gC) makes them useful as negative immunological markers (see Section 1.4.3). There remains a concern that the recombination of gene-deleted vaccines might lead to a virulent virus if an animal is vaccinated with two different sorts of vaccines. However, the extensive field use of gene-deleted vaccines in the face of AD outbreaks has not resulted in recombination.

1.6 Epidemiology

Aujeszky's disease is contagious in pigs and is principally spread by the respiratory route. Spread from farm to farm can be expected to be slow, although within-farm spread will be relatively rapid.

1.6.1 Incubation period

The incubation period can be as short as 2–4 days in sucking pigs and 3–6 days in finishers. Excretion of virus begins 2–5 days after infection and can continue for at least two weeks. It may precede the onset of clinical signs (Pensaert and Kluge 1989). The OIE Code (see Appendix 3) does not give a maximum incubation period for AD.

1.6.2 Persistence of virus

General properties/environment

AD virus is relatively thermostable compared to other herpes viruses. It has a half life of 7 hours at 37°C and the following survival characteristics (Pensaert and Kluge 1989):

- rapidly inactivated at 37°C, in sunlight and in dry conditions;
- infectivity remains fairly stable between pH 5–9, but extreme acidity and alkalinity have a rapid inactivating effect;
- survives for extended periods under winter conditions below 4°C; and
  - in contaminated straw and feeding troughs for 10–30 days at 24°C or for up to 46 days at –20°C; and on other fomites for 2–7 days (Schoenbaum et al 1991),
  - in effluent for up to 3 days,
- in drinking water (unchlorinated) for 7 hours (Beran 1982).

AD virus is a large lipid-containing virus and is generally sensitive to disinfectants including detergents (see Section 2.2.8).

Live animals

In general, pigs excrete virus oronasally during the 2–4 weeks following the primary infection. Longer persistence with continuous excretion has been reported (6 months in one United States study) but is probably rare (Pensaert and Kluge 1989). However, a very high percentage of pigs become latent carriers for up to one year or longer, with
intermittent virus excretion at times when the animal is stressed, such as at parturition. There is circumstantial evidence that some latently-infected pigs may be undetected by conventional serological tests (Morrison 1992).

**Animal products**
AD virus can survive in pigmeat, the time depending on the temperature at which the meat is held (Pensaert and Kluge 1989).

### 1.6.3 Modes of transmission

**Live animals**
The most important method of infection is via oral and nasal secretions. AD virus is spread principally by nose-to-nose contact. Other methods of spread are:

- via semen or vaginal secretions (see below);
- by transplacental infection; or
- via the colostrum or in milk.

Most outbreaks originate from the introduction of infected pigs to susceptible herds. Humans are not carriers of AD and have not been implicated in the spread of the disease other than by the use of infected equipment, such as hypodermic needles and syringes.

Most animals other than pigs die after an illness of short duration, usually 2–3 days after the appearance of clinical signs.

Rats and wildlife may have some role as reservoirs but this requires further study. In south-eastern United States 19% of feral pigs are seropositive (van der Leek and Gibbs 1992).

**Artificial breeding**
Acutely infected boars can transmit virus through semen and carriers must also be expected to excrete the virus intermittently. Acutely and chronically infected sows can be expected to excrete the virus into the reproductive tract. Although AD virus has been reported to be capable of infecting embryos (Bolin 1982), embryo transfer has been successfully used to derive AD virus-negative embryos from infected sows (James et al 1983). Washing will not remove all of the virus attached to the embryo but trypsin treatment will remove residual attached virus and it is highly unlikely that the virus can be transmitted under natural circumstances by embryo transfer. (See the Artificial Breeding Centres Enterprise Manual.)

**Animal products and by-products**
The virus can survive in pigmeat, and dogs fed meat from viraemic pigs die. Contact with infected carcases, while not a common cause of virus transmission to infected pig herds, has been reported, although experimental studies have not confirmed this observation. The dose of virus necessary to infect pigs orally is much greater than for respiratory route infections (Wittmann and Rziha 1989). The virus has been spread from animals (eg cats and rodents) that have died from the disease and inadvertently contaminated grain bins (Kluge et al 1992).

**Fomites**
AD virus can be spread by contaminated drinking water. Likewise vehicular spread has not been documented. It has been suggested that birds can carry AD virus on their feet, but AD virus rapidly loses infectivity after being placed on the feet of birds. Although AD
virus has been found on flies experimentally infected with AD virus, there is insufficient
evidence to indicate that flies can transmit AD virus.

Spread on veterinary instruments within and between herds has been reported (Kluge et al 1992).

**Windborne spread**

Under certain favourable conditions windborne spread of the virus from farm to farm can
occur over distances of more than 2 km in densely populated pig-farming areas (Gloster et al 1984). The disease spread from northern Germany to Denmark in the air over a
distance of 15–40 km and, in one case, 80 km (Christensen et al 1990). This is why
Denmark maintains a vaccine buffer zone in northern Germany to minimise incursions of
the disease. Windborne spread over substantial distances (up to 80 km) can be modelled
and the distribution predicted (Christensen et al 1990).

The specific prerequisites for windborne spread are:

- large amounts of virus (ie large herds infected);
- the correct strain of virus;
- appropriate environmental conditions (ie low temperatures and high humidities); and
- topography suitable for windborne spread and the close proximity of other pig herds
  (Morrison 1992).

It is highly unlikely that these favourable conditions would be met in Australia.

**Vectors**

There are no insect vectors. There are no reliable reports that the virus survives in birds,
or on their feet, in biting insects or on flies beyond 24 hours or that is mechanically
transmitted by them.

**1.6.4 Factors influencing transmission**

The emergence of AD as a significant disease in many countries coincided with
intensification of pig farming practices. The increased density of both animals and farms
probably influenced the spread of the disease over substantial distances. A high
percentage of pigs become latent carriers for up to a year or longer (see Section 1.6.2).
Movement or import of live animals can therefore be an important means of introduction
of the disease.

**1.7 Manner and risk of introduction**

The disease would most likely enter Australia with illegally-imported pigs or semen. A
live animal introduction seems the most likely source. The risk of introduction of AD into
Australia is considered to be low (Geering 1990).
2 PRINCIPLES OF CONTROL AND ERADICATION

2.1 Introduction

Experience in other countries indicates that AD may go undetected for considerable periods after its introduction. In infected herds, AD virus will spread rapidly to all pigs in the herd, but is likely to spread slowly from herd to herd, as direct contact between pigs is the principal method of transmission. The circumstances in which AD is most likely to occur in Australia (ie low density of herds, high temperatures) are unlikely to be conducive to windborne spread of AD virus (see Section 1.6.3). Transporting pigs from an AD virus-infected herd to slaughter does not pose a threat of AD virus spread. Meat from abattoir-slaughtered pigs is very unlikely to present a risk of spread of infection to pigs or other susceptible animals, as infected pigs are viraemic for a short time, the amount of virus is reduced by the pH changes postmortem, and freezing inactivates AD virus. Meat inspection procedures should ensure that clinically-infected pigs, detected at ante or postmortem inspection are condemned and rendered. In addition, AD virus-infected pigmeat would have to be fed directly to pigs to infect them. A ban on swill-feeding is a precaution against this transmission.

2.2 Methods to prevent spread and eliminate pathogens

AD has been successfully eliminated from infected farms and countries by either:

- immediate depopulation (with salvage through an abattoir) where acute cases of disease are present, and/or when more than 25% of the breeding herd reacts serologically;
- progressive depopulation over a 7-month period to minimise the slaughter of pigs at normally unsaleable weights; or
- removal of serological reactors when these comprise less than 20–25% of the breeding herd.

These options are discussed further in Section 2.2.5, below.

In Denmark infected herds with fewer than 25% reactors are tested every 28 days and seropositive sows removed after each test until there were two clear tests followed by a test 6 months later. England eradicated AD by slaughtering either infected herds, or only seropositive pigs in herds with low prevalence and no evidence of infection spread.

Improved standards of management and hygiene can also help to prevent spread.

2.2.1 Quarantine and movement controls

The imposition of quarantine that allows the movement of pigs direct to slaughter, but prohibits the movement of pigs to other properties and to saleyards is generally a highly effective control mechanism in stopping the spread of disease. Quarantining infected premises (IPs), dangerous contact premises (DCPs) and suspect premises (SPs) allows time for a thorough assessment of the herd disease prevalence to be made (see Appendix 1). This is similar to establishing a restricted area of up to a 10 km radius, around the
infected premises, to implement an intensive surveillance program. Prolonged movement restrictions should be avoided.

**Zoning**
Due to the nature of AD spread (direct animal transmission and slow herd-to-herd spread), and the extensive movement of breeding and slaughter pigs, declaration of control areas or States is unlikely to be applied in Australia. Zoning may affect the normal trade of pigs within Australia, but is unlikely to have international trade implications.

**2.2.2 Tracing**
Recognition and confirmation of infection in herds within a minimum three kilometre radius of an infected herd, or dangerous contact/suspect herds identified by trace-back and trace-forward from an infected herd, can be achieved by clinical assessment, examination of herd reproductive records and serological testing.

The period covered by trace-forward and trace-back will depend on the type of herds involved and a period of several months should be considered.

**2.2.3 Surveillance**
Herd prevalence (local/regional/state/national) can be determined by serology. Priority for serological testing (serosurveillance) is given to those animals that originated from an infected premises (IP) and animals in contact with those pigs. In addition, a sample of pigs from groups with the highest risk of infection will be tested (see Appendix 4).

**Identification of Aujeszky's disease-free herds**
In countries with endemic AD, spread has been reduced by identifying AD-free herds as a source of replacement breeding stock.

Certification is based on serological monitoring — two clean tests 30 days apart, annual retests, absence of clinical signs and appropriate security (including testing or certification of introductions). Disease-free artificial insemination centres should also be accredited.

**2.2.4 Treatment of infected animals**
The treatment of infected animals is inappropriate and ineffective.

**2.2.5 Destruction of animals**

**Depopulation — single event**
Depopulation by slaughtering all pigs from an IP at an abattoir over a short period is an effective but expensive control measure (Zimmerman et al 1989).

**Progressive depopulation**
Progressive depopulation over a period of months is an equally effective, much cheaper, but slower eradication method. All sows are culled after weaning litters and initially finishers are removed as they reach the normal slaughter weight for the property. In order to minimise the time the piggeries are underutilised at the end of the depopulation program, younger pigs will be slaughtered as soon as possible.

**Culling seropositive breeders**
Culling of seropositive breeders can be an effective method of control providing the herd prevalence is sufficiently low, however eradication may not be achieved (Thawley and Morrison 1988, Morrison 1992). The major factor determining the likelihood of success is
the degree of segregation that can be achieved between replaced and non-replaced animals. Nose-to-nose contact and coitus between the two classes of stock must not be permitted. Replacements from the grower herd should be serotested prior to introduction into the breeding herd. The whole breeding herd should be serotested periodically.

**Euthanasia on the property**

Pigs and other susceptible species in the piggery showing CNS signs should be killed on the property for humane reasons and disposed of by burial. Pathological samples may be collected and dispatched.

### 2.2.6 Treatment of animals products and by-products

Seropositive animals may be forwarded to approved abattoirs for salvage. Normal meat pH changes and the storage of the product at –20°C will inactivate the virus. Any clinically-infected animals detected at antemortem or postmortem inspection must be condemned and subjected to rendering at approved temperatures.

All waste material must be decontaminated prior to disposal.

### 2.2.7 Disposal

Animals slaughtered as part of a depopulation program or culled seropositive breeders as described in Section 2.2.5 above will be transported to an abattoir for slaughter.

Animals slaughtered on the property should be disposed of by burial or rendering.

### 2.2.8 Decontamination

Outside areas on farms will clean themselves within 48 hours because sunlight kills AD virus. Effluent pits do not have to be emptied because AD virus will not persist beyond 3 days. However, depopulation followed by a decontamination program provides an opportunity to eradicate other pig pathogens in addition to AD virus. Although spread of AD virus by mechanical transmission is minimal, decontamination of trucks that have carried pigs from infected and dangerous contact premises, should be undertaken.

Routine cleaning with detergents (including household detergents) followed by disinfection using any of the common disinfectants will eliminate the virus (see Section 1.6.2). The recommended disinfectants are sodium hydroxide, hypochlorite or Virkon. Refer to the **Decontamination Manual, Tables 2.3, 3.3 and 4**, for specific details of cleaning and disinfection.

### 2.2.9 Vaccination

Vaccination prevents clinical signs but vaccinated pigs can still become infected. However, these pigs shed less virus, suffer limited invasion of tissues, usually limited to the lower respiratory tract and do not transmit virus across the placenta.

Passive, maternal immunity inhibits the development of immunity from vaccination, therefore young pigs should not be vaccinated until about 8–10 weeks of age. Different vaccines (attenuated virus vaccines, inactivated vaccines or gene-deleted vaccines) have been shown to be effective. Serological tests (ELISA) can differentiate naturally-infected pigs from those vaccinated with the gene-deleted vaccines (see Section 1.4.3). Vaccination, as part of a test and removal program, is appropriate in herds with high prevalence of AD, in order to reduce spread of AD virus and stabilise infection. Gene-
deleted vaccines are the vaccines of choice in a control program where vaccination is used (see Appendix 5).

At present no AD vaccine is registered for use in Australia. It has been experimentally demonstrated that when pigs are inoculated with two different gene-deleted vaccines, recombination can occur. Although this is unlikely to occur in the field, only one gene-deleted vaccine should be used in Australia. In addition the use of only one vaccine will minimise the number of ELISAs necessary for diagnostic use.

2.2.10 Wild animal control

Reduction in rodent numbers is important and must be considered as part of the decontamination program on infected farms. As an infected farm is depopulated, the rodent population is likely to move to neighbouring farms with the risk of spreading the infection.

Feral pigs present a risk to the domestic population if they are able to gather in any numbers in close proximity to a commercial farm. The best way of protecting farms is to erect a pig-proof fence around the farm. In the United States, double fencing effectively prevented the spread of the disease amongst pigs housed outside. Depopulation may be feasible in the case of a very localised outbreak in feral pigs. Vaccination by baiting with oral vaccines is being developed in the United States. For more details refer to Wild Animal Control Manual (in press).

2.2.11 Vector control

Insects have not been incriminated in spreading AD virus.

2.2.12 Sentinel and restocking measures

There is no requirement for sentinels. Total repopulation should be allowed to commence 30 days after completion of decontamination. The breeding herd should be monitored serologically 30 days after repopulation.

2.2.13 Public awareness

Public awareness programs should emphasise that:

- AD is spread by direct animal transmission and slow herd-to-herd infection;
- tools exist to achieve effective eradication;
- there have been no reports of human infection with AD virus; and
- although a wide range of species can be affected, close contact with pigs is necessary for infection to occur and spread between other species is extremely rare.

The use of the alternative name for the disease — pseudorabies — must be avoided. For further information see the Public Relations Manual.

2.3 Feasibility of control in Australia

The chance of a successful eradication program in domestic pigs (in both extensive and intensive production systems) is high. Even if the disease spread to the wild pig population it could be prevented from spreading to major industry operations by appropriate fencing. There is no evidence that this disease will establish in Australia’s native animal population.
## 3 POLICY AND RATIONALE

### 3.1 Overall policy for Aujeszky's disease

Aujeszky's disease is an OIE List B disease that has significant production effects and is important for the trade in pigs and pig products. 

The policy is to eradicate the disease as quickly as possible by using the following strategies:

- **quarantine and movement controls** on animals, animal products and things on infected premises to prevent spread of infection;
- **tracing and surveillance** to determine the source and extent of infection and to provide proof of freedom from the disease;
- **slaughter and sanitary disposal of clinically affected and seropositive animals** to free herds of infection (this may include slaughter of such animals at approved abattoirs under supervision);
- **vaccination** in combination with other measures may be an option where the disease is present at high prevalence in larger herds, or if other measures are not effective;
- **decontamination** of facilities, products and things to eliminate the virus on infected premises and to prevent spread;

This policy is based on the nature of the disease. If eradication is reasonably rapid it is likely to have a limited economic impact on the pig industry. Aujeszky's disease is unlikely to spread rapidly between farms under Australian conditions.

Aujeszky's disease is not included in the Commonwealth/States cost-sharing agreement.
The CVO(s) in the State(s)/Territory(s) in which the outbreak(s) occurs will be responsible for implementing disease control measures (in accordance with relevant legislation), and will make ongoing decisions on follow-up disease control measures in consultation with the Consultative Committee on Exotic Animal Diseases (CCEAD), the State/Territory and Commonwealth governments, and representatives of the affected industries. The detailed control measures adopted will be determined using the principles of control and eradication (Section 2) and epidemiological information about the outbreak. For further information on the responsibilities of the State/Territory disease control headquarters and local disease control centre(s), see the Control Centres Management Manual.

3.2 Strategy for control and eradication

Following the initial diagnosis of AD and the identification of an IP(s) (see Appendix 1), the priority is to implement quarantine and movement controls and to establish the size and number of infected herds and disease prevalence within herds through tracing and surveillance.

The selected strategy will depend on the results of the tracing and surveillance program, which may take some weeks. There is no urgency in selecting an appropriate control strategy as prompt implementation of quarantine and movement controls on infected premises will effectively contain the spread of disease.

The selected strategy should be directed to salvaging as many animals as possible at the least cost of control measures and may include:

- immediate depopulation with salvage through abattoirs where acute cases are present and/or more than 25% of the breeding herd is serologically affected;
- progressive depopulation to take into consideration the presence of unsaleable pigs at the time of detection of disease; and
- removal of serological reactors where these comprise less than 20–25% of the breeding herd.

3.2.1 Stamping out

Stamping out is not the most appropriate strategy for the eradication of Aujeszky’s disease in the first instance except under special circumstances and only in agreement with industry and the individual producer(s). This strategy may be considered if tracing and surveillance shows that the disease is limited and confined, and/or there is a high prevalence in the breeding herd and eradication is likely to be prolonged and costly to individual producers and a major disruption to commercial operations.

3.2.2 Quarantine and movement controls

The initial detection of the IP(s) will require immediate implementation of quarantine to contain the disease within the confines of the IP. Follow up measures of tracing and surveillance will assist in determining further action but will readily identify further IPs, DCPs and SPs, which must be placed under immediate quarantine.

Quarantine will include measures to prevent contact with wild pigs, eliminate or exclude rodents and prevent the migration of rodents to other premises.
The movement of animals to other pig-producing premises or saleyards will not be permitted but movement of animals, free from clinical disease, direct to slaughter will be allowed.

See Appendix 2 for further details.

**Zoning**

Zoning is inappropriate for this disease (see Section 2.2.1), unless the disease is widespread and areas of different disease status need to be established.

### 3.2.3 Treatment of infected animals

The treatment of infected animals is inappropriate and ineffective.

### 3.2.4 Treatment of animal products

Seropositive animals may be salvaged through approved abattoirs as meat pH changes and frozen storage of the product inactivates the virus. Clinically-infected animals will be condemned and subjected to rendering (see Sections 2.2.6 and 2.2.7).

All waste material must be decontaminated prior to disposal.

### 3.2.5 Vaccination

Vaccination prevents clinical disease but not infection. Vaccinated animals shed less virus, still suffer limited invasion of tissues and do not transmit virus across the placenta.

Passive, maternal immunity inhibits the development of immunity from vaccination up to about 8–10 weeks of age. Various vaccines have been shown to be effective but a gene-depleted vaccine is probably the most appropriate and is the vaccine of choice. The ELISA serological test can differentiate between natural and gene-depleted vaccine-infected animals. Laboratory experiments have shown that if pigs are vaccinated with two different gene-depleted vaccines recombination can occur. It is important that only one gene-depleted vaccine is approved although recombination is unlikely to occur in the field. The use of one vaccine will reduce the number of ELISAs necessary for diagnosis.

Vaccination could be considered for breeding animals in herds of high prevalence of infection, ensuring that all breeders and all growers that have lost their natural immunity (>10–12 weeks of age) are vaccinated. For further details see Appendix 5.

### 3.2.6 Tracing and surveillance

Tracing and surveillance should be carried out within a minimum three mile radius of an infected herd(s) or dangerous contact/suspect herds identified through tracing (see Table 2). This will involve clinical inspections, an examination of herd reproduction records and serological testing. Trace-back and trace-forward should involve tracing of movements over a period of several months and the period will depend on the type of herd involved.

Serological testing, to determine the herd and individual prevalences, should be prioritised to those herds that have received animals from an infected premises and to animals that have originated from an infected premises and contacts.

The level of serological testing will be determined by a number of factors:

- the number of introduced animals;
- the time since introduction;
• the degree of direct contact between introduced and other pigs in the herd;
• the extent to which the flow of pigs through the herd compares to ‘all-in-all-out’; and
• the herd size.

3.2.7 Decontamination

Fomites do not play a major role in the spread of AD. It is strongly recommended however, that the areas where the disease has been detected is decontaminated as should vehicles that have carried animals from infected farms. Sunlight will effectively inactivate the virus in open areas.

3.3 Social and economic effects

Losses to individual producers and to the industry as a whole could be substantial if the disease is allowed to proceed uncontrolled over the long term. The cost of endemic disease has justified the undertaking of eradication in a number of countries.

The strategy to act quickly when the disease is detected in Australia and the likely slow spread between herds in this country gives a high likelihood of succeeding with an eradication plan. This is likely to be the case even if the disease goes undetected for some time but losses would be greater.

Table 2 Tracing and surveillance

<table>
<thead>
<tr>
<th>Tracing</th>
<th>Herd type</th>
<th>Activity</th>
<th>Priority</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trace-back</td>
<td>Properties that exported live pigs to IPs</td>
<td>Bh, C</td>
<td>2</td>
</tr>
<tr>
<td>Trace-forward</td>
<td>DCPs /SPs in restricted area</td>
<td>Bm, C</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Herd imported breeders from IP</td>
<td>A, Bh, C</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Herd imported non-breeders from IP</td>
<td>A, Bm, C</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Herd imported pigs from gatherings of pigs that included pigs from IP</td>
<td>Bl, C</td>
<td>4</td>
</tr>
</tbody>
</table>

Activity key:

A  Serological testing of identifiable imports
B  Serological testing of random samples with:
    Bh  high intensity
    Bm  medium intensity
    Bl  low intensity
C  Clinical and production records examination

Priority key:

1  Highest priority
4  Lowest priority

The initial loss to individuals with infected herds, particularly if depopulation is undertaken, could be considerable in the short term and producers of breeding stock will lose their sales of breeding stock while premises are under quarantine. While commercial producers will still be subjected to losses, a well-planned eradication program and the judicious marketing of saleable stock will assist in alleviating this loss.
Australia does not have a substantial export market in live pigs or pigmeat but the market is being developed and an outbreak of Aujeszky’s disease could seriously set-back these initiatives, at least in the short term. Aujeszky’s disease is unlikely to be transmitted in meat if the product has been subjected to adequate meat inspection procedures and has been frozen for an approved period. The OIE Animal Health Code (Appendix 3) does not recommend a prohibition on the importation of live pigs from AD-infected countries but Australia would need to be able to provide sound evidence of free herds through adequate serological surveillance.

3.4 Criteria for proof of freedom

After an outbreak it will be desirable to prove freedom to our trading partners so as to re-establish access to export markets.

Re-establishment of freedom will require a well planned and documented serosurveillance program of piggeries within a 10 km radius of the previously-infected premises. The survey should only be implemented after at least 6 months has elapsed since the pivotal property was determined free of Aujeszky's disease.

A survey outside this area would also be necessary to substantiate the free status. For further details see Appendix 4.

3.5 Funding and compensation

As Aujeszky's disease is not included in the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases, funds to pay the costs of eradication, including compensation, will have to be found from other sources. Possible sources are:

- State government funds;
- State disease compensation funds;
- Commonwealth government funds;
- special industry levies; and
- other agreed arrangements.

Alternatively, the costs and losses might have to be borne by individual owners.

3.6 Strategy if the disease becomes established

The disease would be considered to have become established if epidemiological investigations show the disease is widespread throughout the industry.

Infected premises will still be subject to quarantine and movement controls and vaccination will play a major role together with testing and removal of infected animals. Increased industry liaison and producer education to improve management practices will play an important role. All-in-all-out marketing and decreasing pig density are two strategies that should be followed.

The matter of ongoing costs of eradication may need further review.
APPENDIX 1 Guidelines for classifying declared areas

Infected premises (IP)
A premises classified as an IP will be a defined area (which may be all or part of a property) in which clinical AD has been diagnosed or is believed to exist. An infected premises is subject to quarantine served by notice and to eradication or control procedures. This classification would be removed 6 months after the last ‘clean’ test for AD.

Dangerous contact premises (DCP)
Premises classified as DCPs will be:

- all neighbouring properties of an IP with pigs; and
- all properties to which susceptible animals have moved from an IP within 30 days of the initial signs of AD on the IP.

Suspect premises (SP)
Premises classified as SPs will be:

- all properties with pigs up to a 10 km radius of the infected animals on an IP;
- all other properties owned or managed in conjunction with an IP;
- all properties from which the IP obtained susceptible animals in the 30 days preceding the first clinical signs on the IP; and
- all properties where it is considered the disease could have spread from an IP to by movement of susceptible animals from saleyards, movement of vehicles, people, equipment or feedstuff during the 30 days prior to the first clinical signs on the IP.

SPs will be subject to quarantine and intensive surveillance.

Restricted area (RA)
If all properties that fall into the above categories are quickly placed in quarantine, the need to declare a restricted area will be averted. Alternatively an RA with at least a 10 km radius from the IP will be established.

Control area (CA)
Not appropriate (see Section 2.2.1).
APPENDIX 2  Recommended quarantine and movement controls

Infected and suspect premises

Movement out of susceptible animals:

**Pigs.** Approved under permit direct to immediate slaughter within 4 hours of arrival at an approved abattoir. Routes to be specified. See Notes 1–5 below.

**Other species.** Movement restrictions apply under certain circumstances. See Notes 6–8.

Movement in of susceptible animals:

**Pigs.** Restricted to vaccinated breeding pigs only.

**Other species.** No restrictions as long as they are segregated from the pigs on arrival.

Movement out of specified products:

Dead pigs permitted off the premises for rendering or burial only. No other restrictions.

Movement out of other animals:

Provided that the piggery can be identified as a separate farm area from the rest of the property, there are no movement restrictions on other animals on the IP or SP, provided that they have had no contact with pigs. All animals other than pigs on an IP or SP must be separated from the piggery or the pigs in a manner that prevents direct contact.

Movement in and out of people:

Movement should be restricted to essential visitors. Protective clothing including boots should be provided on the property for visitors. Before leaving visitors should wash and disinfect their hands.

Movement in and out of vehicles and equipment:

No restriction on vehicles although movements should be kept to a minimum. Veterinary instruments should be sterilised before leaving.

Movement out of crops and grains:

Grain stored on an IP or SP must be sieved to detect rodents or cats that have died from AD.

Notes:

(1) Approval for pig movements under permit only.

(2) All pigs to be consigned directly to an approved abattoir for immediate slaughter.

(3) Routes to avoid roads where there are pigs housed within 100 metres of the road.

(4) Multiple consignments per truck prohibited unless by special approval from the local disease control centre (LDCC) controller, subject to:
   - IP or SP being the last pick-up;
   - whole consignment being for immediate slaughter (within 4 hours of arrival);
   - truck being cleaned and disinfected to the satisfaction of a meat inspector at the abattoir;
   - no movements being allowed to saleyards or to other properties.

(5) The piggery can be identified as a separate farm area from the rest of the property.

(6) There are no restrictions on other animals on an IP or SP that have no contact with pigs.

(7) Animals other than pigs on an IP or SP must be separated from the piggery, or the pigs in a way which prevents direct contact.

(8) Animals other than pigs on an IP or SP must be separated from pigs for 7 days and be certified clinically normal by an inspector, before movement will be permitted.
APPENDIX 3  OIE International Animal Health Code for Aujeszky's disease

[NB  The following text is taken directly from the OIE International Health Code (1992); Chapter 3.1.2.  For definitions, Appendixes, etc see the original text.  The OIE Codes are amended every year in May.  There have been no amendments to the code for AD in 1993, 1994 or 1995.]

Preamble:  For diagnostic tests, reference should be made to the Manual (B2) [§ see OIE publications under References].

Article 3.1.2.1.

Veterinary Administrations of importing countries should require:

for breeding pigs from unvaccinated herds

the presentation of an international animal health certificate attesting that:

a) the animals:

1) come from a herd in which no clinical sign of Aujeszky's disease (AD) was officially reported during the 12 months prior to shipment;
2) were isolated in the establishment of origin for 30 days before entry into a quarantine station, were subjected to diagnostic tests for AD with negative results and were clinically healthy;
3) were kept in a quarantine station for the 30 days prior to shipment and, not less than 21 days following the test referred to in paragraph 2) above, were subjected to diagnostic tests for AD with negative results;

b) all pigs in the quarantine station satisfy all the requirements of paragraph a) above.

Article 3.1.2.2.

Veterinary Administrations of importing countries should require:

for fresh meat and meat products of pigs

the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals slaughtered in an abattoir and found to be healthy before and after slaughter.
APPENDIX 4 Procedures for surveillance and proof of freedom

Proof of freedom from AD virus is not as important for trade as it is for other diseases. Evidence of freedom, which comprises serological testing of previously-infected herds, in-contact herds, herds with stock movements from the IP, and other pig herds within a 10 km radius of the previously-infected premises, will be collected at least six months after the pivotal property was determined free of the disease. Conducting a national survey would be resource intensive, but some survey outside the area defined above would be necessary to substantiate free status.

The number of animals to be serologically tested depends on the circumstances of the particular herd. A herd that was on an IP and is undergoing test-and-removal procedures, will have all breeding stock tested periodically. A DCP will have animals that are directly related (by way of movements) to an IP tested. In addition a sample of animals sufficient to detect a 5% prevalence of AD with a 95% confidence would need to be tested. Depending on resources and the number of herds involved, this may entail testing all breeding animals in the DCP. Further proof of freedom would include sampling a proportion of cull sows and boars from the restricted area or State.

OIE Animal Health Codes do not recommend that importation of breeding pigs be restricted to countries free from AD. Certification of freedom of clinical disease in the herd of origin in the preceding 12 months, and serological testing and isolation of the animals are considered satisfactory. The OIE Code recommends importation of meat and meat products from animals slaughtered in an abattoir and found to pass health inspections before and after slaughter.
APPENDIX 5 Procedures for vaccination

Vaccination is an integral part of the control and eradication strategy. The recommended vaccine is a gene-deleted vaccine that meets OIE specification (OIE Manual of Standards for Diagnostic Tests and Vaccines, 1992; B2). Gene-deleted vaccines used at a national level must be restricted to one type, eg gE (glycoprotein I)-deleted vaccine, for which an ELISA is carried out by AAHL.

The vaccination program is based on immunisation of breeding pigs according to the registered directions for use.

**AD vaccines available**

<table>
<thead>
<tr>
<th>Manufacturer</th>
<th>Vaccine</th>
<th>Gene deletion</th>
<th>Diagnostic test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooper</td>
<td>Aużyvac²</td>
<td>gE</td>
<td></td>
</tr>
<tr>
<td>Rhone-Merieux</td>
<td>Gesky-pur²</td>
<td>viral subunit</td>
<td></td>
</tr>
<tr>
<td>IDEXX</td>
<td>Herdfend</td>
<td>gG</td>
<td></td>
</tr>
<tr>
<td>Solvay</td>
<td>Ingelvac KV³ &amp; MVL¹</td>
<td>gE</td>
<td>Herdfend</td>
</tr>
<tr>
<td>Boehringer Ingelheim</td>
<td>Nobi-vac² &amp; Nobi-porvac¹</td>
<td>gE</td>
<td>Ingelvac</td>
</tr>
<tr>
<td>Intervet</td>
<td>Omnivac-PRV¹</td>
<td>TK</td>
<td></td>
</tr>
<tr>
<td>Norden</td>
<td>PR-Vac²</td>
<td>gE</td>
<td>ClinEase-PRV</td>
</tr>
<tr>
<td>SmithKline Beecham</td>
<td>PRV-Vac³</td>
<td>gE</td>
<td></td>
</tr>
<tr>
<td>Syntrovet</td>
<td>PRV/Marker¹ &amp; PRV/Marker gold¹</td>
<td>TK &amp; gG</td>
<td></td>
</tr>
<tr>
<td>Pitman-Moore</td>
<td>Pseudovax³</td>
<td>?</td>
<td></td>
</tr>
<tr>
<td>Solvay-Duphar</td>
<td>Suvaxyn¹</td>
<td>gE</td>
<td></td>
</tr>
<tr>
<td>UpJohn</td>
<td>Tolvid¹</td>
<td>TK &amp; gG</td>
<td></td>
</tr>
</tbody>
</table>

1  Attenuated vaccine TK = Thymidine kinase
2  Inactivated vaccine ? = Not known
3  Not known
4  Commercially available test kits
# GLOSSARY

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-in-all-out production</td>
<td>A method of production in which all stock leave the premises followed by total restocking.</td>
</tr>
<tr>
<td>ANEMIS</td>
<td>Animal Health Emergency Information System. A system for the collection, assimilation, actioning and dissemination of essential disease control information using paper documentation and a computer database</td>
</tr>
<tr>
<td>Animal products</td>
<td>Meat products and products of animal origin (e.g., eggs, milk) for human consumption or for use in animal feeding.</td>
</tr>
<tr>
<td>AUSVETPLAN</td>
<td>A series of documents that describe the Australian response to exotic animal diseases, linking policy, strategies, implementation, coordination and emergency-management plans.</td>
</tr>
<tr>
<td>Consultative Committee on Exotic Animal Diseases</td>
<td>A committee of State/Territory CVOs, AAHL and CSIRO, chaired by the CVO of Australia (Commonwealth DPIE), to consult in emergencies due to the introduction of an exotic disease of livestock, or serious epizootics of Australian origin.</td>
</tr>
<tr>
<td>Control area</td>
<td>A bigger area than a restricted area (possibly as big as a State) where restrictions will reduce the chance of the disease spreading further afield. (See Appendix 1).</td>
</tr>
<tr>
<td>Dangerous contact animal</td>
<td>An animal showing no clinical signs of disease but which, by reason of its probable exposure to disease, will be subjected to disease control measures.</td>
</tr>
<tr>
<td>Dangerous contact premises</td>
<td>Premises that contain a dangerous contact animal(s) (see Appendix 1).</td>
</tr>
<tr>
<td>Decontamination</td>
<td>Includes all stages of cleaning and disinfection.</td>
</tr>
<tr>
<td>Depopulation</td>
<td>Planned removal of a host population from a particular area to control or prevent the spread of disease.</td>
</tr>
<tr>
<td>Disposal</td>
<td>Sanitary removal of animal carcases and things by burial, burning or some other process so as to prevent the spread of disease.</td>
</tr>
<tr>
<td>ELISA</td>
<td>Enzyme-linked immunosorbent assay — a serological test designed to detect and measure the presence of antibody or antigen in a sample. The test uses an enzyme reaction with a substrate to produce a colour reaction when antigen–antibody binding occurs.</td>
</tr>
<tr>
<td>Encephalomyelitis</td>
<td>Inflammation of the brain and spinal nerves.</td>
</tr>
<tr>
<td>Fomites</td>
<td>Inanimate objects (e.g., boots, clothing, equipment, vehicles, crates, packagings) that can carry the exotic agent and spread the disease through mechanical transmission.</td>
</tr>
<tr>
<td>Glial cells</td>
<td>Supporting cells of the brain and spinal cord.</td>
</tr>
<tr>
<td>Glycoproteins</td>
<td>Surface antigenic proteins (of the virus); formerly gI, gIII and gX; now reclassified by international convention as gE, gC, gG, respectively.</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
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<tr>
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</tr>
<tr>
<td>Incubation period</td>
<td>The period which elapses between the introduction of the pathogen into the animal and the occurrence of the first clinical signs of the disease.</td>
</tr>
<tr>
<td>Infected premises</td>
<td>See Appendix 1.</td>
</tr>
<tr>
<td>Latex agglutination</td>
<td>A standard serological test involving the clumping of latex particles in the presence of virus antibodies in serum.</td>
</tr>
<tr>
<td>Local disease control centre</td>
<td>An emergency operations centre responsible for the command and control of field operations in a defined area.</td>
</tr>
<tr>
<td>Movement controls</td>
<td>Restrictions placed on movement of animals, people and things to prevent dissemination of disease.</td>
</tr>
<tr>
<td>Premises</td>
<td>A defined area or structure, which may include part or all of a farm, enterprise or other private or public land, building or property.</td>
</tr>
<tr>
<td>Prevalence</td>
<td>The number of cases of a specific disease (or infection or positive antibody titre) occurring in a given population at a particular time (cf incidence, which is the number of new cases that occur over a specified period).</td>
</tr>
<tr>
<td>Quarantine</td>
<td>Legal restrictions imposed on a place, animal, vehicle or other things limiting movement of specified animals, persons or things.</td>
</tr>
<tr>
<td>Rendering (of carcases)</td>
<td>Processing by heat to inactivate infective agents. Rendered material may be used in various products according to particular disease circumstances.</td>
</tr>
<tr>
<td>Restricted area</td>
<td>A relatively small declared area (compared to a control area) around an infected premises that is subject to intense surveillance and movement controls (see Appendix 1).</td>
</tr>
<tr>
<td>Salvage (of carcases)</td>
<td>Recovery of some (but not full) market value by treatment and use of products, according to disease circumstances.</td>
</tr>
<tr>
<td>Sentinel animals</td>
<td>Animals of known health status monitored for the purpose of detecting the presence of a specific exotic disease agent.</td>
</tr>
<tr>
<td>Serotype</td>
<td>A subgroup of a genus of microorganisms identifiable by the antigens carried by the members.</td>
</tr>
<tr>
<td>Serosurveillance</td>
<td>Surveillance of an animal population by testing serum samples for the presence of antibodies to disease agents.</td>
</tr>
<tr>
<td>Serum neutralisation test</td>
<td>A type of serological test designed to detect and measure the presence of antibody in a sample. The test is based on the ability of an antibody to neutralise the biological activity of an antigen.</td>
</tr>
<tr>
<td>Stamping out</td>
<td>Eradication procedures based on quarantine and slaughter of all infected animals exposed to infection.</td>
</tr>
<tr>
<td>State/Territory disease control headquarters</td>
<td>The emergency operations centre that directs the disease control operations to be undertaken in the State/Territory.</td>
</tr>
<tr>
<td>Suppurative</td>
<td>Discharging pus.</td>
</tr>
<tr>
<td>Surveillance</td>
<td>A systematic program of inspection and examination of animals or things to determine the presence or absence of an exotic disease.</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
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</tr>
<tr>
<td>Susceptible species</td>
<td>Animals that can be infected with the disease (for AD—pigs, cattle, goat, sheep, cats, dogs, deer, mice, rabbits, rats).</td>
</tr>
<tr>
<td>Suspect animal</td>
<td>An animal that may have been exposed to an exotic disease such that its quarantine and intensive surveillance, but not pre-emptive slaughter, are 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</td>
<td>Premises containing suspect animals that will be subject to surveillance <em>(see Appendix 1).</em></td>
</tr>
<tr>
<td>Swill</td>
<td>Food scraps of placental mammal origin that have not been obtained from approved slaughter facilities or treated by an approved process.</td>
</tr>
<tr>
<td>Swill feeding</td>
<td>Swill feeding is the feeding of swill to pigs; unlicensed swill feeding is illegal in Australia.</td>
</tr>
<tr>
<td>Tracing</td>
<td>The process of locating animals, persons or things that may be implicated in the spread of disease, so that appropriate action be taken.</td>
</tr>
<tr>
<td>Vaccines</td>
<td></td>
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<tr>
<td>– inactivated</td>
<td>A vaccine prepared from a virus that has been inactivated (‘killed’) by chemical or physical treatment.</td>
</tr>
<tr>
<td>– attenuated</td>
<td>A vaccine prepared from infective or ‘live’ microbes that have lost their virulence but have retained their ability to induce protective immunity.</td>
</tr>
<tr>
<td>– gene-deleted</td>
<td>An attenuated or inactivated vaccine in which genes for non-essential surface glycoproteins have been removed by genetic engineering. This provides a useful immunological marker for the vaccine virus compared to the wild virus.</td>
</tr>
<tr>
<td>Vector</td>
<td>A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. A <em>biological</em> vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A <em>mechanical</em> vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.</td>
</tr>
<tr>
<td>Viraemia</td>
<td>The presence of viruses in the blood.</td>
</tr>
<tr>
<td>Zoning</td>
<td>The process of defining disease-free and infected zones in accord with OIE guidelines, in order to facilitate trade. A high level of movement control between zones will apply.</td>
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### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>AAHL</td>
<td>CSIRO Australian Animal Health Laboratory, Geelong</td>
</tr>
<tr>
<td>AD</td>
<td>Aujeszky’s disease</td>
</tr>
<tr>
<td>ANEMIS</td>
<td>Animal health emergency information system</td>
</tr>
<tr>
<td>ARMCANZ</td>
<td>Agriculture and Resource Management Council of Australia and New Zealand</td>
</tr>
<tr>
<td>BSE</td>
<td>Bovine spongiform encephalopathy</td>
</tr>
<tr>
<td>CCEAD</td>
<td>Consultative Committee on Exotic Animal Diseases</td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
</tr>
<tr>
<td>CVO</td>
<td>Chief veterinary officer</td>
</tr>
<tr>
<td>DNA</td>
<td>Deoxyribonucleic acid</td>
</tr>
<tr>
<td>DCP</td>
<td>Dangerous contact premises</td>
</tr>
<tr>
<td>ELISA</td>
<td>Enzyme-linked immunosorbent assay</td>
</tr>
<tr>
<td>EM</td>
<td>Electron microscopy</td>
</tr>
<tr>
<td>EMC</td>
<td>Encephalomyocarditis</td>
</tr>
<tr>
<td>gE, Gc etc</td>
<td>Glycoproteins</td>
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<tr>
<td>HEV</td>
<td>Haemagglutinating encephalomyelitis</td>
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<tr>
<td>IP</td>
<td>Infected premises</td>
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<tr>
<td>LDCC</td>
<td>Local disease control centre</td>
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<tr>
<td>OIE</td>
<td>World Organisation for Animal Health [Office International des Epizooties]</td>
</tr>
<tr>
<td>RNA</td>
<td>Ribonucleic acid</td>
</tr>
<tr>
<td>SP</td>
<td>Suspect premises</td>
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</table>
REFERENCES


**Training resources**

*Exotic Diseases of Pigs* (56 slides), available from the Animal Diseases/Incidents Section, DPIE, Canberra.

[See the Summary Document for a full list of training resources.]

**OIE publications**


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