

AUSTRALIAN VETERINARY EMERGENCY PLAN

AUSVETPLAN

1996

Disease Strategy

Vesicular exanthema

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 and this electronic version which we can unfortunately not avoid.

© Commonwealth of Australia and each of its States and Territories 1996
ISBN 0 642 24506 1

This work is copyright and apart from any use as permitted under the *Copyright Act 1968*, no part may be reproduced without the written permission from the publisher, the Department of Primary Industries and Energy, acting on behalf of the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ). Requests and inquiries concerning reproduction and rights should be addressed to the AUSVETPLAN Coordinator.

The Commonwealth/States/Territories gives no warranty that the information contained in *AUSVETPLAN* is correct or complete. The Commonwealth shall not be liable for any loss howsoever caused whether due to negligence or other arising from use or reliance on this code.

PREFACE

This **Disease Strategy** for the control and eradication of **vesicular exanthema (VE)** is an integral part of the **Australian Veterinary Emergency Plan**, or AUSTVETPLAN (Edition 2.0). AUSTVETPLAN structures and functions are described in the **Summary Document**.

This strategy sets out the disease control principles that were approved in February 1991 by the then Australian Agricultural Council out-of-session at meeting number 135 for use in a veterinary emergency caused by the introduction of VE to Australia. This strategy has been updated and approved by the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ) out-of-session in January 1996.

Vesicular exanthema is not designated as either a List A or List B disease by the Office International des Epizooties (OIE) and therefore does not provide any guidelines for the importation of livestock and products from countries infected with VE. In practice, the OIE guidelines for swine vesicular disease may be applicable (see the **Swine Vesicular Disease Strategy, Appendix 3**).

VE is included in the list of diseases for which arrangements exist under the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases. Information on the cost-sharing arrangements can be found in the AUSTVETPLAN **Summary Document** and in the **Valuation and Compensation Manual**.

Detailed instructions for field implementation of the strategies are contained in the AUSTVETPLAN **Operational Procedures Manuals** and **Management Manuals**. Cross-references to strategies, manuals and other AUSTVETPLAN 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:

The AUSTVETPLAN Coordinator
Animal Diseases/Incidents Section
Livestock and Pastoral Division
Department of Primary Industries and Energy
GPO Box 858
Canberra ACT 2601
Tel: (06) 272 5540; Fax: (06) 272 3372

Membership of writing group

Laurie Denholm (convenor)	NSW Agriculture
Ian Bell (previous convenor)	NSW Agriculture
David Kennedy	NSW Agriculture
Harvey Westbury	Australian Animal Health Laboratory, VIC
Bob Cottam	Department of Primary Industries ,QLD

The writing group was responsible for drafting this strategy. However, the text may have been amended at various stages of the consultation/approval process and the policies expressed in this version do not necessarily represent the views of all members of the writing group. Contributions may also have been made by other people not listed above and the assistance of all involved is gratefully acknowledged.

CONTENTS

PREFACE.....	iii
Membership of writing group.....	iv
1 NATURE OF THE DISEASE	1
1.1 Aetiology.....	1
1.2 Susceptible species.....	1
1.3 World distribution and occurrence in Australia	1
1.4 Diagnostic criteria	2
1.4.1 Clinical signs	2
Pigs	2
Marine mammals	3
1.4.2 Pathology.....	3
1.4.3 Laboratory tests	3
Specimens required.....	3
Transport of specimens.....	3
Laboratory diagnosis	3
1.4.4 Differential diagnosis.....	4
1.5 Resistance and immunity	4
1.5.1 Innate and passive immunity	4
1.5.2 Active immunity	4
1.5.3 Vaccination.....	4
1.6 Epidemiology	5
1.6.1 Incubation period.....	5
1.6.2 Persistence of virus.....	5
General properties/environment.....	5
Live animals.....	5
Animal products and by-products.....	5
Fomites	6
Vectors.....	6
1.6.3 Modes of transmission.....	6
Live animals.....	6
Artificial breeding.....	6
Animal products/pig feed.....	6
Airborne spread	7
Fomites	7
Vectors.....	7
1.6.4 Factors influencing transmission	7
Environmental temperature.....	8
On-farm surveillance for signs of VE	8
Shedding of virus.....	8
1.7 Manner and risk of introduction into Australia	8
2 PRINCIPLES OF CONTROL AND ERADICATION	9
2.1 Introduction.....	9
2.2 Methods to prevent spread and eliminate pathogens.....	9
2.2.1 Quarantine and movement controls	10

	Quarantine of infected and dangerous contact premises.....	10
	Movement controls	11
	Zoning.....	11
2.2.2	Tracing.....	11
2.2.3	Surveillance	11
2.2.4	Treatment of infected animals	12
2.2.5	Destruction of animals.....	12
2.2.6	Treatment of animal products and by-products	12
2.2.7	Disposal	13
2.2.8	Decontamination.....	13
2.2.9	Vaccination.....	14
2.2.10	Wild animal control	14
2.2.11	Vector control.....	15
2.2.12	Sentinel and restocking measures.....	15
2.2.13	Public awareness	15
2.3	Feasibility of control in Australia.....	15
3	POLICY AND RATIONALE.....	16
3.1	Overall policy for vesicular exanthema.....	16
3.2	Strategy for control and eradication	17
3.2.1	Stamping out.....	17
3.2.2	Quarantine and movement controls	17
	Zoning.....	18
3.2.3	Treatment of infected animals	18
3.2.4	Treatment of animal products and by-products	18
3.2.5	Vaccination.....	19
3.2.6	Tracing and surveillance.....	19
3.2.7	Decontamination.....	19
3.2.8	Media and public relations	19
3.3	Social and economic effects.....	20
3.4	Criteria for proof of freedom.....	21
3.5	Funding and compensation	21
3.6	Strategy if the disease becomes established.....	21
APPENDIX 1	Guidelines for classifying declared areas.....	23
APPENDIX 2	Recommended quarantine and movement controls	24
APPENDIX 3	OIE International Animal Health Code	27
APPENDIX 4	Procedures for surveillance and proof of freedom	27
GLOSSARY		28
	Abbreviations	30
REFERENCES.....		31
	Further reading	31
	Video/training resources.....	32
	OIE publications.....	32
INDEX		33

1 NATURE OF THE DISEASE

Vesicular exanthema (VE) is an acute, highly infectious viral disease of pigs and marine mammals. The disease is characterised by marked elevation of body temperature and formation of vesicles that are clinically indistinguishable from those caused in pigs by foot-and-mouth disease (FMD) virus. Although morbidity of VE in pigs is high, mortality is low (usually less than 5%), except in young piglets. Production losses in VE from severe weight loss, abortion and cessation of lactation may be significant. VE has not been reported in pigs anywhere in the world for more than 30 years, but VE viruses remain active in marine mammal and fish populations off the Pacific coast of North America.

1.1 Aetiology

VE viruses are members of the Caliciviridae family. At least 13 serotypes have been isolated from pigs. Several different serotypes, collectively referred to as San Miguel sea lion virus (SMSV), are also found in marine mammals. Related caliciviruses have been isolated from opaleye perch (*Girella nigricans*) and from a liver fluke of sea lions. VE virus serotypes contain numerous plaque types that vary in virulence.

1.2 Susceptible species

The pig is the only domestic animal in which natural outbreaks of clinical VE have been described. A clinically similar disease to VE has been reported in pinniped marine mammals (sea lions, fur seals and elephant seals).

Antibodies have also been detected in several species of whale, seals, sea lions and walrus, and in wild pigs, sheep, buffalo, donkeys and foxes on the Santa Barbara Channel Islands and in farmed mink in the United States that were fed on seal meat. Virus has been isolated from calves in Oregon and primates in San Diego (all on the US west coast).

Experimentally, lesions have occasionally been produced at the inoculation site in guinea pigs, hamsters, horses, monkeys, dogs and calves. Dogs may also experience a slight fever and there is one unconfirmed report of natural infection of a dog used for working pigs. Infected calves may develop pneumonia and continue to shed virus for more than 6 weeks. Mice, rats, rabbits, hedgehogs and chickens are resistant to infection.

Human cases have not been reported. However, APHIS (1983) argues that the VE viruses are probably zoonotic and Thomson (1994) reports that laboratory infection with VE virus has occurred.

1.3 World distribution and occurrence in Australia

VE has never been identified in pigs in Australasia or the Western Pacific region. Except for isolated outbreaks in Iceland, Hawaii and the mid-western and eastern states of the United States (Bankowski 1965), VE in pigs has only been reported from the Pacific coast states of the United States, notably California, where it remained endemic until 1957. VE was eradicated from domestic pigs in the United States in 1959, almost three decades after its first appearance in 1932. However, marine mammals off the west coast of North America, from Alaska to Mexico, are endemically infected with SMSV. Clinical disease

is observed in these populations from time to time. Limited serological surveys have produced no evidence of SMSV infection in marine mammals off the eastern and southern coasts of Australia.

1.4 Diagnostic criteria

[For terms not defined in the text see Glossary]

VE is clinically indistinguishable from other vesicular diseases of pigs, notably FMD. Any vesicular disease in pigs must therefore be regarded as suspicious of FMD until proven otherwise. (Recent or concurrent disease in other livestock should be investigated to assist the differential diagnosis.)

1.4.1 Clinical signs

Pigs

In pigs, the earliest clinical sign is the onset of high fever, usually within 1–3 days of infection, depending on virulence of the VE virus strain. Affected pigs have a loss of appetite, are lethargic and often unwilling to stand. Sows in late pregnancy may abort and lactating sows may stop producing milk. Disease may not be noticed in a herd until obvious lameness and vesiculation are present.

Blanched epithelium and fluid-filled blisters (vesicles) appear on the snout, in the mouth (lips, gums or tongue), on the feet (soles, interdigital skin, coronary bands and dewclaws) and occasionally on the teats and udder. In some outbreaks the foot lesions may predominate, in other outbreaks they are insignificant. Vesicles may be up to 30 mm in diameter and either flat or raised. Snout blisters may be so numerous and raised that they have the appearance of a cluster of grapes. The epithelial covering readily ruptures to leave a raw eroded ulcer, which subsequently scabs and heals within a few days.

Fever and vesiculation may be biphasic, each phase lasting 1–3 days. Primary vesicles develop during the first febrile phase, at the site of infection. The second febrile phase follows 1–2 days later, after the onset of viraemia, which persists for 3–4 days, and this phase is accompanied by the development of secondary vesicles elsewhere on the body.

Many pigs recover rapidly and uneventfully. Some cases are so mild that the disease may pass unobserved. In other cases, complications occur. Local lymphatics may swell and, in severe cases, the legs and joints may become oedematous. Affected pigs are very reluctant to walk and show considerable pain when forced to do so.

Growth rate is often retarded and target weights may not be regained before marketing. Oral and nasal lesions may be sufficiently severe to interfere with breathing and eating. Affected sows may abort and have a delayed or non-return to oestrus. Pneumonia, septicaemia, myocarditis or encephalitis occur rarely.

Foot lesions often become infected with pyogenic (pus forming) bacteria that impair healing. In these cases the hoofs may be completely lost by sloughing. A dark line of demarcation between old and new hoof growth may appear and gradually grow out (see the **Swine Vesicular Disease Strategy, Section 1** for an indication of the rate of hoof regrowth).

Morbidity is very variable but can be high. Mortality is usually negligible unless lesions become secondarily infected with bacteria. However, high mortality can occur in young piglets.

Experimentally, more severe lesions and a greater febrile response are elicited if the viral inoculum is contaminated with bacteria. This suggests that the severity of clinical disease might depend on the presence of other symbiotic or opportunistic organisms. Such organisms are more likely to be present in dirty piggeries than in those which maintain a higher standard of hygiene. The severity of clinical disease is also related to the virulence of the infecting strain and the amount of virus to which the pigs were exposed.

Marine mammals

In marine mammals, vesicles appear on the flippers. Abortion may occur, and both pneumonia and encephalitis are thought to be associated with VE infection of pinniped mammals.

1.4.2 Pathology

Gross lesions are restricted to vesicles as described in Section 1.4.1. Microscopic lesions are not highly specific.

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

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;
- from recently dead animals — fresh and formalised samples of several tissues, including brain.

Specimens should be left wherever possible to a specialist diagnostic team. For additional information see Geering et al 1995.

Transport of specimens

Unpreserved tissue and blood specimens should be chilled and forwarded to the laboratory with frozen gel packs. If delays of more than 48 hours in transit are expected, these specimens should be forwarded with dry ice. For further information see the **Laboratory Preparedness Manual, Section 6 and Appendix 3**.

Laboratory diagnosis

AAHL tests. A range of cell cultures is used for isolation and characterisation of the virus. Serological tests including serum neutralisation are available for use in contact tracing, epidemiological studies and surveillance. The diagnostic tests currently available at AAHL are shown in Table 1.

Table 1 Diagnostic tests currently available at AAHL for vesicular exanthema

Test	Specimen required	Test detects	Time taken to obtain result
Electron microscopy	vesicular fluid / epithelium	virus particles	4–6 hours
Virus isolation in tissue cultures	tissues	infectious virus	less than 24 hours
Virus identification	virus isolate	serotype identification	2–3 days
Serum neutralisation	serum	antibody	4 days
Animal inoculation	virus isolate	virus	3 days

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

Other tests. Enzyme-linked immunosorbent assay (ELISA) has been developed overseas for VE but this is not currently used at AAHL.

1.4.4 Differential diagnosis

Other infectious diseases or management problems in which signs or lesions similar to VE may be seen include:

- *exotic viral diseases* — FMD, vesicular stomatitis (VS), swine vesicular disease (SVD)
- *dermatitis* — scalding, wetting, contact dermatitis, photosensitisation
- *phytophotodermatitis* — plants containing furocoumarins (especially Umbelliferae — parsnips, celery, parsley) causing photosensitisation (Montgomery a, b 1987, Pathak 1962)
- *trauma*
- *lameness* — laminitis, bad floors, new concrete, mud, erysipelas.

1.5 Resistance and immunity

1.5.1 Innate and passive immunity

There is no available information on this type of immunity for vesicular exanthema.

1.5.2 Active immunity

Little is known about the immune reactions to vesicular exanthema virus of swine. Animals that have recovered from the disease are usually immune to that particular strain. They are not immune to challenge with other strains. Immunity persists for at least 6 months (Madin 1989).

1.5.3 Vaccination

There is no vaccine available for vesicular exanthema of swine. Vaccines are not feasible because of the large number of antigenic types of the virus. Because it has not reappeared since 1959, no research effort has been invested in this problem (Madin 1989).

1.6 Epidemiology

Key factors in the epidemiology of VE are:

- marine animals are probably the natural reservoir for VE viruses and primary source of infection for pigs;
- pigs are infected by ingestion of contaminated feedstuff or by direct contact with infected pigs;
- the virus mutates readily;
- epidemics behave unpredictably;
- the virus is fairly resistant to environmental inactivation;
- the disease may be mild and difficult to detect; and
- infected pigs excrete the virus in saliva and faeces, but not in urine, for about 12 hours before vesicles appear.

1.6.1 Incubation period

The incubation period in natural outbreaks is usually 1–3 days, although extremes of 12 hours to 12 days have been observed. Experimentally-infected pigs develop fever and local lesions within 12–72 hours of intradermal inoculation, and secondary vesiculation elsewhere on the body occurs 1–2 days later. Oral, intravenous or intramuscular infection may result in lesions anywhere on the body in 1–10 days. There is no OIE Code for VE and hence no standard maximum incubation period.

1.6.2 Persistence of virus

General properties/environment

VE virus is reasonably resistant to inactivation. The virus retains infectivity in contaminated food scraps at 7°C. It is destroyed at pH 3 or below, above pH 9. It has been further reported that VE virus has similar stability to FMD virus. It is inactivated in 60 minutes at 62°C or in 30 minutes at 64°C (MacDiarmid 1991).

Madin (1981) claimed that VE virus is moderately resistant to inactivation, and that heavily contaminated premises must be considered infectious for several months in the absence of rigorous decontamination. Bankowski (1965) reported that a pig farm that had suffered recurrent outbreaks of VE was depopulated and 7 days later, and 7 weeks after the peak of the most recent outbreak, 30 susceptible pigs were introduced and allowed to roam over the premises for one month. No disease or other evidence of infection were found in the group.

The VE virus does not contain lipid and is therefore not susceptible to detergent but can be inactivated by most common disinfectants, including acids (see Section 2.2.8).

Live animals

Experimentally infected pigs shed virus for 12 hours before vesiculation occurs, and thereafter for 84–108 hours. Susceptible pigs placed in contact with infected pigs 100 hours after vesiculation did not become infected (Bankowski 1965). Virus was present in a wide range of pig tissues for at least seven days after infection. Tissues from a herd that had last shown signs of disease 84 days previously were still infective with a strain of low virulence (Bankowski 1965).

Animal products and by-products

VE virus can survive in laboratory media for up to 6 weeks at room temperature and for years at refrigerator temperatures. Contaminated meats remain infectious for up to 4 weeks at 7°C and for 18 years at -70°C. Earlier reports claimed that cooking under pressure at 84°C was not enough to destroy infectivity (Traum and White 1941) and boiling for 30 minutes was required. A more recent report indicated, however, that to destroy the virus meat must be subjected to heat treatment so that the core temperature reaches either 80–100°C or higher for 2–3 minutes; or 70°C or higher for 25 minutes (MacDiarmid 1991).

Fomites

The potential risk of mechanical spread of VE on contaminated objects should be considered, even though experiments to demonstrate transmission of infection by indirect means have given inconsistent results. Fomites are probably not as significant as a transmission risk in VE as they are, for example, in SVD, because excretion of virus in faeces and urine is lower in VE and environmental persistence of the virus is shorter.

Vectors

It is widely believed that the primary reservoirs and natural hosts of VE virus/SMSVs are lower marine animals such as shellfish or fish. The life cycle of SMSV is not understood. Insect and wild animal vectors have also been proposed, but there is little evidence either for or against this hypothesis.

1.6.3 Modes of transmission**Live animals**

VE virus enters the host through damaged epithelia, usually the skin of the feet or snout or the oral mucosa. The virus multiplies in epithelial cells. The virus localises in lymph nodes, which may contain much virus, particularly during the febrile stage. Large quantities of virus are shed in vesicular fluids, saliva and nasal secretions. Pigs fed faeces and urine from infected pigs did not develop clinical disease but were immune to subsequent challenge (Bankowski 1965).

The disease spreads rapidly by direct contact between pigs, and movement of infected pigs is a major cause of secondary spread of disease. In 1952 VE spread through central and eastern USA with movements of infected pigs, affecting 19 States within 50 days of the index case being reported. On two occasions, diseased pigs imported from California into Hawaii were intercepted before being off-loaded.

Artificial breeding

The virus is present in and can be transmitted via semen. It is possibly in ova and could be transmitted this way.

Animal products/pig feed

Experimentally, VE has been induced in pigs by inoculation of SMSV and by feeding meat from infected seals. Meat is the main route by which the VE virus and the other closely related marine caliciviruses are transmitted to livestock. Seal meat fed to mink has served as a vehicle for a closely-related marine calicivirus. Fresh, frozen, chilled and even some cooked pork products could serve as vehicles for VE virus (MacDiarmid 1991).

Feeding of raw garbage was the principal means of spread in the United States. During a 20-year period in California, VE occurred on every commercial garbage-feeding pig farm,

accounting for 96% of all outbreaks. On the other hand, less than 1% of grain-feeding farms experienced the disease.

It is assumed that primary outbreaks start when domestic pigs are fed raw garbage containing scraps from infected marine animals, or commercial pig feed containing inadequately treated feed components. Secondary outbreaks occur when pigs are fed swill contaminated with infected pork products. Outbreaks in central and eastern United States between 1952 and 1956 were traced to premises where pigs were fed raw garbage from a transcontinental train that originated in California, where the disease was endemic. VE entered Iceland in 1955 via garbage from a US military base.

Airborne spread

There is no evidence for airborne spread.

Fomites

The disease might be spread mechanically on contaminated objects, but experiments to demonstrate transmission of infection by indirect means have given inconsistent results.

Vectors

It is possible that parasites of pigs might play a part in transmission. Lice (*Haematopinus suis*) that had fed on pigs during the acute stages of the disease remained infectious following storage at -20°C for 11 months (Bankowski 1965).

Wild animals (other than marine animals as a food source) are not thought to be involved in the spread of infection to domestic pigs.

1.6.4 Factors influencing transmission

The factors influencing transmission of VE virus are not entirely understood and some aspects of the history of VE remain unexplained.

- The disease was not reported before 1932.
- The geographical range of the disease was very limited. Almost all outbreaks were in California. Between 1952 and 1956, a series of outbreaks occurred in central and eastern United States. The origin of all of these outbreaks, as well as the few overseas events, were traced back to California.
- Sporadic outbreaks occurred in herds that were fed only grains and had no contact with VE-infected piggeries. The sources of infection were never determined.
- Apparently unconnected foci of disease have occurred simultaneously.
- Waves of disease, each lasting one or two years and separated by periods of 1 or 2 years with little disease, were observed in California. No seasonal incidence was apparent. A possible explanation is that, in marine host populations, the level of infection and virulence of endemic virus strains might fluctuate or cycle over a few years. The risk of infection to domestic pigs fed material from these animals would vary correspondingly.
- The virus mutates frequently in host animals. Mild strains might persist undetected in pig populations for extended periods. Following mutation to a more virulent or transmissible form, or under stressful conditions, this new virus type might initiate outbreaks in previously-infected pigs or other herds.
- The disease recurred in the same herd on a number of occasions, each outbreak resulting from infection with a different antigenic type of virus. Sometimes the

outbreaks followed each other so closely that scars from the previous outbreak were still evident as fresh lesions were developing in the same pigs. Two distinct outbreaks occurred in a single closed herd under stringent experimental quarantine conditions; two different antigenic types were isolated over a period of 40 days (Bankowski 1965).

Environmental temperature

VE is relatively stable over a wide range of environmental temperatures. However, it survives longer at cooler temperatures, so transmission may be enhanced in cool climates and cool weather.

On-farm surveillance for signs of VE

The disease may be subclinical or sufficiently mild to escape clinical detection, particularly if foot lesions are uncommon. Infected pigs may be inadvertently transported, for example, to market.

Shedding of virus

Massive amounts of VE virus are shed in vesicular fluid. Virus is also excreted in saliva and nasal secretions during and following the short viraemic phase, which may precede the onset of clinical signs. Risk of contact transmission is highest in the first weeks after exposure.

1.7 Manner and risk of introduction into Australia

In the absence of the disease in domestic pigs anywhere in the world, the most likely route of entry of VE into Australia at the present time would be via uncooked swill, or in feed containing imported infected material. It might also be possible for infection to be introduced by wild pigs scavenging dead marine animals on seashores. The initial outbreak could well go unnoticed and uncontrolled, particularly in wild pigs, if the signs in domestic pigs were mild, or if the owner was reluctant or slow to report sick pigs. The disease could spread widely with pig movements and could gain a substantial foothold before coming to the attention of regulatory authorities. Illegally imported uncooked seafood products and legally imported seafood products from countries with unreliable health certification or food processing technology remain a small risk.

2 PRINCIPLES OF CONTROL AND ERADICATION

2.1 Introduction

The immediate response to a suspected outbreak of VE should be as for any other exotic vesicular disease on the assumption that it is FMD (see the **Foot-and-Mouth Disease Strategy, Sections 2 and 3**). However, it may be economically and politically prudent to implement a more discreet response pending differential diagnosis if VE is strongly suspected.

Once a diagnosis of VE is confirmed, control will rely on:

- early recognition and diagnosis (see Section 1.4);
- imposing rigid quarantine to control the movement of pigs, people, vehicles, equipment and pig products, especially over IPs, DCPs and high risk enterprises (see Section 2.2.1 and Appendixes 1 and 2);
- elimination infection by prompt slaughter and disposal of animals infected with or exposed to VE virus (see Sections 2.2.5 and 2.2.7), including infected feral pigs, if possible (see Section 2.2.10);
- urgent identification of IPs and DCPs; this involves meticulous tracing of contacts with infected herds and repeated inspections and intense surveillance in the areas involved (see Sections 2.2.2 and 2.2.3);
- thorough cleaning and disinfection of the premises and of materials possibly contaminated with virus (see Section 2.2.8);
- prevention of recycling of infection through the porcine food chain by enforcement of the ban on swill feeding (see Section 1.6.3).
- testing IPs for residual contamination by restocking with sentinel pigs (see Section 2.2.12).

Where the source of infection was eliminated, little difficulty was experienced in ‘stamping out’ VE in the United States.

2.2 Methods to prevent spread and eliminate pathogens

VE is mainly spread by direct contact between infected and uninfected pigs (see Section 1.6.3). Immediate creation of a restricted area around the infected premises to prevent movement of susceptible or in-contact animals should prevent spread of the disease. Although the potential for indirect transmission of VE on fomites is low, quarantine of infected premises must include prompt and rigorous disinfection to prevent virus spread by vehicles, fomites or people. Prevention of all swill feeding is critical (unlicensed swill feeding is illegal in Australia).

Effective quarantine and movement controls are essential. Secondary spread of infection is commonly due to the movement of infected pigs. By helping to prevent further spread of virus, movement controls increase the speed and likelihood of successful eradication, and reduce the cost of control programs and compensation payouts. Initially stringent

controls on the movement and congregation of pigs should be imposed. These may be relaxed once the situation has been fully assessed.

2.2.1 Quarantine and movement controls

Quarantine and movement controls should be imposed in declared areas as follows (for further details see Appendixes 1 and 2).

- *Infected premises (IP)* — a premises on which VE is confirmed or presumed to exist. Total movement control will be imposed.
- *Dangerous contact premises (DCP)* — a premises containing susceptible animals that have been in direct or indirect contact with an IP or infected animals. Total movement control will be imposed.
- *Suspect premises (SP)* — a premises to which the possible spread of the disease is suspected. Surveillance and movement controls will be imposed. Provided there is no evidence of infection the premises will revert to normal status.
- *Restricted area (RA)* — imposed around all IPs and DCPs, including as many SPs as practical. A high level of movement control and surveillance will apply.
- *Control area (CA)* — a CA will be imposed around the RA, and include all remaining SPs. The purpose of the CA is to control movement of pigs and potentially contaminated vehicles, etc for as long as is necessary to complete trace-back and epidemiological studies. Less stringent movement control and surveillance will apply. Once the limits of the disease have been confidently defined, the CA boundaries and movement restrictions should be relaxed or removed.

Overseas experience suggests that VE disease can spread even though effective movement controls are in place. This might be due to infection of many herds via a common contaminated feed source.

Quarantine of infected and dangerous contact premises

Quarantine of IPs and DCPs prevents spread of disease by prohibiting movement of infected and potentially infected animals, animal products and materials. It is vital to apply effective quarantine measures as early as possible to limit spread of disease within or out of the area. As the virus survives outside the host, it will be necessary to restrict the movement of animals, animal products, vehicles and materials from the IP until destocking and decontamination procedures have been completed. The earlier such restrictions are imposed, the greater the chance of limiting the spread of disease from the IP.

The movement of personnel, vehicles or equipment from an IP or DCP should not be permitted without appropriate cleaning and disinfection (see Section 2.2.8 below).

While arrangements are being made for the valuation, slaughter and disposal of infected and in-contact pigs on the IP, steps should be taken to minimise risk of disease spread from the premises. Although excretion of VE virus in faeces is minimal, contact of wild animals and other stock with piggery effluent should be prevented. If possible, effluent drainage from the piggery should be blocked, particularly if there is any watercourse drainage or overflow from effluent holding ponds off the property. Effluent spreading onto pasture should be stopped. These latter points are vital if there is any risk of feral pigs coming into contact with effluent on sprayed pasture or in watercourses.

If it is not possible to slaughter all infected and in-contact pigs within a few days of the initial diagnosis, steps should be taken to minimise spread of the disease within the premises before destocking.

This will reduce the viral contamination of the premises and hence the risk of breakdown after decontamination and restocking. Restrictions on animal and personnel movement on the piggery should be practised, particularly movement of pigs between different sheds.

Movement controls

Secondary spread of VE infection is most commonly due to the movement of infected pigs. By helping to prevent further spread of the virus, movement controls increase the speed and likelihood of success of the disease eradication program and reduce the cost of control programs and compensation payouts.

The declaration of an RA will also assist in preventing disease spread by restricting movements of potentially-infected animals and materials that may have had unrecognised direct or indirect contacts with the IP. Less severe movement restrictions will suffice in the CA, which forms a buffer zone of heightened surveillance between the RA and the rest of the industry.

Initially, stringent controls on the movement and congregation of pigs should be imposed in the RA. These may be relaxed once the situation has been fully assessed. The relaxation of restrictions may take the form of a movement slow-down policy whereby no pig can leave a property unless it has been on that property for at least 14 days and has not been in contact with other pigs that have arrived on the property during the previous 14 days. Movement controls should be maintained to some degree until the disease is either eradicated or declared endemic.

Zoning

Once the extent of the outbreak has been defined by a serological survey, consideration should be given to declaring the major part of Australia to be a VE-free zone based on geographic boundaries of the declared CA. In the long term, it should be possible to eradicate VE from the infected zone.

2.2.2 Tracing

Urgent and meticulous trace-back and trace-forward of all contacts with infected animals, premises, vehicles, equipment, people, pig products, feedstuff and other materials is vital if the disease is to be effectively contained.

It is likely that the first reported case will not be the index case, and trace-back will identify other, earlier cases.

Tracing of all movements of animals and fomites for the period back to 28 days before the first case was diagnosed on the IP, should be undertaken by examining new horn growth on the feet of recovered pigs (see Section 1.4.1).

For further information see the **Control Centres Management Manual, Part 1/Section 4.4; Part 2/LRD 101**.

2.2.3 Surveillance

Surveillance during an outbreak should be carefully coordinated to optimise the available resources. Serological surveys are essential to establish the extent of the infection, and to locate mildly infected herds that otherwise may have escaped detection. Surveillance will

be most intense in the RA and will be driven by findings from the epidemiology unit. Factors such as potential spread by wild pigs could warrant increased surveillance in some areas. The intervals between inspections and surveys will depend on the observed incubation period and the resources available. Suspect premises should be inspected every third day. Every effort must be made to educate producers about the clinical signs and to report lesions.

The surveillance program in the RA must be aggressive and driven by findings from the epidemiology unit. Factors such as potential spread by wild pigs warrant increased surveillance. The intervals between inspections and surveys will depend on the observed VE incubation period (which may vary during the outbreak with the virulence of the VE virus strain) and the resources available. Suspect premises should be inspected every third day. Surveillance should be concentrated on premises considered to be at risk as a result of contact with people, vehicles and/or materials from IPs or DCPs (see Appendix 4 for details).

Broad surveillance must be maintained, with farmers, stock agents, veterinarians, abattoir workers and so on, being alerted to watch out for, and promptly report, any suspect clinical signs or lesions of VE.

2.2.4 Treatment of infected animals

There is no known specific cure for VE. Palliative treatment may alleviate the clinical signs, but will not prevent spread of infection and may make detection of infected animals more difficult, thereby permitting further spread of disease. Affected pigs should be destroyed as soon as possible after diagnosis. The use of hyperimmune serum as a prophylactic treatment in piglets exposed to infection, but before the onset of viraemia, is not recommended.

2.2.5 Destruction of animals

As soon as practicable after the diagnosis of VE has been confirmed and valuation of the stock has been agreed upon, all infected and in-contact pigs on the IP will be destroyed. (Methods for humane killing of pigs are given in the **Destruction of Animals Manual, Section 4.3**) Classification of pigs as 'in-contact' should be based on evidence or reasonable suspicion of any direct or indirect contact (eg personnel, equipment, vehicles, effluent drainage) during the period which the disease is likely to have been on the property, plus 60 days.

If the IP is a large multiunit piggery, a decision to slaughter pigs in those units on the IP where there is no evidence of VE should be delayed, in the interests of minimising compensation payments, but only where routine disease barriers between units (eg separate personnel, effective disinfection of feed trucks) were in place before the earliest possible date of introduction of VE. Any units not slaughtered out should be treated as DCPs and intensively monitored until at least 8 weeks after destocking and decontamination of the infected unit.

2.2.6 Treatment of animal products and by-products

VE can be spread if susceptible domestic or wild pigs gain access to uncooked pigmeat products contaminated with VE virus. Contaminated animal products (such as uncooked, cured or fermented pigmeat, offal) from animals that have left the IP in the period back to 30 days before the earliest possible introduction of VE into the premises must be traced

and inactivated by incineration or by heat treatment (see Section 1.6.2). If heat inactivation is to be undertaken in order to permit subsequent use of the products, care should be taken to avoid cross-contaminating equipment and other products during processing of the contaminated material. If the product is in the form of primals or large cuts, monitoring is essential to ensure that the centre of larger pieces reaches the necessary temperature, particularly if the product is frozen. Pigmeat and processed pigmeat products may be used for human or animal consumption after effective heat inactivation.

2.2.7 Disposal

The preferred method of disposal of carcasses and other contaminated material is by burial on the property. VE may remain viable in buried carcasses for some time, so if disposal by burial is used, care must be taken to ensure that carcasses are buried deeply so they cannot become exposed and, further, that the pit does not discharge effluent either to the surface or into groundwater used for stock watering at other locations. The disposal area should be fenced off to prevent access by wild pigs.

Another option is clean removal of carcasses to plants for heat treatment and subsequent salvage of animal protein. However, great care must be taken to ensure that leakage does not occur during transport, the product does not enter animal or human food chains before adequate heat treatment processing, and cross-contamination between treated and untreated products is prevented.

In this case, vehicles used for transport should be decontaminated at their destination between each load. Close monitoring of contamination control and processing procedures at the processing plant should be implemented (see the **Disposal Procedures Manual, Sections 3.1 and 3.5**).

2.2.8 Decontamination

Decontamination of materials, equipment, vehicles and personnel leaving the IP is essential. The risk associated with any vehicle or equipment that has left the IP since the earliest possible date of VE introduction should also be assessed, and if necessary, these should be traced.

Due to the sensitivity of VE virus to most common disinfectants, effective decontamination is not difficult. The virus is sensitive to common disinfectants, such as 2% sodium hydroxide, 0.1% sodium hypochlorite and 2% citric acid. However, thorough cleaning before disinfection is critical. The virus is protected by manure, fats and other organic matter, which must be removed before disinfection by cleaning.

Dead pigs and infected pens should be sprayed with an alkaline disinfectant, then carcasses, manure and other debris removed. All surfaces should be thoroughly cleaned with an industrial detergent and then sprayed with 2% sodium hydroxide or 0.1% sodium hypochlorite. After 48 hours, surfaces are washed with water to limit corrosion. Fourteen days later a further caustic soda spray is applied, followed by a wash.

Any materials that cannot be adequately cleaned and disinfected should be destroyed. Any equipment that must not be destroyed and which cannot be effectively treated with corrosive chemicals should be treated as a possible hazard and disinfected by less damaging means.

For further details see the **Decontamination Manual, Tables 2.8, 3.20 and 4**.

2.2.9 Vaccination

Vaccination has never been used to assist the eradication of VE overseas and it is unlikely to be used to control the spread of VE in Australia. Vaccination is not a practical control method for VE due to the multiplicity of antigenic types and the ability of the virus to mutate rapidly in infected herds. No commercial vaccine is available. The preferred control option is stamping out.

2.2.10 Wild animal control

Wild pigs have the potential to harbour and spread VE and pose a serious threat to disease control. Australia has large and widespread population of wild pigs that can come into contact with extensively housed domestic pigs. Contact with intensively housed stock is unlikely.

An outbreak of VE in Australia, however, involving wild pigs may have serious consequences by delaying the detection of disease, increasing the rate and extent of an outbreak, complicating and delaying disease eradication, and compromising demonstration of disease freedom (Wilson and O'Brien 1989). The actual or potential role of wild pigs must therefore be assessed early in a VE outbreak and the likelihood of contact between wild pigs and infected domestic pigs or pig carcasses should be quickly determined.

If contact could have occurred, trapping, baiting and/or shooting operations should be carried out immediately in the vicinity of IPs to detect VE in wild pigs. If clinical, serological or virological evidence of VE is found, then more extensive and systematic epidemiological studies should be undertaken to monitor the extent and spread of the disease in the wild pig population, and to determine whether the disease initially spread from domestic to wild animals or vice versa.

If serological or epidemiological investigations indicate there has been contact between infected domestic pigs and feral pigs, destruction of all feral pigs in the area should be implemented immediately, being careful to avoid dispersing the local population in the process. If VE is confirmed in wild pigs, the source of infection and the method of spread amongst wild pigs should be determined. If wild pigs are only being infected via domestic pigs, it is possible that once this source is eliminated, infection might die out naturally in low density wild pig populations.

If wild pigs are a primary source of virus or infection is being maintained in wild populations, then monitoring and control programs must be instigated. This might involve improved fencing around extensive piggeries and municipal and other garbage tips, or by containing, reducing or eliminating (without dispersal) wild pig populations in the RA and CA to a level where the disease is unlikely to be transmitted and may die out. Models have been developed to predict the threshold population densities and cull rates at which FMD should die out in wild pig populations (Davidson 1990). Such models could probably be adapted to VE. The best method or combination of methods will depend on the prevailing circumstances, including the distribution and abundance of wild pigs, the terrain, and the availability of suitable labour and equipment.

The **Wild Animal Control Manual (in press)** will contain further details on performing wild pig population surveys, containment, control, and disease surveillance.

2.2.11 Vector control

Vector control is not required in order to limit the spread of VE.

2.2.12 Sentinel and restocking measures

Restocking may commence 14 days after the completion of decontamination. Sentinel replacement animals should come from herds with high health status to ensure that diseases not originally present on the farm are not introduced. VE susceptible pigs (in the order of 10% of full stock numbers) should be in contact with all previously contaminated areas and observed closely for 4 weeks. If there are no signs or serological evidence of infection after 4 weeks, full restocking should be allowed. However, monitoring and movement controls should be maintained for a further 4 weeks.

2.2.13 Public awareness

Outbreaks of VE should be well-publicised, with emphasis on the dangers of feeding animal products to pigs and the fact that unlicensed swill feeding is illegal. People caught feeding or providing material for swill should be promptly prosecuted and successful cases publicised. Security at municipal garbage tips should be improved to prevent wild pigs gaining access to domestic food scraps. The disease was eliminated from the United States within 3 years of the introduction of mandatory cooking of swill for pigs.

The media campaign must also emphasise the importance of farmers inspecting their pigs regularly and promptly reporting any suspicious lesions or unusual illness. The public must not be panicked into avoiding pigmeat products. The campaign should give facts on the disease, control measures, movement restrictions and the safety of products.

2.3 Feasibility of control in Australia

If the VE outbreak is identified whilst confined to a single or a few commercial piggeries, control will not be difficult. Successful strategies for control are known from the United States outbreaks. Conversely, VE introduced into the wild pig population may well become widely spread through remote areas before it is detected. Under these circumstances eradication will be very difficult.

3 POLICY AND RATIONALE

3.1 Overall policy for vesicular exanthema

Vesicular exanthema (VE) is not classified as an OIE List A or B disease, but is considered an important disease of pigs because it can be confused with foot-and-mouth disease (FMD).

The policy is to eradicate VE in the shortest possible period, while limiting economic impact, using a combination of strategies including:

- ☞ *rapid identification of the virus* to differentiate it from foot-and mouth disease (FMD);
- ☞ *stamping out*, which involves quarantine, slaughter of all infected and exposed susceptible animals on infected premises, and sanitary disposal of destroyed animals and contaminated animal products, to reduce the source of infection;
- ☞ *quarantine and movement controls* in declared areas to prevent spread of infection;
- ☞ *decontamination* of facilities, products and things to eliminate the virus on infected premises and to prevent spread in declared areas;
- ☞ *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;
- ☞ *an awareness campaign* to facilitate cooperation from industry and the community;

VE was eradicated from the United States, where it mainly occurred, in 1956. However, it has the potential to reappear as a disease in pigs as reservoirs of virus may still exist in marine and other animals.

VE has similar clinical signs in pigs to FMD and is therefore an important differential diagnosis for FMD as well as an important disease in its own right. Early diagnosis will be important because FMD will have to be considered. Delay in the definitive diagnosis may have a major effect on international trade for a range of commodities until FMD is excluded. If the disease becomes established, ongoing recurrent outbreaks would result in periodic disruption to our international markets. Zoning, under these circumstances, would be a major advantage.

Vesicular exanthema is 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) along with 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, Part 1, Sections 3 and 4**.

3.2 Strategy for control and eradication

The strategy selected will depend on a thorough assessment of the situation at the time. The strategy will need to be reassessed during the course of an outbreak and altered if necessary.

The strategy will be to apply stamping out of infected premises, and quarantine and movement controls to prevent spread. Tracing and surveillance will play important roles in determining the cause and distribution of the disease, so that appropriate levels of control can be imposed. Decontamination will be necessary to eliminate the virus on the infected premises.

Although the disease results in high morbidity, it usually has a low mortality and the destruction of large numbers of animals may not be appropriate if a number of properties become involved (see Section 3.2.1). However, while the virus may only cause mild disease in some cases, it is capable of mutation and can cause severe clinical signs resulting in higher mortality as well as production and economic loss.

3.2.1 Stamping out

Stamping out is the only viable option for the eradication of VE in domestic pigs. The possibility of confusion with FMD makes the presence of VE in Australia unacceptable and its eradication of vital importance.

Action on properties to which dangerous contacts have been traced will depend on tactical decisions taken according to circumstances, including the presence of other domestic pigs or wild pigs in the immediate area, design of the piggery with respect to containment and isolation, and the numbers of animals at risk. If the DCP contains only small numbers of pigs, stamping out will be undertaken. Alternatively, if a large number of animals are involved and effective quarantine of the DCP can be established, close monitoring of stock in the DCP for signs of disease or seroconversion may be preferable to stamping out, with a resultant saving in compensation.

3.2.2 Quarantine and movement controls

Quarantine and movement controls will be imposed immediately a vesicular disease is suspected and will include controls on the IP as well as on DCPs and SPs, as these are identified through tracing and surveillance.

A restricted area and a control area will be declared to ensure adequate control measures are imposed. The RA will have a radius of at least 1 km around the IP — which may be

altered as other properties are identified — and will include the IP and DCPs and as many SPs as possible. The boundary size of the RA must take into account the presence of wild pigs. These must be included in the area, particularly if it is not known whether there is infection in the population.

The CA boundary must be at least 10 km outside of the RA boundary. At the beginning of the control program the CA may cover a larger area but this can be modified over time as the extent of disease spread becomes clearer.

There will be controls on IP, DCP and SPs and movements of live animals, products, people and things will be strictly controlled. Pigs on the IP and, most probably, the DCP(s) will be slaughtered. Pigs on an SP will be subjected to surveillance for a period of at least 28 days and, if the results are negative for VE, they may be moved (under permit) for slaughter at an approved abattoir. In most cases movement out of the RA will be prohibited even from properties that are not affected.

There should be controls over disposal of faeces and effluent as well as pig products.

A thorough investigation into possible swill-feeding practices must be undertaken and the practice stopped.

Movement controls within the CA will not be as severe but will be consistent to enable effective control of pigs, people and things.

Live pig sales within the RA will not be permitted but it would be helpful to producers and for the control strategy, to have a processing establishment within the RA to enable restricted marketing of animals.

In view of the possibility that VE may be carried by humans (see Section 1.2), personnel involved in slaughter and decontamination procedures on IPs should not have contact with domestic pigs for one week following completion of decontamination.

For further information see Appendixes 1 and 2.

Zoning

Zoning may be adopted if eradication is prolonged and there would be advantages for both the domestic trade and in maintaining access to our few overseas markets. Zoning may also be important if the disease entered the feral pig population and became difficult to eradicate.

The infected zone would need to include the RA and the CA, and also comply with the recommendations of individual trading partners.

3.2.3 Treatment of infected animals

The treatment of infected animals will not be permitted under any circumstances as it would interfere with eradication.

3.2.4 Treatment of animal products and by-products

The virus may persist in, and be transmitted by, animal products and by-products and these must be subjected to adequate controls. Product from infected animals and infected premises will be destroyed and disposed of by burial, on the premises if possible.

Carcases of non-affected animals from premises that are under controls may be salvaged if the meat is heat treated, allowing for a safety margin, at 100°C for 1 hour (see Section 1.6.2). Normal rendering temperatures will destroy the virus and carcasses of infected and

in-contact animals may be disposed of in this manner. Infected (or suspect) feed may also be rendered.

3.2.5 Vaccination

During eradication campaigns vaccination may hinder detection of infection by masking clinical signs. The virus does not spread in a manner that necessitates containment of infection by ring vaccination. Vaccination therefore has no useful role in the control of VE.

3.2.6 Tracing and surveillance

All live pigs, people, vehicles, products and things should be traced for the period of 28 days before the first signs of clinical disease and up to the time that quarantine is imposed.

All IPs, DCPs and SPs must be quickly identified and appropriate controls imposed. Surveillance will be required to determine the distribution of the disease so that appropriate control areas may be declared. SPs will be subject to surveillance for at least the incubation period of 28 days and, for DCPs, depending on the strategy, any remaining animals would also be surveyed for this period. The surveillance program will involve inspections of pigs on DCPs and IPs every third day.

Product from IPs must be traced and destroyed. As swill feeding is a major cause of spread, any swill feeding must be stopped.

Feedstuff should be included in any trace-back and surveillance strategies to attempt to determine the cause of the outbreak.

Wild pig populations in the vicinity of the IP must be included in the surveillance program.

Sentinel animals may be introduced onto IPs and DCPs 28 days after depopulation and decontamination. These animals will be surveyed, including inspections, for a period of at least 28 days.

A detailed and statistically-based surveillance program will need to be undertaken following eradication to confirm proof of freedom. This must include feral pig populations (see Appendix 4).

3.2.7 Decontamination

The decontamination and disposal of products, people and vehicles leaving any infected premises will need to be undertaken. Faeces will be treated and buried. Effluent must be controlled and treated before release and must not be used for disposal onto pastures or crops.

Fomites, although a minor cause of spread, must be decontaminated. Feed, particularly fishmeal, must be destroyed. If carcasses are taken off the premises for disposal by rendering or other means it will be necessary to ensure the area of disposal is cleaned and disinfected after the operation.

3.2.8 Media and public relations

Industry must be fully conversant with the policies and eradication strategies that will be adopted and their views on how to implement them must be considered. This will require ongoing liaison to ensure cooperation.

The media will need to be correctly informed so that it may inform the public about the disease and the measures being taken for its control. Any information to the public should be directed towards seeking cooperation and maintaining confidence in the safety of the product for consumption.

3.3 Social and economic effects

The extent of the social and economic effects of VE would depend, largely, on how quickly it was differentiated from FMD, the severity and location of the outbreak, and the speed with which it was contained and eradicated, and the reaction of overseas countries. Different countries could apply variable criteria and might tend to overreact.

VE could affect the viability of some producers. However, the overall effect on the pig industry and on the national economy would be minor compared to a confirmed outbreak of FMD. There may, however, be some initial and short-term reactions from overseas countries until the possibility of FMD has been eliminated. This reaction could have a significant short-term financial effect on the whole of the livestock and export industries (see the **Foot-and-Mouth Disease Strategy, Section 3** for details of the potential economic and social consequences of this scenario).

For this reason, initial investigations of, and responses to, any vesicular disease in pigs should be performed rapidly but prudently until a differential diagnosis is obtained (within 48 hours of reporting). A carefully considered and balanced response must be made so as to contain infection through movement controls and alert animal health authorities of an impending exotic disease campaign, whilst minimising speculation and alarm within the industry, media and general public. There will be some disruption to both domestic and international trade.

Estimated gross value of pig production in Australia in 1993 was \$711 million, 3% of the total value of agricultural production (ABARE 1994). Less than 3% of pigmeat produced was exported.

VE might affect profitability of some producers due to production losses and lost export opportunities, but the overall effect on the pig industry and national economy would be expected to be minor. Clinical effects of VE are of little economic importance.

However, because most (97%) of Australian pigmeat is consumed domestically, bans imposed by trading partners would have little economic effect. Restrictions on movements of products within and between States would be more significant, causing local surpluses or shortages of pig meats with corresponding changes in domestic prices. Disruption to normal trade could reduce profitability throughout the industry. Adverse public reaction to publicity about the disease could lead to a consumer move away from pigmeat products to alternative meats.

Sporadic outbreaks would have a more prolonged effects on the pig industry. In the face of ongoing VE outbreaks, veterinary authorities would have to frequently initiate vesicular disease control procedures, resulting in disruption to domestic trade and a drain on financial and labour resources. An outbreak of FMD in pigs might not be reported or might not be responded to with sufficient speed due to complacency in the mistaken assumption that it was due to VE virus.

The economic loss in California due to VE during the early 1950s was estimated to be 20% of pig farm income (then \$US 887 000 annually).

3.4 Criteria for proof of freedom

OIE does not provide any guidelines for proof of freedom. Different countries could apply variable criteria to Australia. If VE was identified in wild pigs, it would also be necessary to demonstrate freedom in those populations.

For SVD, the OIE Code states that a country is considered to be free from SVD when it has been shown that the disease has not been present for at least the past 2 years. This period may be reduced to 9 months if a stamping-out policy has been implemented to deal with a discrete outbreak. OIE considers an infected zone to remain as such until at least 60 days after the last case following the completion of a stamping-out policy, or 12 months after the clinical cure or death of the last affected animal if a stamping-out policy is not practised.

In order to demonstrate that the disease had been successfully contained and eradicated, it is essential to embark on a systematic and accurate disease surveillance program during and following any outbreak of VE.

Surveillance needs to be maintained for at least 6 months to satisfy criteria for proof of freedom. This would involve intensive surveillance for 1 month followed by less intensive surveillance for 5 months. Surveillance would involve routine inspections of herds, with specimens taken from any suspicious animals, and serological surveys. Because of the possibility of virus mutation, the serology tests employed should be capable of detecting group antigens or a range of serotypes.

The number of farms examined and the number of animals on each farm inspected or sampled would be determined at the time and would depend on the severity and extent of the outbreak. Feral pig populations in and around the RA will be included. Sentinel animals should be placed and closely monitored on all former IPs and DCPs. Surveillance should be greatest in the RA. In the CA outside this area, ongoing awareness campaigns should be conducted to educate and inform pig producers. For further details see Appendix 4.

3.5 Funding and compensation

Vesicular exanthema is included in the list of diseases for which arrangements exist under the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases. Information on the cost-sharing arrangements can be found in the **AUSVETPLAN Summary Document, Appendix 3** and in the **Valuation and Compensation Manual**.

3.6 Strategy if the disease becomes established

Given the risks associated with VE and the ease with which the disease may be contained and eradicated, it is unlikely that VE would become truly endemic in Australia such that, in the long term, eradication was either not feasible or uneconomic. The situation could arise, however, where VE was regarded as an endemic disease in certain areas or domestic herds or in wild pig populations, for a period of time pending the development and application of long-term eradication strategies.

Under these circumstances, zoning could be adopted in an attempt to contain the infection and to regain partial access to international and local markets. Zoning may be applied to geographical areas, or to individual premises that are well isolated and quarantined. A

voluntary accreditation scheme could be established and producers forced to purchase replacement stock from VE-free herds. It is unlikely that vaccination would ever be justified, due to the mildness of the clinical disease, but it is an option that must be considered.

APPENDIX 1 Guidelines for classifying declared areas

Infected premises (IP)

A premises classified as and IP will be a premises (which can be all or part of a property) on which clinical VE is diagnosed or from which VE virus is isolated. This classification should remain for at least 1 month after completion of decontamination on premises that have not been restocked, or for 4 months after restocking.

Dangerous contact premises (DCP)

Premises classified as DCPs will be:

- all premises sharing a common boundary with an IP where pigs have been kept during a period back to 28 days before onset of disease on the IP;
- all premises to which any pigs or equipment that have been in contact with infected or suspect infected pigs have been moved during a period back to 28 days before the onset of clinical signs of VE on the IP; and
- all premises on which pigs have been destroyed on suspicion of VE.

Premises classified as a DCP should remain so and under quarantine and close surveillance until 30 days after the last contact with the IP, subject to continual satisfactory surveillance and a final inspection.

Suspect premises (SP)

Premises classified as SPs will be:

- all premises owned or managed in conjunction with an IP or a DCP;
- other premises on which pigs are kept within the RA;
- all premises where pigs are kept from where it is considered that the disease could have spread to the IP during the period 28 days before the onset of signs of VE on the IP, whether by movement of animals (including wild pigs), people, vehicles, equipment or feedstuff; and
- all premises to which the disease could possibly have spread from an IP by way of movement of people, vehicles, equipment or feedstuff during the period back to 28 days before signs of VE on the IP.

Subject to satisfactory surveillance, premises should remain as SPs for no more than 30 days.

Restricted area (RA)

An RA will be drawn around all IPs and DCPs, and include as many SPs as practical. The actual distance in any one direction will be determined by factors such as geographic features, and the distribution and movement of livestock and domestic and wild pigs. The boundary of the RA should be at least 1 km from the boundary of the IP or any DCP. There should be at least two stockproof barriers between the IP or DCP and the outer boundary of the RA. In areas where there are wild pigs, the RA should include an area substantially greater than the home range of any feral pigs on the IP or DCP so that any feral pigs that have come into contact with pigs or materials (eg effluent) from the IP or DCP remain within the RA. The boundaries must be modified as new information comes to hand.

Control area (CA)

The boundary of the CA should be at least 10 km from the boundary of the RA and there should be at least two stockproof barriers between the two. The CA must also substantially

exceed the home range of any susceptible feral animals that may enter the area. Initially the CA may be a much larger area pending determination of the extent of the outbreak.

APPENDIX 2 Recommended quarantine and movement controls

Infected and dangerous contact premises

Movement out of pigs:

Prohibited, except under permit for slaughter or disposal only (1).

Movement in of pigs:

Prohibited.

Movement out of pig carcasses, meat, products, offal, wastes:

Prohibited, except under permit for treatment or disposal (3).

Movement in and out of other animals, people, vehicles and equipment:

Allowed under permit (4).

Movement out of pig semen, embryos:

Allowed under permit (5).

Movement out of crops, grains:

Allowed under permit (6).

Suspect premises

May be allowed under permit (2).

May be allowed under permit (3) or after quarantine is lifted.

Allowed under permit (5) or after quarantine is lifted.

Allowed under permit (6) or after quarantine is lifted.

Restricted area

Movement out of pigs:

Prohibited.

Movement in of pigs:

Movement from a free area or CA to an abattoir is allowed under permit. Essential movement to a property may be allowed under permit (2).

Movement within of pigs:

Movement to an abattoir for immediate slaughter or to a farm may be allowed under permit (2).

Movement through of pigs:

Direct movement by road or rail may be allowed under permit, provided the origin and destination are outside the RA or CA and the stock are not unloaded en route.

Movement of pig carcasses, meat, products, offal, wastes:

Control area

Prohibited, except under permit into RA or directly to slaughter.

Movement from free areas directly to an abattoir or farm is allowed under permit (2).

Movement to an abattoir or farm is allowed under permit (2).

As for RA

Movement into or within the RA is allowed under permit (3). Movement out of the RA is prohibited.	Movement into or within the CA is allowed. Movement out of the CA may be allowed under permit (3).
---	--

Movement out of semen, embryos:

Allowed under permit (5).

No restrictions.

Risk enterprises:

May continue to operate under permit.

As for RA.

Sales, shows, etc:

All concentrations of susceptible animals are prohibited.

As for RA.

Movement in and out of people:

Allowed, subject to conditions (4).

Allowed.

Vehicles:

Vehicles used to carry pigs and porcine materials must be decontaminated between loads under supervision.

Vehicles used to carry pigs and porcine materials must be decontaminated between loads.

Notes:

- (1) Pigs on IPs and DCPs preferably should be slaughtered and disposed of on site. However, pigs may be moved within the same RA to an abattoir or knackery for immediate slaughter, subject to precautions listed in (3) below. Pigs on SPs preferably should be held on site and subjected to intense surveillance until quarantine is lifted, but may be moved to slaughter only as for pigs on IPs and DCPs.
- (2) Permits for the movement of pigs onto an IP, DCP or SP, or into the RA or CA, should be issued with caution. Although such movements may pose no risk of spreading infection, compensation may be payable if these animals become infected. Stock must remain on the property for at least 28 days and be inspected before being moved again.
- (3) Pig carcasses, meats, products, offal and wastes from IPs, DCPs and SPs preferably should be disposed of on site, or (for SPs only) held on site until quarantine is lifted. However, porcine materials may be moved within the same RA for rendering or other approved disposal provided:
 - the material is not brought into direct or indirect contact with pigs;
 - every precaution is taken to ensure that effluent or other fluids do not leak out of any transport vehicle;
 - the transport vehicle is decontaminated under supervision between each load;
 - before being released, the material is treated or processed in a manner that will destroy VE virus or ensure that it will not be fed to pigs;
 - cross-contamination between treated/clean and infected material is prevented.
 Porcine materials from other premises within the RA may be moved within but not out of the RA, subject to similar conditions. Movement of porcine materials within the CA should be allowed without restriction, and out of the CA under permit.
- (4) Movement of people, other animals, vehicles and equipment off IPs, DCPs and SPs should be restricted and subject to strict quarantine and disinfection procedures to prevent mechanical spread of VE virus. Wherever possible, movement from IPs and DCPs should be delayed until after the completion of destruction, disposal, cleaning and initial disinfection, and from SPs until after quarantine has been lifted. Within the RA, people who regularly travel from farm to farm and come into contact with pigs must clean and disinfect hands, overgear, tools and vehicles between properties and keep detailed records of their movements (see the **Decontamination Manual, Section 4.1**).
- (5) Semen and embryos collected from pigs on IPs and DCPs within 28 days preceding the first signs of VE should be destroyed and disposed of on site. Genetic material handled at the same time and

potentially cross-contaminated should also be destroyed. Material collected before this time may be removed after decontamination has been completed and the outside surfaces of containers, vials and straws have been disinfected. Other genetic material collected within the RA should be held and only released if the animals and premises of origin remain free of VE for 28 days after collection. If any doubt exists, the material should be destroyed.

- (6) Crops and grains grown on paddocks that have been sprayed with piggery effluent at any time during the 28 days preceding the likely onset of VE on the property must be disposed of on site. Other crops may be removed from IPs and DCPs after the completion of decontamination, and from SPs after quarantine has been lifted. The crops must not be fed to, or used as bedding or litter for, pigs.

APPENDIX 3 OIE International Animal Health Code for vesicular exanthema

There is no OIE Code for vesicular exanthema.

For regulatory purposes the OIE Code for swine vesicular disease can be used (see the **Swine Vesicular Disease Strategy**, Appendix 3).

★ ★ ★ ★ ★ ★ ★

APPENDIX 4 Procedures for surveillance and proof of freedom

Australia's freedom from VE will be considered after a period of six months in which no disease is detected. VE can normally be detected by physical examination of susceptible herds, but the possibility of a VE mutation strain causing only transient mild disease means that a declaration of freedom from VE will need to be supported by extensive serological testing, particularly as infection does not confer cross-immunity against other serotypes.

After the outbreak has been controlled, all properties at risk must be kept under surveillance for six months. Properties considered to be at risk are all those in the RAs as well as any other properties that may have been designated as DCPs or SPs as a result of tracing activities.

Infected premises

On IPs (and DCPs that have been destocked) restocking at 10% of full stocking will be allowed 14 days after decontamination is completed. Thereafter, surveillance visits should be made weekly and should include serological monitoring of a valid sample of the restocked pigs. After a further 4 weeks, if there is no evidence of disease or seroconversion, full restocking should be permitted. Weekly surveillance visits should continue for another month and thereafter every 4 weeks for a further 8 months.

Dangerous contact or suspect premises

Daily physical surveillance will be implemented until 28 days after decontamination of the IP. Thereafter, weekly inspections should be undertaken for a further 8 weeks. All DCPs and SPs should be serologically surveyed during this latter period.

Restricted area

On other properties where pigs are kept in the RA, surveillance visits should be made as soon as possible after detection of the first IP and then weekly for 8 weeks.

At each surveillance visit, every pen of pigs must be inspected and the previously determined numbers accounted for. Farmers should be advised to promptly report all cases of disease or death. Checks for evidence of wild pig activity should also be made as appropriate.

Once there is confidence that the disease has been contained, all pig herds within the RA should be serologically sampled to provide a 95% confidence level that the disease is not present at a prevalence of 10% or more in any herd. This testing should take place one month after the last IP has been restocked and should be repeated 3 months later. Any herds with seropositive results should be further tested for evidence of infection.

GLOSSARY

Animal by-products	Products of animal origin destined for industrial use, eg raw hides and skins, fur, wool, hair, feathers, hooves, bones, fertiliser.
Animal products	Meat products and products of animal origin (eg eggs, milk) for human consumption or for use in animal feeding.
AUSVETPLAN	A series of documents that describe the Australian response to exotic animal diseases, linking policy, strategies, implementation, coordination and counter-disaster plans.
Consultative Committee on Exotic Animal Diseases	A committee of State/Territory CVOs, AAHL and CSIRO, chaired by the CVO of Australia (Cwlth DPIE), to consult in emergencies due to the introduction of an exotic disease of livestock, or serious epizootics of Australian origin.
Control area	A declared area in which defined conditions apply to the movement into, out of, and within, of specified animals or things. Conditions applying in a control area are of lesser intensity than those in a restricted area (<i>see</i> Appendix 1).
Coronary band	Band around the top of the hoof.
Dangerous contact animal	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.
Dangerous contact premises	Premises containing dangerous contact animals (<i>see</i> Appendix 1).
Decontamination	Includes all stages of cleaning and disinfection.
Dewclaw	Functionless false hoof that represents the rudimentary first digit in animals such as deer and pigs.
ELISA	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 change when antigen–antibody binding occurs.
Fomites	Inanimate objects (eg boots, clothing, equipment, vehicles, crates, packagings) that can carry the exotic agent and spread the disease through mechanical transmission.
Immunodiffusion	A serological test to identify antigens or antibodies by precipitation of antibody–antigen complex after diffusion through agar gel.
Immunofluorescence	Technique for the location of antibodies or antigens on cells by binding of a fluorescently-tagged antibody or antigen and examination by fluorescence microscopy.
Incubation period	The time that elapses between the introduction of the pathogen into the animal and the occurrence of the first clinical signs of the disease.

Index case	The first or original case identified to have occurred in a disease outbreak.
Infected premises	<i>see</i> Appendix 1.
Local disease control centre	An emergency operations centre responsible for the command and control of field operations in a defined area.
Movement controls	Restrictions placed on movement of animals, people and things to prevent dissemination of disease.
Premises	A defined area or structure, which may include part or all of a farm, enterprise or other private or public land, building or property.
Prevalence	The number of cases of a specific disease (or infection or positive antibody titre) occurring in a given population at a particular time (expressed as the proportion of sampled animals with the condition of interest at a given time).
Quarantine	Legal restrictions imposed on a place, animal, vehicle or other things limiting movement.
Rendering (of carcasses)	Processing by heat to inactivate infective agents. Rendered material may be used in various products depending on particular disease circumstances.
Restricted area	A declared area in which defined rigorous conditions apply to the movement into, out of, and within, of specified animals, persons or things (<i>see</i> Appendix 1).
Risk enterprise	A livestock or livestock-related enterprise with a high potential for disease spread, eg an abattoir, milk factory, artificial breeding centre or livestock market.
Salvage	Recovery of some (but not full) market value by treatment and use of products, according to disease circumstances.
Sentinel animals	Animals of known health status monitored for the purpose of detecting the presence of a specific exotic disease agent.
Seroconversion	Appearance in the blood serum of antibodies following vaccination or natural exposure to a disease agent.
Serotype	A subgroup of a genus of microorganisms identifiable by the antigens carried by the members.
Stamping out	Eradication procedures based on quarantine and slaughter of all infected animals and animals exposed to infection.
State/Territory disease control headquarters	The emergency operations centre that directs the disease control operations to be undertaken in the State/Territory.
Serosurveillance	Surveillance of an animal population by testing serum samples for the presence of antibodies to disease agents.
Surveillance	A systematic program of inspection and examination of animals or things to determine the presence or absence of an exotic disease.
Susceptible species	Animals that can be infected with the disease (for VE — pigs).

Suspect animals	An animal that may have been exposed to an exotic disease such that its quarantine and intensive surveillance is warranted; OR an animal not known to have been exposed to a disease agent but showing clinical signs requiring differential diagnosis.
Suspect premises	Premises containing suspect animals (<i>see</i> Appendix 1).
Swill	Food scraps of placental mammal origin that have not been obtained from approved slaughter facilities or treated by an approved process.
Swill feeding	Swill feeding is the feeding of swill to pigs; unlicensed swill feeding is illegal in Australia.
Tracing	The process of locating animals, persons or things that may be implicated in the spread of disease, so that appropriate action may be taken.
Vaccine – 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 <i>biological</i> vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A <i>mechanical</i> vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.
Viraemia	The presence of viruses in the blood.
Zoning	Dividing a country into defined infected and disease-free areas. A high level of movement control between zones will apply.

Abbreviations

AAHL	CSIRO Australian Animal Health Laboratory, Geelong
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
CA	Control area
CCEAD	Consultative Committee on Exotic Animal Disease
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CVO	Chief veterinary officer
DCP	Dangerous contact premises
DPIE	Department of Primary Industries and Energy
ELISA	Enzyme-linked immunosorbent assay
FMD	Foot-and-mouth disease
IP	Infected premises
OIE	World Organisation for Animal Health [Office International des Epizooties]
RA	Restricted area
SMSV	San Miguel sea lion virus
SP	Suspect premises
SVD	Swine vesicular disease
VE	Vesicular exanthema
VS	Vesicular stomatitis

REFERENCES

- Australian Bureau of Resource Economics (ABARE) (1990). Gross value of Australian farm and fisheries production; Value of Australian commodity exports. *Agriculture and Resources Quarterly*. 2(2):222-228. ABARE, Canberra.
- Animal and Plant Health Inspection Service (APHIS) (1983). *Foreign Animal Disease Report*, US Department of Agriculture, APHIS. No. 11(3) September.
- Bankowski R.A. (1965). Vesicular exanthema. *Advances in Veterinary Science*. 10:23-64.
- Cutler R. (1984). Appraisal of contingency planning for exotic diseases of pigs. Victorian Department of Agriculture, Bendigo.
- Davidson S. (1990). Foot-and-mouth disease: the feral pig factor. *Rural Research*. 148:20-26.
- Geering W.A., Forman A.J. and Nunn, M.J. (1995). *Exotic Diseases of Animals: A Field Guide for Australian Veterinarians*, Bureau of Resource Sciences, Australian Government Publishing Service, Canberra.
- Henderson W.M. (1947). Vesicular lesions in farm animals. *Veterinary Record* 59:497-498.
- MacDiarmid S.C. (1991). The Importation into New Zealand of Meat and Meat Products, Ministry of Agriculture and Fisheries (MAF), New Zealand.
- Madin S.H. (1981). Vesicular exanthema of swine. In *Virus Diseases of Food Animals*, Vol 2, Disease monographs,(ed E.P.J. Gibbs), Academic Press, London, p 383-397.
- Madin S.H. (1989). Vesicular Exanthema Virus. In *Virus Infections of Porcines (Virus Infections of Vertebrates, 2)*, (ed M.B. Pensaert), Elsevier, Amsterdam, p 267-271.
- Montgomery J.F., Oliver R.E and Poole W.S.H. (1987a). A vesiculo-bullous disease in pigs resembling foot-and-mouth disease, 1 Field cases. *New Zealand Veterinary Journal*, 35:21-26.
- Montgomery J.F., Oliver R.E, Poole W.S.H. and Julian A.F. (1987b). A vesiculo-bullous disease in pigs resembling foot-and-mouth disease, 2. Experimental reproduction of the lesion. *New Zealand Veterinary Journal*, 35:27-30.
- Pathak M.A., Farrington D. and Fitzpatrick T.B. (1962). The presently known distribution of furocoumarins (psoralens) in plants. *Journal of Investigative Dermatitis*, 39:225.
- Thomson, G.R. (1994). Vesicular exanthema of swine. In *Infectious Diseases of Livestock* (eds J.A.W. Coetzer, G.R. Thomson and R.C. Tustin), Oxford University Press, South Africa.
- Wilson G.R. and O'Brien P.H. (1989). Wildlife and exotic animal disease emergencies in Australia: planning an effective response to an outbreak. *Disaster Management*, 1(3):30-35.

Further reading

- Geering W.A. (1990). Vesicular exanthema. In *A Qualitative Assessment of Current Exotic Disease Risks for Australia*, Bureau of Rural Resources, Department of Primary Industries and Energy, Canberra, p 89.
- Sahu S.P. (1987). Focus on swine vesicular disease. *Foreign Animal Disease Report*, Winter 1987, United States Animal and Plant Health Inspection Service, Veterinary Services, Emergency Programs p 8-11.
- Sawyer J.C. (1976). Vesicular exanthema of swine and San Miguel sea lion virus. *Journal of the American Veterinary Medical Association*, 169:707-709.

Smith A.W. (1976). Vesicular exanthema of swine. *Journal of the American Veterinary Medical Association*, 169:700-703.

Video/training resources

A pig's tale — why swill feeding is banned (video), AAHL 1993 (available from the Animal Diseases/Incidents Section, DPIE, Canberra; or AAHL)

Foot-and-mouth disease and other vesicular diseases (50 slides), available from the Animal Diseases/Incidents Section, DPIE, Canberra.

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

OIE publications

OIE Code (1992). *International Animal Health Code* (6th edition), OIE, Paris, France.

OIE Manual (1992). *Manual of Standards for Diagnostic Tests and Vaccines* (2nd edition), OIE, Paris, France.

INDEX

- AAHL diagnostic tests, 4
- Abbreviations, 30
- Aetiology, 1
- Airborne spread, 7
- Animal by-products, 5, 12, 18
- Animal products, 5, 6, 12, 18
- Australian Animal Health Laboratory, 3
- CCEAD, 17
- Chief veterinary officer, 17
 - States, 3
- Clinical signs, 2
- Compensation, 21
- Control and eradication
 - principles, 9
 - strategy, 17
- Control area, 10, 23
- Control in Australia
 - feasibility, 15
- Cost-sharing agreement, 21
- Dangerous contact premises, 10, 23, 27
- Declared areas
 - classifying, 23
- Decontamination, 13, 19
- Destruction, 12
- Diagnosis
 - criteria, 2
 - differential, 4
 - laboratory, 3
- Disposal, 13
- Environmental temperature, 8
- Epidemiology, 5
- Established disease
 - strategy, 21
- Fomites, 6, 7
- Funding, 21
- Immunity, 4
 - active, 4
 - innate, 4
 - passive, 4
- Incubation period, 5
- Infected premises, 10, 23, 27
- Introduction into Australia, 8
- Laboratory tests, 3
- Lesions, 2
- Media, 15
- Media and public relations, 19
- Movement controls, 10, 11, 17, 24
- Occurrence in Australia, 1
- OIE Code, 27
- OIE publications, 32
- Pathology, 3
- Persistence of virus, 5
 - environment, 5
 - general properties, 5
 - live animals, 5
- Pig feed, 6
- Policy
 - overall, 16
- Policy and eradication, 16
- Proof of freedom, 27
 - criteria, 21
- Public awareness, 15
- Quarantine, 10, 17, 24
- Resistance, 4
- Restocking measures, 15
- Restricted area, 10, 23, 27
- Sentinel, 15
- Shedding of virus, 8
- Social and economic effects, 20
- Specimens, 3
 - transport, 3
- Specimens required, 3
- Stamping out, 17
- Surveillance, 8, 11, 27
 - surveillance, 19
- Susceptible species, 1
- Suspect premises, 10, 23, 27
- Tracing, 11, 19
- training resources, 32
- Transmission, 7
 - artificial breeding, 6
 - live animals, 6
- transmission, 6
- Treatment
 - infected animals, 12, 18
- Vaccination, 4, 14, 19
- Vector control, 15
- Vectors, 6, 7
- Virus
 - transmission, 6, 7
- Wild animal control, 14
- World distribution, 1
- Zoning, 11, 18