AUSTRALIAN VETERINARY EMERGENCY PLAN

AUSVETPLAN

1996

Disease Strategy

Transmissible gastroenteritis

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.

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PREFACE

This **Disease Strategy** for the control and eradication of **transmissible gastroenteritis** (TGE), 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 for use in a veterinary emergency caused by the introduction of TGE to Australia. It has been approved by the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ) out-of-session in January 1996.

TGE 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 TGE conform with the **OIE International Animal Health Code 1992** (OIE Code; Appendix 3).

TGE is not included in the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases.

Detailed instructions for field implementation of the strategies are contained in the AUSVETPLAN **Operational Procedures Manuals** and **Management Manuals.** Cross-references 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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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

1 NATURE OF THE DISEASE

Transmissible gastroenteritis (TGE) is an acute, highly contagious viral disease of pigs. The disease is mainly seen in very young piglets and is characterised by profuse diarrhoea and vomiting with high morbidity and mortality. Pigs of all ages are susceptible to infection, however in pigs older than 5 weeks, infection is milder and mortality rates are low.

1.1 Aetiology

TGE is caused by a virus of the family Coronaviridae. Coronaviruses are responsible for two other exotic pig diseases: porcine epidemic diarrhoea and porcine respiratory coronavirus infection. There is only one serotype of TGE virus.

1.2 Susceptible species

The clinical disease occurs only in pigs. Dogs, cats, foxes and house flies may spread the diseases they shed infective virus in their faeces for a variable period of time after ingestion of the virus. Starlings have also been implicated as possible mechanical vectors. Seroconversion has been recorded following experimental oral infection in dogs and cats, though no clinical disease was recorded (McClurkin et al 1970).

Infection does not occur in humans.

1.3 World distribution and occurrence in Australia

TGE is present in most of Europe, North, South and Central America, China, Japan, The Philippines, Korea, Nepal, Myanmar (Burma), Southeast Asia and limited areas of West Africa (Ghana and Ivory Coast).

No outbreaks have been recorded in Australia, New Zealand or Norway.

1.4 Diagnostic criteria

The high level of morbidity and mortality, the age group mainly affected and the clinical signs will all assist the diagnosis of TGE in this country. Whilst TGE can produce syndromes of variable severity, the condition is most spectacular in immunologically naive (susceptible) populations, as would be the case in the Australian pig herd.

1.4.1 Clinical signs

When introduced into a susceptible herd, the disease usually spreads rapidly with some degree of appetite loss and vomiting or diarrhoea in most animals. Within 2–3 days most animals are affected. Piglets under a week old are the worst affected and the severity of clinical signs, duration of the disease and mortality rates all decline with age.

Piglets

- Piglets less than 3 weeks old become very sick, vomit, develop a profuse watery-yellow diarrhoea, lose weight and become severely dehydrated.

- Morbidity rate is usually 100% — most pigs under 10 days old will die within 2 to 7 days of the appearance of clinical signs; piglets older than 3 weeks usually survive, but are likely to fail to thrive.
- In young pigs the diarrhoea is usually profuse, foul smelling and contains curds of undigested milk.

Growing, finishing and adult pigs

- Clinical disease in older stock and adults is usually limited to loss of appetite and diarrhoea, for one or a few days and occasional vomiting.
- Some lactating sows may become ill, developing fever, failure of milk supply, vomiting, loss of appetite and diarrhoea (illness in these sows would further contribute to piglet mortality).
- Whilst the severity of clinical disease is usually mild in growing, finishing and adult pigs, the morbidity may approach 100% and mortalities of 25–30% have been recorded in 2–6 month-old pigs (Bachmann et al 1972).

When TGE becomes endemic in a herd, the clinical disease is less severe and mortality in piglets is usually less than 10–20%. The clinical disease may be restricted to diarrhoea affecting suckling pigs aged about six days or older and post weaning diarrhoea seen during brief episodes of overt clinical re-emergence of disease (Pritchard 1987). The disease is most likely to persist in large units, with some herds experiencing clinical re-emergence of disease every 3–4 months. Endemic TGE in suckling or recently-weaned pigs can be very difficult to diagnose clinically and must be differentiated from other types of endemic diarrhoea, ie colibacillosis, coccidiosis and rotaviral diarrhoea. The possibility of mixed infections should be considered, especially where treatment of an assumed endemic disease is ineffective.

It is not known whether the source of virus during a clinical reappearance of the disease is due to reactivation of virus shedding in carrier pigs or reintroduction of virus into the herd.

1.4.2 Pathology

Gross lesions

In natural infections lesions are confined to the gastrointestinal tract. The stomach is often distended with curdled milk and may be congested. A small area of haemorrhage on the diaphragmatic surface of the stomach is found in about 50% of cases. The small intestine is distended with yellow foamy fluid and contains curdled milk. The wall of the intestine may be inflamed, but is generally thin and almost transparent due to severe atrophy of the intestinal villi.

Microscopic lesions (histopathology)

Histologically, the primary lesion is marked shortening of the intestinal villi in the jejunum and ileum. The villus–crypt ratio, which is normally about 7:1, is reduced to about 1:1 in affected piglets.

Pathogenesis

The pathogenesis has been reviewed by Saif and Wesley (1992). Whether infection is via the oral or nasal route, once the virus is swallowed it passes undamaged through the stomach and attaches to the susceptible villous epithelial cells of the small intestine. Infection results in a rapid and extensive loss of functional epithelial cells and acute malabsorption syndrome. The virus is capable of multiplying in the respiratory tract and

lactating mammary glands. During acute infections virus may be shed through nasal secretions and milk (Kemeny et al 1975). Kemeny and Woods (1977) demonstrated that sows infected via intramammary inoculation subsequently shed virus in milk, faeces and nasal secretions. Natural infection of the pig foetus has not been recorded.

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

Loops of affected ileum, preferably from acutely ill cases should be tied off and stored in sterile containers on ice. Viral antigen is best detected in piglets sacrificed at a very early stage of disease. Additional sections of gut wall, both unpreserved and in neutral buffered formalin, should be collected from different parts of the small intestine. Blood samples for serology should be collected from acute and convalescent animals. Neutralising antibodies can be detected in serum as early as 7–8 days after infection.

Transport of specimens

Unpreserved tissue specimens and blood samples should be chilled and forwarded with water, ice or frozen gel packs. Intact loops of intestines must be transported to the laboratory without delay (Geering et al 1995). For further information see the **Laboratory Preparedness Manual, Section 6 and Appendix 3**.

Laboratory diagnosis

AAHL tests. A rapid presumptive laboratory diagnosis can be made by electron microscope examination of intestinal contents for virus particles or by the detection of viral antigens in intestinal epithelial cells by immunofluorescence. The diagnostic tests currently available at AAHL are shown in Table 1.

Table 1 Diagnostic tests currently available at AAHL for transmissible gastroenteritis

Test	Specimen required	Test detects	Time taken to obtain result
Electron microscopy	intestinal contents	virus particles	12–24 hours
Animal inoculation	intestinal contents	virus	2–5 days
Serum neutralisation	serum	antibody	3–5 days
Immunfluorescence	intestinal epithelial cells/frozen sections of small intestine	viral antigens	12–24 hours
Virus isolation and identification	intestinal contents	virus	5–10 days

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

Other tests. Polymerase chain reaction test can be used to differentiate transmissible gastroenteritis from other porcine coronaviruses.

1.4.4 Differential diagnosis

The following diseases with clinical signs or lesions similar to TGE need to be considered in the differential diagnosis.

Endemic diseases:
- colibacillosis
- coccidiosis
- haemagglutinating encephalomyelitis
- porcine rotavirus
- swine dysentery
- arsenic poisoning
- salmonellosis

Exotic diseases:
- classical and african swine fever
- porcine epidemic diarrhoea
- *Clostridium perfringens* type C

1.5 Resistance and immunity

1.5.1 Innate and passive immunity

An age-dependent resistance to clinical disease is well demonstrated. Infective doses of TGE virus for a 6-month-old pig are in the order of 10^4 higher than those needed to infect a 2-day-old piglet. Slow cellular turnover rates, immature enterocyte cell types and depressed cell-mediated immunity in neonates are thought to facilitate infections.

Exposing sows in late pregnancy (but more than 3 weeks prior to farrowing) to virulent virus (ie the gut and/or gut contents from infected piglets) will minimise losses from their litters. This protection is mediated through secretory immunoglobulin A (IgA) in the milk, not through colostral IgG. An uninterrupted supply of IgA in sows milk over the lactation period is required for effective protection.

1.5.2 Active immunity

Pigs that recover from enteric infections with TGE virus develop immunity shown by the appearance of antibodies of all isotypes in the circulation, as well as IgA antibodies in intestinal secretions. The IgA antibodies in intestinal secretions are the ones that provide protection rather than circulating antibodies, presumably due to local immunity within the intestinal mucosa. After primary infection there is complete protection against enteric reinfection for at least six months. If reinfection occurs after this time the effects are usually short and subclinical (Pensaert et al 1994).

The role of cell-mediated immunity in either recovery or protection against reinfection is still not clear.

1.5.3 Vaccination

Although vaccination of pregnant sows has been investigated using a variety of antigenic preparations and routes of administration, reliable vaccines have not been developed. Attenuation of oral vaccine strains reduces their ability to replicate in the sows intestine and to stimulate IgA production and secretion in milk. In seronegative sows, parenteral

vaccines tend to produce a low level of IgG antibodies in the milk and protective secretory IgA antibodies have been detected in intestinal fluids and serum after oral, but not after parenteral, inoculation of seronegative sows with TGE virus. In sows with previous exposure to infection, parenteral vaccines can significantly boost milk TGE virus antibody levels.

There is some evidence that oral vaccination of neonates with attenuated TGE virus does not provide effective protection from infection. Protection due to active immunity generally takes at least 5 days to develop. Vaccination of the piglet shortly after birth cannot provide protection during the first critical few days of life.

Immunisation of suckling or weaned pigs may be useful in the control of enzootic infections, although there is evidence that the presence of maternal antibodies suppresses active antibody production.

1.6 Epidemiology

Key factors in the epidemiology of TGE are:

- the very short incubation period;
- rapid spread of disease within herds; and
- age-related severity of clinical disease.

In large herds the disease is likely to become endemic following the initial outbreak with permanent ongoing losses. In smaller herds TGE virus may disappear from the herd following the outbreak, with a subsequent reversion to susceptibility to further outbreaks.

1.6.1 Incubation period

The incubation period in natural infections is 18 hours to 3 days. The OIE Code (Appendix 3) does not give a maximum incubation period for regulatory purposes but the maximum *infective period* is given as 40 days.

1.6.2 Persistence of virus

General properties/environment

- The virus can survive in the environment for up to 3 days, is extremely stable when frozen but is labile at room temperature or above.
- The half-life of TGE virus at 37ºC is less than 2 hours; virus in excreted faeces stored at 21ºC was found to be non-infective 10 days after excretion (Young et al 1955).
- Although there are strain differences in physical properties, TGE virus is considered to be very light sensitive; faecal material containing 10^5 pig infective doses was inactivated within 6 hours when exposed directly to sunlight.
- The virus is trypsin resistant, stable in pig bile and stable at pH 3;
- TGE virus is sensitive to lipid solvents and is inactivated by a wide variety of detergents and disinfectants (see Section 2.2.8).

Live animals

Faecal shedding is considered to persist for up to 2 weeks after infection. TGE virus has been detected in tonsil samples from slaughtered pigs and has been detected in nasal swabs for up to 11 days after exposure. The virus has also been isolated from intestinal

contents or homogenates and from lung homogenates for post-exposure periods of up to 104 days (Underdahl et al 1975). The ability of pigs to excrete viable infective virus from the respiratory and gastrointestinal tracts over these prolonged periods is unknown. The OIE Code gives the maximum infective period, for regulatory purposes, as 40 days (see Appendix 3).

The longer-term carrier status of recovered animals is difficult to assess (see Section 1.5.2).

Animal products and by-products

Carcase material from infected pigs can be a source of infection for susceptible pigs that come into contact with it (Forman 1991, Cook et al 1991). Freezing or post-slaughter acidification do not significantly affect the infectivity of TGE virus in pig products. Cooking will destroy the virus. The survival of TGE virus in salted and cured meats is unknown but even during acute infections viraemia has been difficult to detect and carcase muscle tissue is not considered a major reservoir of virus.

Vectors

Flies are believed to play a role in the mechanical transmission of the virus within piggeries, but are not considered to represent a risk of infection between farms under Australian farming conditions. No other insects have been implicated in the transmission of TGE virus.

1.6.3 Modes of transmission

The main sources of infection in 60 United Kingdom herds were believed to be the movement of pigs on and off infected premises; movement of livestock trucks that had carried pigs; and local spread to nearby farms without any obvious contact.

Within piggeries, infection is likely to spread as a result of ingestion of infected faeces from in-contact pigs, inhalation or ingestion of droplets of faeces, transfer of carrier stock, indirect transmission on implements and mechanical transmission by flies.

Live animals

Outbreaks usually start following the introduction of infected pigs. Large amounts of TGE virus are present in the faeces of affected animals. TGE virus is believed to be excreted in the faeces of recovered pigs for up to 2 weeks, although there is one report of excretion up to 10 weeks after infection (Taylor 1981).

While infection generally spreads very rapidly through a susceptible population, spread may be slower during the summer months.

Virus has been recovered from the nasal tract of infected pigs and from the milk of sows during the acute stage of the disease and piglets may become infected in this way (Kemeny et al 1975).

Artificial breeding

There are no reports of naturally-occurring transplacental infection, or of transmission by semen or ova.

Animal products

Forman (1991) and Cook et al (1991) have demonstrated that the disease may be transmitted to pigs following ingestion of uncooked muscle and lymph node material derived from slaughtered pigs from a population where TGE is endemic.

Fomites

Mechanical spread on contaminated footwear, clothing and equipment may occur but is unlikely due to the fragility of the virus..

Windborne spread

Although TGE virus can replicate in the respiratory tract, spread of the virus by aerosols does not seem to occur (Pensaert and Callebaut 1994).

Wild animals

The virus may be transmitted passively in the gut of cats, dogs, foxes and starlings. Following experimental infection, dogs, cats and foxes were found to shed faecal virus for up to 14, 22 and 15 days, respectively (Haelterman 1962). Starlings are considered to play a prominent role in transmission between herds in the United States during the winter months. The virus has been detected in the faeces of starlings for up to 32 hours after ingestion (Pilchard 1965).

Feral pigs are capable of transmitting the virus over wide distances. Apart from the domestic pig, feral pigs are the animals most likely to amplify and maintain the virus.

1.6.4 Factors influencing transmission

In North America and the United Kingdom, TGE outbreaks commonly occur in winter. Outbreaks become rare with the onset of summer. It is considered that the susceptibility of TGE virus to heat and light inactivation is responsible for the seasonal incidence of outbreaks. Winter conditions in these countries are more conducive to mechanical spread via fomites. Whilst outbreaks are rare during summer, enzootic infections are able to persist over summer, by spreading slowly through grower herds. Persistence of infection is also likely in herds with a continuous farrowing schedule.

Whilst said to be a winter disease in Europe and North America, TGE used to flourish in Singapore where the mean daily maximum temperature hovers around 30^{o}C for most of the year (R. Webster, Queensland Department of Primary Industries, pers. comm.).

1.7 Manner and risk of introduction

The greatest risk of introduction of TGE would occur if pigs were to be imported from countries with endemic infection. As commercial importation of live pigs into Australia is currently prohibited, the greatest risk lies in the introduction of the virus in fresh or frozen pigmeat and the feeding of infected meat as swill to pigs. Unlicensed swill feeding is illegal in Australia.

2 PRINCIPLES OF CONTROL AND ERADICATION

2.1 Introduction

The importance of TGE lies in the economic impact of the clinical disease. With TGE it is unlikely that introduced infections will go unnoticed in herds with a continual farrowing production system.

Eradication of TGE has neither been attempted nor achieved in any country once infection has become widespread. Ireland is the only country to claim freedom from TGE after a successful eradication program. In this case, clinical disease was restricted to one breeder unit, although serological reactors were found in two associated units. The eradication program was based on quarantine and depopulation of the clinically infected herd. On the other two units extensive serological testing of weaner pigs and breeding animals led to the slaughter of serological reactors. When evidence of seroconversion was found in finisher pigs a program of serial depopulation, cleaning and disinfection was undertaken in the fattening rooms. More recently eradication has been achieved on individual farms in the United States (Harris et al 1987, Wiseman et al 1988, Fitzgerald and Welter 1990).

There are three possible methods to eradicate TGE, each of which is described briefly below.

2.1.1 Stamping out

Quarantine of infected properties (IPs) and dangerous contact premises (DCPs), and destruction of all pigs on IPs (and possibly some on DCPs, according to circumstances) would be the most reliable method to eliminate TGE virus. However, not all infected piggeries may be identified because of owner reluctance to report suspect infection especially if compensation is not available. This option could be considered when the infected herd is small and isolated.

Under this policy live pigs would not be permitted to move from the IPs or DCPs. Only carcases could be moved to another property for burial or to an approved place for rendering.

Personnel and fomites entering and leaving infected premises should be restricted to minimise movements and cleaning and disinfection procedures should be instigated.

All movements of pigs to saleyards, or equivalent centres, within an area in which the disease is suspected or present should be prohibited.

Release from quarantine may occur two weeks after all pigs have been removed and the decontamination/disinfection program has been completed.

2.1.2 Salvage and slaughter out

This method of control requires quarantining of the IP and slaughter of all saleable infected pigs at an abattoir. Animals that are not saleable would be destroyed on the IPs. Pigs showing clinical symptoms should not be sent to an abattoir and should be destroyed on the IP, or held in quarantine until the symptoms pass.

Pigs would be permitted to move to an approved abattoir for immediate slaughter. Immediate slaughter will minimise the contamination of lairages by pigs shedding TGE

virus, thus preventing infection of pigs from other piggeries. Killing all pigs from an IP within 4 hours of arrival at the abattoirs and ensuring that all pigs received at that abattoir are killed within 18 hours will minimise the number of viraemic carcases entering the food chain.

Personnel and fomites entering and leaving IPs should be restricted to minimise movements and cleaning and disinfection procedures should be instigated.

All movements of pigs to saleyards, or equivalent centres, within an area in which the disease is suspected or present should be prohibited.

Release from quarantine may occur two weeks after all pigs have been removed and the decontamination/disinfection program has been completed.

2.1.3 Eradication by controlled exposure

This on-farm procedure has been shown, in clinical trials, to eliminate the virus on infected properties.

The method involves quarantine of the infected premises followed by active dissemination of the virus throughout the infected herd, to ensure all pigs are infected and develop an active immunity. This will reduce the susceptible population within the piggery. As the virus is excreted from infected animals for only a short period of time after infection, this process of active infection, followed by disinfection of the piggery, is designed to eliminate the virus from the piggery. The virus can either be sourced from field strains or attenuated ('live') vaccines.

Pigs permitted to move to an approved abattoir should be slaughtered immediately to minimise the contamination of lairages by pigs shedding TGE virus, and thus preventing infection of pigs from other piggeries. Killing all pigs from an infected premises within 4 hours of arrival at the abattoirs and ensuring that all pigs received at that abattoir are killed within 18 hours will minimise the number of viraemic carcases entering the food chain.

Personnel and fomites entering and leaving an IP should be restricted to minimise movements, and cleaning and disinfection procedures should be instigated.

All movements of pigs to saleyards, or equivalent centres, within an area in which the disease is suspected or present should be prohibited.

Release from quarantine should occur following the satisfactory monitoring of sentinel animals over a 60-day period. The whole controlled exposure program will require a minimum of 130 days to complete.

See Appendix 5 for further details.

2.2 Methods to prevent spread and eliminate pathogens

2.2.1 Quarantine and movement co ntrols

Prevention of the spread of TGE virus from infected piggeries would require the imposition of stringent quarantine and movement controls. Whilst movement of infected and virus-excreting pigs represents the greatest risk of spread, spread by fomites is well established and spread by birds and other animals is suspected. Effective movement controls include: standstill on pig movements off the infected premises (IP), dangerous contact premises (DCPs) and suspect premises (SPs); decontamination of all vehicles in

contact with infected pigs; restricted movement of people and vehicles on and off the IPs, DCPs and SPs.

A *restricted area* (RA) and *control area* (CA) will be declared to provide the necessary control to enable eradication measures to be implemented. The RA should include the IPs, DCPs and as many of the SPs as possible. The size of the declared areas will depend on epidemiological information at the time of the outbreak and should be as large as is necessary for satisfactory control. There would be a ban on live pig sales within the RA.

For further details see Appendixes 1 and 2.

Zoning

If a disease is endemic in only part of a country, it is possible to establish diseased and disease-free zones. Tight controls on the movement of pigs would have to be enforced between zones. Zoning is of most benefit when there are implications for international trade. TGE infection is not considered an impediment in international meat trading, although the small export market in breeding stock is likely to be affected.

Zoning may help to prevent spread of infection within regions of Australia. It may also help or hinder domestic trade, depending on whether the state or region is a net exporter or importer of pigs and/or pig products. Zoning restrictions placed along State borders would severely restrict existing marketing arrangements involving the frequent long distance and interstate transport of both breeding and slaughter pigs across Australia.

The risk of disease transmission through contaminated product is associated with swill feeding. Product movement controls should only be imposed if the regulation of swill feeding is not considered satisfactory.

2.2.2 Tracing

When infection is suspected or confirmed in a piggery, trace-back and trace-forward identify other infected piggeries. In an outbreak of TGE the most important tracing is of pig movements. The movement of personnel and fomites is of secondary consideration, with movements of other domestic animals and wild animal movements of least importance. Trace-back should extend to those piggeries receiving pigs from infected properties 30 days prior to the first recorded clinical case.

2.2.3 Surveillance

Surveillance will be initially targeted to estimate the spread of infection, thus assisting in developing the control strategy. Further surveillance may be needed in implementing the control strategy.

As live pig movements are the most likely route of disease spread, special attention should be placed on piggeries with a history of recent introductions and piggeries selling breeding or grower stock. Closed herds have a much lower likelihood of introducing TGE virus and therefore breeding stock sold from closed herds are less likely to spread the disease.

The purpose of surveillance is to identify any infected piggeries not already identified by tracing. Activities include locating piggeries followed by physical inspection of pigs and examination of production records for evidence of piglet scours and mortalities.

Serosurveillance would be of most value in herds where the clinical syndrome is not classical, that is, herds where infection is well established or has become endemic, and in grower units where only mild signs may be seen due to the age of the susceptible animals.

2.2.4 Treatment of infected animals

There is no specific cure for TGE. Treatment of affected animals is limited to nursing and supportive care, that is fluid therapy to alleviate starvation dehydration and acidosis, and provision of extra warmth for young pigs.

2.2.5 Destruction of animals

The need for destruction/stamping-out procedures will depend on which policy for the eradication of TGE is adopted (see Sections 2.1.1–2.1.3). If the stamping-out policy is adopted (2.1.1) pigs will be slaughtered on the IPs, and possibly on DCPs, according to circumstances. If the policy is for salvage (2.1.2) the pigs will be transferred to an abattoir for slaughter. Non-saleable or clinically-affected pigs not suitable for slaughter at an abattoir must be slaughtered on the IP.

See the **Destruction Manual, Section 4.3,** for appropriate methods for the destruction of pigs.

2.2.6 Treatment of animal products

Current processing (except possibly curing, see section 1.6.2) and rendering techniques of pig products are sufficient to inactivate TGE virus. Therefore such products present minimal threat in spreading disease. The only threat is from meat from viraemic pigs when fed raw to susceptible pigs (see Section 1.7). Hence intensifying publicity and policing of swill-feeding bans is appropriate during the outbreak.

2.2.7 Disposal

There are no special considerations in disposal of TGE virus infected pigs. Pig carcases may be rendered (see the **Disposal Manual, Section 3.5**).

2.2.8 Decontamination

Total decontamination is only appropriate after piggeries have been depopulated. TGE virus is susceptible to sunlight, high temperatures and a range of chemicals including 0.03% formalin, 1% phenol, 0.01% beta propiolactone, sodium hypochlorite, sodium hydroxide, iodines, quaternary ammonium compounds, ether and chloroform (see the **Decontamination Manual, Tables 1, 2 and 3**). Thorough cleaning and disinfection of vehicles used to transport infected pigs, loading ramps at abattoirs and other potentially infected fomites, will minimise spread of infection. Disinfection is also a component of the 'eradication by controlled exposure' procedure outlined in Section 2.1.3 and Appendix 5.

Destruction of animals other than pigs is unnecessary. Destruction of property is unlikely to be necessary as TGE virus is susceptible to sunlight, high temperature, cleaning and disinfection.

2.2.9 Vaccination

Commercial TGE virus vaccines, currently available overseas, provide poor protection in susceptible (naive) TGE pigs but are effective in boosting immunity in previously-infected pigs (see Section 1.5.3).

Vaccination should not form the basis of a control program where eradication of the virus is intended. The use of specific vaccination may be indicated as an adjunct to specific control programs (see Section 2.1.3; Appendixes 4 and 5).

2.2.10 Wild animal control

Controlling wild animals capable of becoming infected with TGE virus, ie feral pigs, foxes, dogs, cats and birds, by preventing access to infected piggeries including effluent disposal sites and carcase burial or disposal sites, will prevent wild animals from spreading the disease. This is achieved by perimeter fences, bird-proofing and population control. As birds are considered important for the spread of TGE in North America, this may be a significant supportive control measure.

2.2.11 Vector control

The role of flies in spreading TGE virus from infected to uninfected piggeries has not been established. Fly control may minimise spread of the virus on infected premises.

2.2.12 Sentinel and restocking measures

Destocked piggeries can be restocked with minimal risk of reinfection 14 days after completion of decontamination.

Sentinel animals would be required in the program of eradication by controlled exposure (see Section 2.1.3) as infective virus may remain in the piggery and in carrier pigs. Sentinels should be by monitored for clinical disease and absence of seroconversion over a 60-day period (Wiseman et al 1988). The absence of seroconversion should be considered the more sensitive test (see Appendixes 4 and 5).

2.2.13 Public awareness

Outbreaks of TGE 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 tightened to prevent wild pigs gaining access to domestic food scraps.

Piggery owners should be advised to adopt adequate precautions to prevent entry of TGE virus. Ideal precautions are:

- no pig introductions (unless from herds known to be TGE-virus free);
- minimise the number of visitors. Those that do enter, to use boots and overalls held on the piggery;
- perimeter fences to exclude wild and domestic animals;
- feed bins on perimeter fences;
- pig loading facilities at perimeter fences;
- cleaning and disinfection of pig-carrying trucks after unloading; and
- bird proofing.

For further information see the **Public Relations Manual**.

2.3 Feasibility of control or eradication in Australia

In the face of an outbreak, authorities may attempt to actively intervene, or allow the industry to develop and adopt its own control measures with minimum regulation. Control of the disease would then be up to individual farmers, and it is likely that most farmers would have to live with the disease. A number of producers would avoid the disease through herd security measures, however, currently the Australian pig industry is largely structured on the movement of replacement breeding stock around the country. These movements will increase the risk of spread. Following infection some producers would attempt to eliminate infection, through farm-based eradication programs. Success of these programs relies on a high level of planning, skilled farm management and all-in-all-out farm facilities.

In Ireland following an outbreak in 1984 involving three pig herds TGE was successfully eradicated. The disease has also been successfully eradicated on an individual herd basis in the United States. This indicates eradication may be feasible if the disease is diagnosed promptly after the initial infection and there has been very limited spread. This must be assessed after intensive tracing and surveillance. Environmental conditions in Australia are frequently not conducive to TGE virus spread. Another important consideration in assessing feasibility is that farm management of the IP is of high standard and able to cope with the complex and ongoing arrangements that may be required in an eradication program.

Notwithstanding this, there is a high likelihood that the index case will occur in a herd that either contains illegally-imported pigs or has been illegally swill fed. In such a case it is not likely that the criteria favouring successful eradication, ie rapid diagnosis and limited spread, will be met and eradication in such circumstances may not be feasible.

3 POLICY AND RATIONALE

3.1 Overall policy for transmissible gastroenteritis

Transmissible gastroenteritis (TGE) is an OIE List B disease that would significantly increase the cost of production on infected piggeries if introduced into Australia.

The policy is to eradicate TGE by the most cost-effective method using one or more of three strategies in infected piggeries:

- ***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; OR**
- ***modified stamping out,*** **which involves quarantine, slaughter with salvage of all saleable exposed pigs at approved abattoirs; OR**
- ***controlled rapid exposure*** **of herds to infection, subsequently allowing immunity to develop and eliminate infection from individual herds.**

These strategies will be supported by:

- ***quarantine and movement controls*** **on animals, animal products and things on infected and suspect premises to prevent spread of infection;**
- ***decontamination*** **of facilities, products and things to eliminate the virus on infected premises and to prevent spread to other properties;**
- ***tracing and surveillance*** **to determine the source and extent of infection and to provide proof of freedom from the disease;**
- ***a public awareness campaign*** **to facilitate cooperation from industry and the community.**

Vaccination is unlikely to be used but may be approved in special circumstances.

The disease is capable of rapid spread within herds and once established within the country will require special industry commitment to achieve eradication.

TGE 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, Part 1, Sections 3 and 4**.

3.2 Strategy for control and eradication

The strategy is to use stamping out sparingly and to attempt to salvage as many animals as possible. Controlled exposure to infected material may be adopted to eradicate infection from large herds with high prevalence of disease. The disease can spread rapidly between farms if quarantine of IP(s) and movement controls are not immediately introduced. Tracing and surveillance will be important to determine the distribution of the diseases and the herd prevalence, so that the best strategy may be selected. If animals are sent to slaughter for salvage, this must be carried out as quickly as possible to reduce the spread of virus and reduce contamination. IPs must also be decontaminated. Any control measures will need to be thoroughly discussed with the industry and individual producers to arrive at strategies that will provide cooperation in their implementation.

3.2.1 Stamping out

Stamping out as a major strategy has no great advantage over a slaughter and salvage strategy. Stamping out will be considered in circumstances where the disease is restricted to a few herds; the herds are small; the disease is contained and unlikely to spread; and stamping out is highly likely to quickly eradicate the disease. The slaughtered animals will be disposed of by the most appropriate means for the particular situation.

Other strategies

Alternative strategies may involve the slaughter out of IPs with immediate slaughter and salvage of saleable animals at an approved abattoir and disposal of non-saleable animals. These are discussed in Sections 2.1.2 and 2.1.3.

3.2.2 Quarantine and movement controls

Quarantine and movement controls will be imposed on IPs, DCPs and SPs as these are identified through tracing and surveillance (see Appendix 1).

An RA and CA will be declared to provide the necessary control to enable eradication measures to be implemented. The RA should include the IPs, DCPs and as many of the SPs as possible and should be as large as is necessary for satisfactory control. The movement of pigs within the RA will be subject to movement restrictions. Most pig movements from the RA will be permitted if direct to slaughter. It is important that animals from the IPs, DCPs and SPs should not be moved off the premises except direct to an abattoir for immediate slaughter. Animals should not be permitted to enter any IP unless they are part of an official eradication program. If movement of pigs from a free

herd is required for breeding, the animals will be subjected to test before a permit is issued for movement.

Live pig sales within the RA will be banned to reduce the possibility of spread and any movements will be subjected to permit. There is no need to impose restrictions on other premises in the RA once they have been cleared from infection and have been cleaned and decontaminated as necessary.

People and vehicles should be subjected to normal restrictions and decontamination and as fomites are implicated in the spread of TGE these items will be controlled.

Dogs and cats must be confined as they have been implicated in transmitting the disease. Wild birds, particularly starlings, have also been implicated as a possible means of transmission and they should be excluded. Foxes may also be infected so prevention of contact with pigs is necessary.

For further details see Appendixes 1 and 2.

Zoning

Zoning may provide some advantages to limit the spread of the disease and to enable better controls for the movements of live animals, products and fomites. There is, however, little advantage for international trade purposes and major disadvantages would be attributed to producers and processes in the domestic market. Major controls can be maintained on the IP to prevent spread beyond these premises.

Zoning is likely to be an advantage only for specific international markets where individual countries may demand certain requirements. The worth of these markets must be balanced against the cost to domestic trade. The same may also apply if individual States impose restrictions.

3.2.3 Treatment of infected animals

Treatment is limited to supportive care, which will tend to reduce mortality rates. Most animals, apart from piglets less than three weeks of age, are likely to recover.

3.2.4 Treatment of animal products and by-products

Animals with clinical signs must not be sent to slaughter but seropositive animals may be sent for immediate slaughter. The animals sent to abattoirs for immediate slaughter must be handled with care to prevent contamination of meat with intestinal contents, and head and neck meat with lymph nodes should be removed and disposed of by rendering. There is no need for other treatment.

Extra policing of swill-feeding regulations is advisable during and after the outbreak.

The TGE virus is readily destroyed by heat and infected carcases and parts can be rendered with safety.

3.2.5 Vaccination

The currently available vaccines are unlikely to be used to protect against infection but may be used to boost immunity in previously-infected animals as part of a specific eradication strategy.

Immunity may also be increased in a herd undergoing TGE eradication by the use of controlled exposure to infective faecal contents (for further details see Appendix 5).

3.2.6 Tracing and surveillance

Tracing from the infected premises must be undertaken on the movement of live pigs, people, vehicles and fomites for at least 30 days prior to the first clinical signs and up to the time that quarantine is imposed. Live pigs are the main source of infection and tracing should concentrate on these.

Surveillance needs to be undertaken on premises that have received any pigs from the IP and on other premises, particularly breeder properties, so that other IPs, DCPs or SPs can be identified.

Where premises have been destocked they will be restocked with sentinel pigs at least 14 days after depopulation and decontamination, and surveillance of these animals will be maintained for a period of 60 days. Sentinel animals will also be used in eradication programs where the premises are not wholly depopulated.

Surveillance will need to be maintained throughout the eradication period and after so that proof of freedom may be supported with reliable scientific information (see Appendix 4).

3.2.7 Decontamination

Premises must be decontaminated following destocking and special decontamination measures must be implemented where eradication is being undertaken while animals are still present on the premises.

Vehicles and people must be decontaminated before leaving IPs or DCPs; vehicles must also be decontaminated after transporting pigs from an IP or DCP.

Bird-proofing and control of insects, particularly flies, must be undertaken to reduce the possibility of spread of virus. Fencing to prevent the entry of dogs, foxes and cats must be effective.

3.2.8 Media and public relations

The veterinary authorities must explain the control measures to the industry and to individuals who are directly affected to gain their confidence in the measures being imposed. The media and public must be informed about the disease and the control arrangements so that buyer confidence in the product is maintained and any effect on the market diminished.

A special publicity campaign should be instituted about the swill-feeding regulations and the potential role that untreated swill has with TGE.

3.3 Social and economic effects

Social and economic effects would be largely restricted to the effect of disease on farm productivity. When newly introduced into a herd, TGE causes significant mortalities in the younger pigs and reduces growth rates in the weaner and grower pigs. There are few published estimates of the costs of a TGE outbreak in the Australian pig industry. Baldock and Webster (1990) presented a preliminary assessment predicting that in the first year following infection, the annual cash surplus of an average 100-sow piggery in Queensland would be less than half that expected in a normal year. These authors did not go on to predict the economic effects in subsequent years once TGE had become endemic.

Though the major effects would be felt in the first year following infection in most herds, the disease is likely to persist in herds with a regular clinical recrudescence. The presence of TGE in a breeding herd will affect the marketability of breeding stock. There should not be any reason why abattoirs would be unwilling to slaughter and process pigs from IPs, however local pressures may disrupt some trade practices.

The presence of this disease in Australia should not affect the current limited export in pork products. However, trade of Australian breeding stock to countries free of TGE is likely to be affected. A decrease in consumption of pork and pork products can be anticipated at least in the short term. A public awareness campaign that this disease does not infect humans, cause disease in domestic pets or affect meat quality would be appropriate.

Where herds are depopulated, either by stamping out or by being sold for slaughter, producers will suffer a prolonged loss of income.

If the eradication by controlled exposure program is implemented, the eradication process will take a minimum of 130 days to complete. This process will necessitate changes in management standards on the IP.

Movement controls will be largely restricted to the IPs and will not cause major disruptions other than by prohibiting live pig sales. Zoning would potentially interrupt the free movement of breeding stock, the movement of pigs to slaughter at preferred markets and the movement of pig meat to markets.

3.4 Criteria for proof of freedom

Declaration of freedom may allow the resumption of trade in live breeding stock to countries that are TGE free.

After an outbreak of TGE, a statistically valid serological survey would have to be undertaken to demonstrate proof of freedom (see Appendix 4). The survey would concentrate on the RA(s) in which disease was present and the high risk herds, based on the results of tracing and pig movements.

3.5 Funding of operations and compensation

As TGE 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 endemic

Individual producers will see significant production benefits from preventing TGE virus from establishing in their herds. The strategy to prevent TGE entering previously uninfected herds is the same as for many other pig diseases. Considerable productivity improvements can be gained by eliminating TGE virus from infected herds. However, the costs of disease eradication programs such as depopulation/repopulation or eradication by controlled exposure are high. Availability of clean replacement stock and risks of reinfection need to be incorporated into a benefit–cost analysis before embarking on such programs.

The objective is for eradication of the disease. The disease has never been eradicated from a country where it has become established but if tracing and surveillance can identify the infected herds the disease could be eradicated if the industry has the will to apply strict movement controls and good hygiene and management practices.

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 TGE exists or is believed to exist. An IP is subject to quarantine served by notice and to eradication or control procedures.

Dangerous contact premises (DCP)

Premises classified as DCPs will be those containing contact animals, being animals that either have been in contact with or are animals sourced from an IP. This includes animals sourced from an IP up to 30 days prior to the outbreak.

Suspect premises (SP)

Premises classified as suspect premises will be those within 2 km from an IP

Restricted area (RA)

The RA should include all the IPs, DCPs and as many of the SPs as possible and should be as large as is necessary for satisfactory control, based on epidemiological evidence, geographical features, and other factors. The movement of all pigs within the RA will be subject to restrictions.

Control area (CA)

A CA may be declared around the RA with less intensive restrictions applying to those in the RA. To meet OIE generic requirements a total of 10 km radius should be considered.

APPENDIX 2 Recommended quarantine and movement controls

Infected and suspect premises

Movement out of susceptible animals:
Approved under permit direct to immediate slaughter within 4 hours of arrival at an approved abattoir. Routes to be specified. See Notes (below).

Movement in of susceptible animals:
Restrictions apply, depending on the selected strategy for control.

Movement out of specified products:
Dead pigs permitted off the premises for rendering or burial only.

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

Movement in and out of people:
Movement should be restricted to essential visitors only. 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:
No restrictions.

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 meters of the road.
(4) Multiple consignments per truck will be prohibited unless by special approval from the local disease control centre (LDCC) controller, subject to:
    - the IP or DCP being the last pick-up;
    - the whole consignment being for immediate slaughter (within 4 hours of arrival);
    - the 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.

APPENDIX 3 OIE International Animal Health Code for transmissible gastroenteritis

[NB The following text is taken directly from the OIE International Animal Health Code (1992); Chapter 3.5.5. 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 TGE in 1993, 1994 or 1995.]

Preamble: For diagnostic tests, reference should be made to the *Manual* (B53) [☞see OIE publications under References].

Article 3.5.5.1.

For the purposes of this *Code,* the *infective period* for transmissible gastroenteritis (TGE) shall be considered to be 40 days.

Article 3.5.5.2.

Veterinary Administrations of *importing countries* should require:

for pigs for breeding or rearing

the presentation of an *international animal health certificate* attesting that the animals:

1) showed no clinical signs of TGE on the day of shipment;

AND EITHER

2) come from an *establishment* free of clinical signs of TGE during the 12 months prior to shipment; and

3) showed negative results to a diagnostic test for TGE during the 30 days prior to shipment and were kept isolated during this period;

OR

4) come from a country in which TGE is officially notifiable and no clinical *case* has been recorded in the previous three years.

Article 3.5.5.3.

Veterinary Administrations of *importing countries* should require:

for pigs for slaughter

the presentation of an *international animal health certificate* attesting that the animals:

1) showed no clinical signs of TGE on the day of shipment;
2) come from an *establishment* in which no *case* of TGE was officially reported during the 40 days prior to shipment.

Article 3.5.5.4.

Veterinary Administrations of *importing countries* should require:

for *semen*

the presentation of an *international animal health certificate* attesting that:

1) the donor showed no clinical signs of TGE on the day of collection;

AND EITHER

2) the donor animals have been resident for at least 40 days on an *AI centre*, and all the pigs on this AI centre were free of clinical signs of TGE during the 12 months prior to collection;

And

3) for fresh semen, the donor animals showed negative responses to a diagnostic test during the 30 days prior to collection;

4) for frozen semen, the donor animals showed negative responses to a diagnostic test at least 14 days after collection;

OR

5) the donor animals have been resident since birth in a country in which TGE is officially notifiable and no clinical case has been recorded in the previous three years;

and in all situations:

6) the semen was collected, processed and stored strictly in accordance with Appendix 4.2.2.1.

★★★★★★★

APPENDIX 4 Procedures for surveillance and proof of freedom

Proof of freedom

In a herd with no history of infection, serological evidence of freedom would be sufficient. Testing should be at a level to detect a 1% prevalence with 95% confidence.

In a previously-infected herd containing seropositive animals, sentinel animals must be placed in the farrowing, weaner and grower accommodation and monitored for seroconversion to TGE virus over 60 days. Sentinel animals (ideally 20–40 weaner pigs) must be seronegative.

Proof of freedom would rely on serological evidence of freedom, resulting from a valid national survey.

Surveillance

On farrow-to-finish units in continual production, the presence or absence of clinical signs of the disease would need to be ascertained. As confirmation, the level of neonatal and preweaning mortalities from diarrhoea must be determined and serum samples submitted for testing.

On units with fattener pigs only, the presence or absence of clinical signs must be determined. Serological testing would be the only way of confirming freedom.

APPENDIX 5 Eradication by controlled exposure

This program was developed for the elimination of TGE from breeder herds in the United States (Harris et al 1987, Wiseman et al 1988, Fitzgerald and Welter 1990). The exact protocol to be adopted would be dependent on the facilities and management level on the infected premises. This protocol would be developed by veterinarians skilled in swine disease management but would include steps 1–7 as follows.

1) Day 1 — diagnosis of TGE; pig movements off the IP restricted to direct slaughter only.

2) Day 1–21 (until the cessation of clinical signs) — introduction of all breeding stock replacements necessary for a 4–6 month period. This should include animals of differing weight ranges. No further additions to the herd until sentinel pigs are brought in (step 5).

   Exposure of entire herd (including replacements) to intestines and intestinal contents from dead or moribund pigs affected with TGE. Feedback could include an attenuated oral vaccine. Feedback should begin with sows in late gestation and continue backwards to the sows and boars in the breeding area. Continue feedback until clinical signs are observed in all pigs. See below for further information on the source of the virus and manner of collection and administration.

3) After clinical signs have subsided, begin strict all-in, all-out movement of stock for farrowing and weaner rooms; clean, disinfect (and if possible fumigate) rooms between groups. Continue to monitor for clinical signs of diarrhoea; use laboratory facilities to differentiate aetiology.

4) Thirty days after cessation of clinical signs of TGE, place approximately 20–40 sentinel pigs from a herd known to be free of TGE in weaner, grower, breeding and gestation buildings.

5) Observe the sentinel pigs for clinical signs of TGE daily over the 60-day sentinel period. If diarrhoea occurs kill and necropsy acutely affected pigs and submit tissues to a diagnostic laboratory.

6) Collect blood from sentinel pigs on 3 occasions: immediately prior or upon entry into the herd; 30 days; and 60 days after entry to the herd. Assay sera for antibodies to TGE; negative serum neutralisation test results, titre of 1:2 or less, indicate that TGE virus has been eliminated.

7) If step 6 shows that sentinel pigs are unaffected, quarantine may be removed.

It should be recognised this technique has been successfully implemented in herds in the United States with highly competent management. The technique must be critically evaluated as to its applicability in the context of a herd in Australia with less competent management and without all-in-all-out facilities.

Specific issues to be addressed before this technique is adopted are:

- responsibility for day-to-day management during the program may involve input from a skilled manager (possibly a pig industry livestock officer);
- compensation arrangements; and
- availability of infective material — delay in diagnosis may mean a lack of infective material.

Source of virus for feedback

Virus held in laboratories in cell culture attenuates rapidly with loss of virulence and is unsuitable as a source in a feedback program.

Wiseman et al (1988) described a technique to ensure that sufficient infective material is harvested to allow infection of the entire herd. These techniques were developed for an eradication program in a 330 sow herd with endemic infection, where clinical disease was not dramatic and only small amounts of infective material were available at any one time.

1) Infective samples of intestines/intestinal contents were collected from within the herd and frozen.
2) Ten sows at 110 days gestation were introduced from a seronegative herd.
3) When the first of these farrowed, the frozen material saved from previous clinical episode was used to infect 3 baby pigs at 12–24 hours old.
4) As these pigs became clinically ill, they were sacrificed. Intestinal tracts and lungs were collected and homogenised in cold saline. This homogenate was used to infect all pigs born to the 10 TGE seronegative sows by 72 hours of age.
5) Ill infected pigs were sacrificed when clinical signs appeared. Lungs and intestinal tracts were collected again and homogenised with cold saline at a ratio of 1:1.
6) This cocktail was administered orally as a 5–10 mL dose to the entire breeding herd, all replacement stock and all pigs from the farrowing house through to the weaner room. Sows and boars were restrained by snaring.
7) As any of the animals in the herd broke with scours, the scour material was fed back to the rest of the herd. Lungs and intestines were also fed back from any additional suckling pigs dying with clinical signs.

GLOSSARY

All-in-all-out production	A method of production in which all stock leave the premises (or area) followed by total restocking.
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 emergency-management 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 (*see* Appendix 1).
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 (*see* Appendix 1).
Decontamination	Includes all stages of cleaning and disinfection.
Enterocytes	Cells lining the small intestine responsible for the final digestion and absorption of nutrients and water.
Feedback	The deliberate feeding of infective material to susceptible animals, which is usually sourced from within the same farm.
Fomites	Inanimate objects (eg boots, clothing, equipment, vehicles, crates, packagings) that can carry the exotic agent and spread the disease through mechanical transmission.
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 property	The property on which the first or original case (index case) in a disease outbreak is identified to have occurred.
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.

Immunoglobulin	Antibody proteins
– IgA	Humoral antibody mainly secreted from mucosal surfaces.
– IgG	The main form of immunoglobulin produced in response to an antigen. It is mainly found in body fluids.
Infected premises	*see* 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.
Parenteral	Administration of a drug/vaccine by a route other than the digestive tract (eg by injection).
Polymerase chain reaction	A method of amplifying and analysing DNA sequences that can be used to detect the presence of virus DNA.
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	Processing by heat to inactivate infective agents. Rendered material may be used in various products according to 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 (*see* Appendix 1).
Risk enterprise	A livestock or livestock-related enterprise with a high potential for disease spread, eg an abattoir, 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.
Serosurveillance	Surveillance of an animal population by testing serum samples for the presence of antibodies to disease agents.
Serotype	A subgroup of a genus of microorganisms identifiable by the antigens carried by the members.

Serum neutralisation	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.
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.
Surveillance	A systematic program of inspection and examination of animals or things to determine the presence of absence of an exotic disease.
Susceptible species	Animals that can be infected with the disease (for TGE — 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 (*see* Appendix 1).
Swill	Food scraps of placental mammal origin that have not been obtained under licence 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 be taken.
Vaccine – attenuated	A vaccine prepared from infective or 'live' microbes that have lost their virulence but have retained their ability to induce protective immunity.
Vector	A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. A *biological* vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A *mechanical* vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.
Viraemia	The presence of viruses in the blood.
Zoning	Dividing a country into defined infected and disease-free zones. A high level of movement control between zones will apply.

Abbreviations

AAHL	Australian Animal Health Laboratory, Geelong
AI	Artificial insemination
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zeal and
CA	Control area
CCEAD	Consultative Committee on Exotic Animal Diseases
CVO	Chief veterinary officer
Ig	Immunoglobulin
IP	Infected premises
LDCC	Local disease control centre
OIE	World Organisation for Animal Health [Office International des Epizooties]
RA	Restricted area
SP	Suspect premises
TGE	Transmissible gastroenteritis

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Training resources

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

Exotic diseases of pigs (56 slides), available from the Foreign Diseases Unit, 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.

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