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Importation of Sausage Casings into Australia

Import Risk Analysis

DECEMBER 1999

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AUSTRALIA

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1. Background.

1.1. What are casings?

Sausage casings may be natural or artificial. Natural Casings are derived from the fibrous, connective tissue layer of the intestinal tract of animals (Andriessen, 1987). They are usually preserved by natural means, including salting, for storage and transport. Most parts of the digestive tract may be used as casings for sausage. In this paper, natural casings from ruminants (cattle and sheep) and porcines will be considered.

Artificial casings may be prepared from collagen, which is largely derived from the corium layer of cattle hides. These are referred to as edible collagen casings. The collagen-rich corium undergoes extensive treatment with heat, acids and ammonia during the manufacturing process. Natural casings are currently not permitted entry to Australia other than from approved countries. Edible collagen casings (ie artificial casings) may currently be imported from all countries subject to the requirement to obtain a permit based on an individual assessment of the production process.

Other types of artificial casings which are prepared from specially impregnated papers etc are not considered to represent a risk of introduction of exotic animal disease. Provided they do not contain any animal or biological material, they may be imported from all countries subject to inspection on arrival.

1.2. Preparation of casings

Intestines of the various species used for preparations of casings are collected from the slaughter floor of the meatworks after evisceration and inspection of the carcasses. The runners (intestines) are taken into the runner-room where they are separated from the mesentery and put through a stripping machine to remove intestinal contents. Next, runners are passed through a crusher, which breaks up mucosal, serosal and muscular layers and expels this macerated debris. The runners then go through a de-threader which removes any residual material and leaves the casings cleaned to the muscularis and serosal layers. The casings are then stored in cold brine solution for 24 hours or more. After this the casings are graded for size, length and colour and quality. Casings are then made up in hanks, salted down and packed in barrels or containers ready for shipment. The brine solution used for storage of sausage casings contains approximately 26.4% sodium chloride and has a pH range of 6.7 to 7.3. In some food processing procedures, the casings can be stored in crystalline salt or in saturated salt solutions. Before use the casings are washed and may be soaked in solutions of hydrogen peroxide, sodium peroxide, tartaric or lactic acid. These treatments reduce bacterial plate counts and effect a complete kill of *Salmonella* spp.

The importation of sausage casings is therefore unlikely to present a risk of introduction of any exotic disease agent that is inactivated by a high salt environment.

1.3. Size of market

In 1988, Australia imported a total of 914,296 units of sausage casings with a total value of AU$8,341,000. In the same year, imports of sausage casings totaled 96,385 units with a value of AU$729,000. By contrast, in 1991, the total imports was only 365,762 units, with a value of AU$3,904,000 and 261,029 units with a value of AU$1,418,000.

Information available as at 17 April 1998 indicates that there were, at that time, only 5 current permits to import sausage casings. All the current permits were issued to the same company, and three of these permits covered samples for in vitro evaluation only at quarantine approved premises. The import permits for evaluation purposes were for casings from countries that have not previously been approved as a source of casings for import to Australia. This indicates that there is some interest in expanding the list of countries from which the product may be sourced.

1.4. Source of imported product

Currently, imports of casings are restricted to those sourced from Canada, New Zealand, Northern Ireland, Republic of Ireland, and the United States of America. A previous report (Parsonson 1992) indicates that

> "In 1991, there were importations of small quantities of sausage casings in bundles into Western Australia from Italy, and into NSW from Germany. Both these countries have endemic pathogenic agents which are exotic to Australia."

These imports were included in data obtained from the Australian Bureau of Statistics, and raise the possibility that casings may have been imported from countries which were not approved, or that there has been incorrect recording of details. It is also possible that these imports were for product evaluation purposes only.

Also of concern are persistent allegations concerning the origins and certification of some batches of casings over the last few years. It has been alleged that there has been collusion between importers and exporters to import into Australia casings from ineligible countries with false documentation. Any review of conditions for importation of casings should take into account these allegations, and attempt to find a solution which will remove the temptation to smuggle casings into Australia by the use of false certification.

1.5. Existing controls

1.5.1. Legislation

Currently, import of casings is subject to Quarantine Proclamation 1998. The relevant section of this Proclamation states:

"39 Importation of meat and meat products

(1) In this section:

meat means a part of an animal (other than a fish, a mollusc, a crustacean, a cnidarian, an echinoderm or a tunicate) that is intended or able to be used as food by a human being or an animal (whether or not cooked, dried or otherwise processed), and includes:

(a) blood; and

(b) bone meal, meat meal, tallow and fat.

meat product means a product that contains meat, or of which meat is an ingredient.

(2) The importation into Australia of meat or a meat product is prohibited, other than meat or a meat product that is mentioned in table 13 and complies with any condition stated in that item for the meat or meat product.

(3) However, subsection (2) is not taken to prohibit the importation by a person of meat or a meat product if a Director of Quarantine has granted the person a permit to import the meat or meat product into Australia.

Table 13 Dead animals and animal parts (relevant sections)

Meat and meat products

30	Meat products, if canned, containing less than 5% by weight of meat, and not requiring refrigeration to maintain quality
31	Meat products, if canned, not containing pork meat, not requiring refrigeration to maintain quality, and if for the personal consumption of the person wishing to import the product
32	Meat or meat products, other than pork or avian meat, if declared to be of New Zealand origin and: (a) clearly labelled with the date of processing; and (b) clearly labelled with the name and address of the processing premises; and (c) the outermost wrapping of the largest packaged unit is labelled 'Product of New Zealand' *Note* If the container is a full sealed shipping container, it is not necessary for each individual package to carry the 'Product of New Zealand' label.
33	Meat or meat products, other than pork or avian meat, if clearly labelled as a product of New Zealand, and if for the personal consumption of the person wishing to import the article

Matters which the Director must consider when deciding whether to grant such a permit are as follows:

> **"70 Things a Director of Quarantine must take into account when deciding whether to grant a permit for importation into Australia**
>
> 1) In deciding whether to grant a permit to import a thing into Australia, a Director of Quarantine:
>
> (a) must consider the quarantine risk if the permit were granted; and
>
> (b) must consider whether, if the permit were granted, the imposition of conditions on it would be necessary, to limit the quarantine risk to a level that would be acceptably low; and
>
> (c) may take into account anything else that he or she knows that is relevant.
>
> (2) In this section:
>
> ***quarantine risk*** means:
>
> (a) the likelihood that, if the permit is granted, the importation will lead to the introduction, establishment or spread of a disease or a pest in Australia; and
>
> (b) the likelihood that any such introduction, establishment or spread of a disease or pest will result in harm being caused to human beings, animals, plants, other aspects of the environment or economic activities as a result of the introduction, establishment or spread of the disease or pest; and
>
> (c) the likely extent of any such harm."

Until the entry into force of Quarantine Proclamation 1998, the importation of casings was subject to the requirements of Quarantine Proclamation 134A (dated 21 December 1987). Under Proclamation 134A, the Director of Quarantine was required, by instrument, to certify that certain requirements had been met, and grant a permit to import. Schedule 2 to Proclamation 134A required that (in the case of porcine casings) the country of origin be free of foot and mouth disease, rinderpest, African swine fever, classical swine fever and swine vesicular disease for a period of 6 months prior to the slaughter of the animals from which the casings were derived. In the case of casings derived from ruminants, Schedule 2 to Proclamation 134A required that the country of origin must be free of foot and mouth disease and rinderpest, for a period of 6 months prior to the slaughter of the animals from which the casings were derived.

1.5.2. Guidelines for the Importation of Uncanned Meat

Guidelines for the importation of uncanned meat, issued on 10 October 1989, on behalf of the Director of Animal and Plant Quarantine, state that the country of origin of uncanned pig meat

must be have been free of Aujeszky's disease, in addition to foot and mouth disease, rinderpest, African swine fever, classical swine fever and swine vesicular disease, for a period of six months prior to the slaughter of the animals for preparation of casings for export to Australia. The requirement for freedom from Aujeszky's disease, which is contained in these guidelines, is additional to the requirements of the legislation as listed above.

1.5.3. Quarantine Operations Manual

The current entry for animal casings in the Quarantine Operations Manual (dated 2 August 1996) limits the entry of casings to those from Canada, New Zealand, Northern Ireland, Republic of Ireland, and the United States of America. Casings originating in the USA may also be imported into Australia after calibration in New Zealand.

There are provisions for the transshipment of casings from Ireland through ports in the UK, or through New York, subject to certification that the casings were only transshipped, and not otherwise handled. There are also provisions for the re-importation of Australian casings subject to their not having passed out of Customs control in the overseas port, and the casks of casings not having been opened for any reason.

There have been requests for approval to ship Australian casings overseas for further processing and subsequent re-importation. To date, this has not been permitted. Re-importation of Australian casings is only permitted where the casings have not left Customs control in the overseas country and the containers have not been opened for any purpose.

2. Exposure pathway

2.1. Criteria for agent entry and disease establishment in Australia

MacDiarmid (1991) has defined criteria that must be met before meat or meat products can serve as a vehicle for the introduction of animal disease into an importing country. The following criteria are closely based on those provided by MacDiarmid, modified to suit the particular case of the importation of natural sausage casings into Australia:

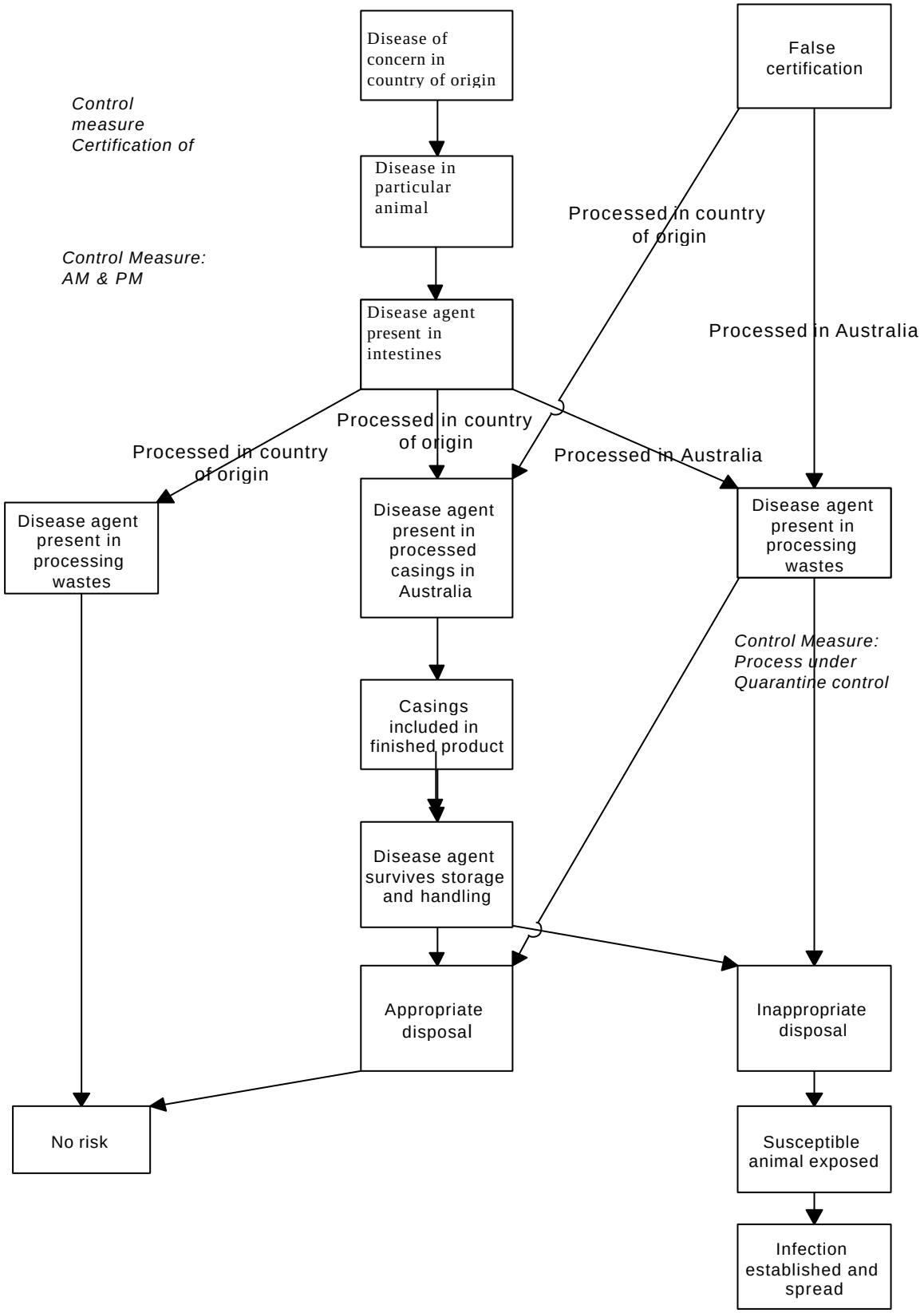
1. the disease must be present in the country of origin;
2. the disease must be present in the particular animal slaughtered;
3. the pathogen must be present in the intestine;
4. the infected meat must pass inspection procedures;
5. the pathogen in the meat must survive storage and processing and be present in sufficient numbers to provide an infectious dose in the quantities that may be eaten by Australian animals;
6. the pathogen must be able to establish an infection by the oral route;

7. scraps of the product must find their way into a susceptible animal of the appropriate species after importation into Australia; and
8. should the pathogen establish infection in a susceptible host in Australia, local conditions must be such that the disease could spread and cause an outbreak.

It is also possible that the casings may become contaminated during post-processing handling or storage, in which case points 5-8 above, would apply.

Figure 1 provides a diagrammatic representation of the pathways by which a disease agent of quarantine concern could be introduced into Australia through the importation of animal casings.

Figure 1: Exposure pathway



3. Hazard Identification

The disease agents of concern include those OIE List A, List B and FAO List C diseases of ruminants and pigs which are exotic to Australia, which are likely to be found in intestines of cattle, sheep or pigs, and which could be considered likely to be transmitted through meat and meat products. In addition, diseases not included on OIE/FAO Lists, but which may have a significant effect on susceptible Australian animal populations are also considered.

3.1. Level of risk

In order to determine the level of risk relating to each of the diseases of concern, it is necessary to consider a number of factors relating to the country of origin, the disease and the product involved. Some of these factors are listed below.

3.1.1. Country factors

1. Has the product been sourced from the country of export or from another country(s)?
2. Is the disease absent, well controlled or endemic in the country/zone of origin?
3. Can vaccinated animals excrete the organism?
4. Can the competent authority effectively monitor and control disease in livestock?
5. Can the country(s) be regionalised with regard to the disease organism in question?
6. Does the competent authority control health standards in food production?
7. Is certification from the country of origin reliable?

From the above list of factors, it can be seen that there was little apparent risk inherent in allowing the import of casings from those countries which were previously approved (ie Canada, Northern Ireland, the Republic of Ireland, United States of America, and New Zealand). However, a possible additional risk factor is introduced by maintaining these restrictions on imports. This increased risk stems from the temptation to smuggle casings from cheaper sources into Australia using false certification. Smuggling constitutes an increased risk due to lack of control of the product involved, and problems with product are more likely to be concealed, leading to a possible increase in time before detection of an outbreak of potential exotic disease.

A system which would allow controlled import of casings from other countries, while still guaranteeing appropriate levels of confidence that exotic diseases would not be introduced, would be preferable.

3.1.2. Product factors

1. Is the pathogen of concern likely to be present in intestines of the appropriate species of animals (ruminants and pigs)?
2. Has the product been processed in a manner which would destroy the pathogens?
3. Is post processing contamination with animal pathogens possible?
4. Is it possible to test the product for pathogens of concern?
5. Has packaging/storage of product prevented post-processing contamination?
6. Is the small amount of casings in sausages considered safe? What percentage of a sausage is in the casing? How much casing (and therefore sausage) must be eaten to cause disease?

3.1.3. Post import factors

1. Can the product be treated before release from quarantine?
2. Is the processing of casings in Australia likely to result in contamination of the environment (eg by contaminated wastewater from processing plants)?
3. What level of confidence could be guaranteed by testing, and is that level tolerable?
4. Do Australian Food Standards prohibit/permit the treatment of choice?
5. Can adequate controls be exercised over the end use of the product?
6. How likely are susceptible animals to be exposed to the imported product?
7. How easily would the organism become established?
8. How difficult would it be to eradicate the organism?
9. Would the infection of an animal(s) in Australia have serious economic or environmental effects?

3.2. OIE List A diseases

The OIE List A diseases are defined as those "Communicable diseases which have the potential for very serious and rapid spread, irrespective of national borders, which are of serious socio-economic or public health consequence and which are of major importance in the international trade of livestock products." According to this definition, the consequence of the establishment of a List A disease in a previously free country can be considered to be extreme. List A diseases affecting ruminants and pigs are shown in Table 1.

Table 1: OIE List A diseases of ruminants and pigs, which could potentially be introduced in sausage casings, and their status in Australia.

Disease	Species	Methods of Spread	Australian status	Quarantine risk
Foot and Mouth Disease	Ruminants/ Pigs	Contact, ingestion	EXOTIC	Yes
Vesicular stomatitis	Cattle/pigs	Presumably mainly insect vector	EXOTIC	No
Swine Vesicular Disease	Pigs	Contact, ingestion	EXOTIC	Yes
Rinderpest	Cattle	Contact, ingestion	EXOTIC	Yes
Peste des petits ruminants	Sheep/ goats	Close contact, ingestion?	EXOTIC	Yes
Contagious bovine pleuropneumonia	Cattle	Contact only	EXOTIC	No
Lumpy skin disease	Cattle	Mechanical, insects	EXOTIC	No
Rift Valley Fever	Cattle / Sheep	Arthropod borne	EXOTIC	No
Bluetongue	Sheep, cattle	Arthropod borne	Present - No clinical disease	No
Sheep pox and goat pox	Sheep, goats	Mechanical, insects	EXOTIC	No
African Swine fever	Pigs	Contact, ingestion	EXOTIC	Yes
Hog cholera	Pigs	Contact, ingestion	EXOTIC	Yes

From the above table, it can be seen that vesicular stomatitis, contagious bovine pleuropneumonia, lumpy skin disease, Rift Valley fever, bluetongue and sheep pox/goat pox are unable to be transmitted via meat or meat products. No specific quarantine requirements are necessary for these diseases and they will not be considered further in this paper.

3.3. OIE List B diseases

The OIE List B diseases are defined as those "Communicable diseases which are considered to be of socio-economic and/or public health importance within countries and which are significant in the international trade of livestock products." According to this definition, the consequence of the establishment of a List B disease in a previously free country can be considered to be very high. List B diseases affecting ruminants and pigs are shown in Table 2.

Those diseases listed in Table 2 as being unable to be transmitted via meat or meat products, and/or as being present in Australia, do not present a quarantine risk. No specific quarantine requirements are necessary for these diseases and they will not be considered further in this paper.

Table 2: OIE List B diseases of ruminants and pigs, which could potentially be introduced in sausage casings, and their status in Australia.

Disease	Species	Methods of Spread	Australian status	Quarantine risk
Anthrax	Cattle, pigs, sheep, goats	Ingestion of spores, insects	PRESENT	No
Aujeszky's Disease	Pigs	Ingestion, inhalation	EXOTIC	Yes
Echinococcosis/ hydatidosis	Sheep	Ingestion of infective stage	PRESENT	No
Heartwater	Cattle, sheep, goats	Tick vector	EXOTIC	No
Leptospirosis	Cattle, sheep, pigs, goats	Ingestion	PRESENT	No
Q fever	Goats, sheep	Tick vector, aerosols	PRESENT	No
Rabies	Mammals	Wound contamination	EXOTIC	No
Paratuberculosis	Cattle, sheep	Contact, ingestion of infected pasture	PRESENT	No
Screw worm fly	Mammals	Fly	EXOTIC	No
Anaplasmosis	Cattle	Arthropod vector	PRESENT	No
Babesiosis	Cattle	Arthropod vector	PRESENT	No
Bovine brucellosis	Cattle	Direct contact	EXOTIC	No
Bovine genital campylobacteriosis	Cattle	Venereal	PRESENT	No
Bovine tuberculosis	Cattle	Inhalation, ingestion of contaminated food	EXOTIC	No
Cysticercosis	Cattle	Ingestion of infective stage	PRESENT	No
Dermatophilosis	Cattle	Direct via skin abrasions	PRESENT	No
Enzootic bovine leukosis	Cattle	Contact, mechanical transmission	PRESENT	No
Haemorrhagic septicaemia	Cattle	Contact, pre-disposing stress	Exotic	No
Infectious bovine rhinotracheitis	Cattle	Droplet infection	PRESENT	No
Theileriosis	Cattle	Arthropod vector		No
Trichomoniasis	Cattle	Venereal	PRESENT	No
Trypanosomiasis	Cattle	Arthropod vector		No
Bovine malignant catarrh	Cattle	Insect vector?	PRESENT	No
Scrapie/ Bovine Spongiform encephalopathy	Cattle/sheep	Ingestion?	EXOTIC ZOONOTIC	Yes
Ovine epidiymitis (*B ovis*)	Sheep	Venereal	PRESENT	No
B melitensis	Sheep/goats	Direct contact with infected material	EXOTIC	No
Caprine arthritis/	Goats	Ingestion of infected milk	PRESENT	No

Disease	Species	Methods of Spread	Australian status	Quarantine risk
encephalitis				
Contagious agalactia	Sheep/goats		EXOTIC	No
Contagious caprine pleuropneumonia	Goats	Respiratory	EXOTIC	No
Enzootic abortion of ewes	Sheep		PRESENT	No
Pulmonary adenomatosis	Sheep	Direct contact	EXOTIC	No
Nairobi sheep disease	Sheep	Arthropod vector	EXOTIC	No
Salmonella abortus ovis			EXOTIC	No
Maedi visna	Sheep	Direct contact/ inhalation	EXOTIC	No
Atrophic rhinitis	Pigs	Aerosols	PRESENT	No
Cysticercosis (*C cellulosae*)	Pigs	Ingestion of cysts in muscle	EXOTIC	No
Transmissible gastroenteritis	Pigs	Ingestion	EXOTIC	Yes
Trichinellosis	Pigs	Ingestion	EXOTIC	Yes
Enterovirus encephalomyelitis (Teschen/Talfan Disease)	Pigs	Ingestion	TALFAN PRESENT TESCHEN EXOTIC	Yes
Porcine reproductive and respiratory syndrome	Pigs	Uncertain minimum oral infectious dose	EXOTIC	Yes

3.4. FAO List C diseases

Diseases affecting ruminants and pigs which are included in the FAO List C are shown in Table 3. The diseases on the FAO List C are all either present in Australia and/or are not likely to be transmitted by meat or meat products. Warble infestation is exotic to Australia but may be discounted as it is only spread by direct contact with the warble fly. Therefore no specific quarantine requirements are necessary for these diseases.

Table 3: FAO List C diseases of ruminants and pigs, which could potentially be introduced in sausage casings, and their status in Australia.

Disease	Species	Australian status	Quarantine Risk
Listeriosis	Multi-species	PRESENT	No
Toxoplasmosis	Multi-species	PRESENT	No
Melioidosis	Multi-species	PRESENT	No
Blackleg	Multi-species	PRESENT	No
Botulism	Multi-species	PRESENT	No
Other clostridia	Multi-species	PRESENT	No
Other pasteurelloses	Multi-species	PRESENT	No
Actinomycoses	Multi-species	PRESENT	No
Intestinal salmonella infections	Multi-species	PRESENT	No

Disease	Species	Australian status	Quarantine Risk
Coccidiosis	Multi-species	PRESENT	No
Distomatosis (Liver fluke)	Multi-species	PRESENT	No
Filariasis	Multi-species	PRESENT	No
Mucosal disease/Bovine viral diarrhoea	Cattle	PRESENT	No
Vibrionic dysentery	Cattle	PRESENT	No
Warble infestation	Cattle	EXOTIC	No
Contagious pustular dermatitis	Sheep/goats	PRESENT	No
Foot rot	Sheep/goats	PRESENT	No
Contagious opthalmia	Sheep/goats	PRESENT	No
Enterotoxaemia	Sheep/goats	PRESENT	No
Caseous lymphadenitis	Sheep/goats	PRESENT	No
Sheep mange	Sheep/goats	PRESENT	No
Swine erysipelas	Pigs	PRESENT	No

3.5. Other potentially serious exotic diseases.

Geering *et al* (1995) in their publication provide a review of a number of exotic diseases of importance to Australia. In addition, there has been concern expressed by the pig industry in relation to the relatively recently recognised disease, postweaning multi-systemic wasting syndrome (PMWS). These exotic diseases of potential concern are listed in Table 4. Of these, only vesicular exanthema, porcine epidemic diarrhoea and PMWS appear likely to be transmitted in meat or meat products.

Table 4: Other serious exotic diseases.

Disease	Species	Methods of Spread	Australian status	Quarantine Risk
Borna disease	Cattle, sheep	Unknown, probably direct contact	EXOTIC	No
East Coast Fever	Cattle	Tick borne	EXOTIC	No
Jembrana disease	Cattle	Mechanical transmission of blood	EXOTIC	No
Louping ill	Sheep, cattle, pigs	Tick borne	EXOTIC	No
Sheep scab	Sheep	Mite	EXOTIC	No
Wesselbron disease	Sheep, goats	Mosquito	EXOTIC	No
Chaga's disease	Pigs	Insect vector	EXOTIC	No
Japanese encephalitis	Pigs	Insect vector	EXOTIC	No
Porcine epidemic diarrhoea	Pigs	Faecal-oral route	EXOTIC	Yes
Swine influenza	Pigs	Close direct contact	EXOTIC	No
Vesicular exanthema.	Pigs	Ingestion	EXOTIC	Yes
PMWS	Pigs	Uncertain	EXOTIC	Yes

4. Disease risk assessment

4.1. OIE List A diseases

4.1.1. Foot and Mouth Disease

4.1.1.1. Description and aetiology

Foot and Mouth Disease (FMD) is an acute picornavirus disease which affects a wide variety of domestic and wild cloven-hoofed animals, including cattle, sheep, goats, and swine. It is perhaps the most contagious disease of domestic animals, and represents the greatest threat to Australia's animal health status. FMD is not very lethal to adult cattle, but causes serious production loss, and is a major constraint to international trade in livestock and livestock products (Geering *et al* 1995).

4.1.1.2. World distribution

Foot and Mouth Disease has been virtually eradicated from Europe, with outbreaks during the 1990s limited to Italy, Bulgaria, Russia, and Greece. FMD is endemic at high prevalence throughout most of Africa and the Middle East. The disease is endemic in central Asia, the Indian sub-continent, and much of South-East Asia. Sporadic outbreaks occur in peninsular Malaysia, and in the Philippines, the disease is confined to Luzon. Indonesia has been free since 1983. Japan, the Republic of Korea, Taiwan and Singapore have been free for a long time. FMD is endemic in several South American countries, but Argentina, Chile, Guyana, Surinam, French Guiana and Uruguay are free. The Pacific nations, North and Central America and the Caribbean are free. The disease has occurred in Australia, with minor outbreaks in 1801, 1804, 1871 and 1872. It has not occurred in Australia since 1872 (Geering *et al* 1995) .

4.1.1.3. Pathology

The respiratory system is the major route of infection for ruminants. This is also the most common route of infection for pigs, but pigs are much more susceptible to oral infection than are ruminants. After infection, virus is transmitted to the pharyngeal area, local lymph nodes and thence via the bloodstream to the glandular organs, other lymph nodes, epithelial tissues of mouth and feet, and the mammary glands (Geering *et al* 1995).

Virus is excreted in large amounts in expired air, and in all secretions and excretions, as well as from ruptured vesicles.

Excretion of FMD virus can begin up to four days before the clinical disease becomes apparent. After clinical recovery, up to 80% of ruminants may become persistently infected carriers. The persistent infection can establish in the pharyngeal and cranial oesophageal tissues. Maximum reported carrier periods are three years for cattle, nine months for sheep, four months for goats, and five years or more for African buffalo. Vaccinated animals may also become infected and excrete virus for about a week, although they are protected from clinical disease (Geering *et al* 1995). This means that there is a possibility of infected animals

being slaughtered for human consumption, and consequently, of casings being collected from them.

4.1.1.4. Pathogen inactivation

The virus is stable between pH 7 and 9, but inactivation rapidly occurs outside this range. Although the virus is rapidly inactivated in skeletal and heart muscle due to the drop in pH which usually accompanies *rigor mortis*, the virus may persist for long periods in the viscera of infected animals (Blackwell 1984). This persistence is due to the fact that the virus is protected from *post mortem* pH changes (Blaha 1989). Treatment of infected intestines by washing in 0.5% citric or lactic acid for 5 minutes will remove FMD virus (Bohm).

Salting of casings is not sufficient to inactivate FMD Virus (Cottral, 1969). Virus has been isolated from processed casings for up to 240 days (Heidelbaugh, 1968).

4.1.1.5. Disease transmission

Historically, many outbreaks of FMD have been attributed to the feeding of food scraps to pigs (Fenner *et al* 1987). Although swill feeding is no longer practised in Australian commercial piggeries, the possibility that pigs may gain access to food scraps cannot be discounted. Pigs are considered to be important amplifying hosts because of their ability to be infected orally and their ability to excrete large quantities of virus in exhaled air (Geering *et al* 1995).

4.1.1.6. Establishment requirements

In order for FMD to become established in Australia as a result of the importation of infected meat and meat products, the infected material must be fed to pigs (MacDiarmid and Thompson 1997). Although swill feeding of pigs is no longer practised widely, it is possible that pigs could gain access to the material. In addition, it is necessary that the infected material contain a sufficiently high dose of virus to cause infection. Since neither the post-mortem pH changes in casings, nor the normal preservation of casings in salt, are likely to be sufficient to inactivate FMD virus to any great extent, casings from an infected animal could potentially contain sufficient virus to cause infection.

FMD would undoubtedly have the potential to become established in Australian cattle, pig, sheep and goat populations and to spread rapidly over the continent unless diagnosed very early. Long distance windborne spread of infection is very unlikely in most, if not all, of the country. However, the disease could be spread very rapidly through livestock saleyards, and by the movement of animals, contaminated trucks or other items (AUSVETPLAN).

4.1.1.7. Potential effects of disease establishment

The economic effects of an outbreak of FMD, even on a small scale, would be enormous to individuals, the farming industry as a whole and to subsidiary and support industries (Hassal *et al* 1991). In addition to the direct effects on the cattle, sheep and pig industries, the export markets for wild pig meat ($35 million in 1994–95; Australian Game Meat Producers

Association, 1995) and meat from feral goats, and the trade in live camels would also be affected. The loss of export earnings in the first year is estimated at $2000 million. Employment would be affected over a whole range of industries from the farming and subsidiary industries to rural townships and governments. All exports of cloven-hoofed animals and their products would cease for an undetermined period. The export of grain and other feedstuff would also be affected by an occurrence of FMD. The prices of animal products will fall due to the closure of export markets and a domestic oversupply situation. Direct production losses of all affected industries through the disease itself would be minor in comparison (AUSVETPLAN).

4.1.1.8. Feasibility of stamping out in Australia

Australia's policy for eradication of FMD is one of stamping out. This could be supplemented if absolutely necessary by vaccination. Eradication of all but very localised outbreaks would be a massive undertaking in terms of specialist manpower, physical and financial resources. Eradication would be even harder, maybe impossible, if the disease became well established in feral animal populations (AUSVETPLAN).

4.1.1.9. OIE recommended safety precautions

Article 2.1.1.14.

When importing from FMD free countries or zones where vaccination is not practised, Veterinary Administrations should require, for fresh meat (including offal) of all FMD susceptible animals, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in the country or zone since birth, or have been imported from a country or zone free from FMD;

2) slaughtered in an abattoir and found to be free from FMD before and after slaughter.

Article 2.1.1.16.

When importing from FMD free countries or zones where vaccination is practised, Veterinary Administrations should require, for fresh meat (including offal) or meat products of pigs and ruminants other than bovines, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in the country or zone since birth, or have been imported from a country or zone free from FMD (where vaccination either is or is not practised);

2) that have not been vaccinated;

3) slaughtered in an abattoir (located in the free zone, when the animals originate from such a zone) and found to be free from FMD before and after slaughter.

Article 2.1.1.18.

When importing from FMD infected countries or zones, Veterinary Administrations should require, for meat products of domestic ruminants and pigs, the presentation of an international sanitary certificate attesting that:

1) the entire consignment of meat comes from animals slaughtered in an abattoir and found to be free from FMD before and after slaughter;

2) meat has been processed to ensure the destruction of FMD virus according to the procedures in Appendix 4.3.2.1.;

3) necessary precautions were taken after processing to avoid contact of the meat products with any potential source of FMD virus.

4.1.1.10. Recommendation

Because of the extremely severe repercussions of an outbreak of FMD in Australia, it is recommended that imports of casings not be permitted from countries where FMD is present unless the casings are effectively treated under AQIS quarantine control.

4.1.2. Rinderpest and Peste de petits ruminants (PPR)

4.1.2.1. Description and aetiology

These are closely related paramyxoviruses. Rinderpest is an acute, highly contagious disease, primarily of cattle and secondarily of sheep and goats, and all cloven hoofed animals. Pigs may become infected and spread the disease. Peste de petits ruminants (PPR) is an acute viral disease of sheep and goats.

Mortality in both diseases can be very high (Blaha 1989). In naive populations, rinderpest can cause mortality of 90%-100%, and 30% to 50% in endemic situations. Mortalities up to 90% occur in explosive outbreaks of PPR. (Fenner *et al*).

4.1.2.2. World distribution

Rinderpest is present in parts of Africa, the Middle East and South Asia. In South East and East Asia, rinderpest appears to have been eradicated, or to have disappeared naturally. There may, however, be some small pockets of infection remaining in remote areas of Indochina (Geering *et al* 1995).

Peste des petits ruminants (PPR) occurs in West Africa, and into the Arabian peninsula. It may also occur in other countries in the Middle East, and in India, although there is some doubt whether Rinderpest or PPR is the cause of the disease seen in sheep in India. (Geering *et al* 1995).

4.1.2.3. Natural and experimental hosts

Cattle and buffaloes are highly susceptible to rinderpest. Clinical rinderpest is rare in sheep and goats in Africa, although subclinical infections may occur in both species in association with

disease outbreaks in cattle (Geering *et al* 1995). It has also been reported as causing disease in sheep and goats in India, although there is some doubt whether rinderpest or PPR is the cause of this disease. Native breeds of pigs in South East Asia were quite susceptible, although European breeds of pigs were resistant (Geering *et al* 1995).

Goats and sheep are the only natural hosts for PPR. Goats appear to be more susceptible and suffer a more severe clinical disease than sheep. Subclinical infection has been reported in cattle after experimental inoculation and by contact, with subsequent antibody production. Red deer, *Cervus elaphus*, have been infected in a natural outbreak. White-tailed deer, *Odocoileus virginianus*, are susceptible to experimental infection and may develop lesions similar to those seen in sheep and goats. Some deer may become subclinically infected with virus and show no visible signs (Hamby and Dardiri 1976).

4.1.2.4. Pathology

Rinderpest and PPR can not be differentiated on clinical or pathological grounds. Lesions of rinderpest and PPR include mucosal erosions in the mouth, pharynx and anterior oesophagus. Abomasal mucosa is congested, oedematous and eroded. Necrotic patches of epithelium occur in the pyloric region of the abomasum. Small intestine lesions are relatively mild, but more severe lesions are found in the large intestine, particularly in the caecum, caeco-iliac junction and the rectum.

4.1.2.5. Pathogen inactivation

Rinderpest virus is not hardy and is usually readily inactivated by the *rigor mortis* induced fall in pH in skeletal muscle after slaughter (Blaha, 1989; Blackwell, 1984). However, this does not occur in casings so it is likely that rinderpest virus could persist in processed sausage casings. This situation is worsened by the fact that salts appear to reduce the sensitivity of rinderpest to inactivation. Rinderpest virus may survive in salted meat for several months (Blaha 1989). Since salting is a normal part of the processing for sausage casings, this protective action of salt is a significant finding in relation to this disease.

PPR virus appears to be fragile, having a half life of two to three hours at temperatures above 37^{o}C. The virus is stable between pH 5.8 and 9.5, and may be recoverable from lymph nodes for at least eight days at 4^{o}C (Coetzer *et al* 1994).

4.1.2.6. Disease transmission

The normal mode of transmission of rinderpest and PPR is through close contact between animals. However, it is possible for rinderpest virus to persist in meat and offal, and infect pigs fed on food scraps (Geering *et al* 1995). Pigs can then infect other animals such as cattle, sheep and goats, although this mode of transmission is unusual (Blaha, 1989; Fenner *et al* 1993). Because the disease is relatively mild in pigs, the infection may go unnoticed for some time, increasing the chance of spreading the infection to cattle, sheep and goats, should this route of infection occur (Blaha 1989).

4.1.2.7. Establishment requirements

Both diseases are spread, in the main, by close contact between animals. There is no evidence that PPR has been spread by infected meat and meat products, although it is possible that viable PPR virus could remain in sausage casings.

Parsonson (1992) expressed the view that rinderpest poses a relatively low risk, despite the persistence of the virus in salted meats, for two reasons:

1. the disease is so obvious and debilitating that it is unlikely that intestines suitable for casing manufacture would be harvested from affected animals; and

2. it is unlikely that casings would be ingested to cause infection of cattle.

This is not in accordance with the view of Blaha (1989), who believes that infected food scraps may be consumed by pigs, which can become infected and spread the disease to cattle. This would require that infected beef casings were fed to pigs. Limitations on swill feeding make this unlikely, although the possibility that pigs may be exposed cannot be discounted entirely. Feral pigs, for example, may be exposed to meat scraps on rural rubbish tips.

4.1.2.8. Potential effects of disease establishment

4.1.2.8.1. Rinderpest

The potential effects of the establishment of rinderpest in Australia are extremely serious. The disease could spread rapidly through susceptible Australian cattle populations. This would lead to direct losses due to slaughtering of infected and in-contact animals during the "stamping out" phase of disease eradication, as well as loss of export markets.

An uncontrolled outbreak of rinderpest in Africa from 1889 to 1896 killed 90% of ruminants in its path as it spread from the horn of Africa to South Africa. A similar result could be expected in an uncontrolled outbreak in Australia. In a large-scale outbreak, which may take several weeks to control, there would be severe widespread losses in the cattle industry and possibly in the pig, sheep and goat industries. The resulting financial losses both at the local level and the loss of export markets would have a serious effect throughout the country. Job losses both on farms and in support industries would occur during a prolonged outbreak.

An outbreak of rinderpest in Australia might reasonably be expected to cause a very high mortality in infected herds. The implementation of a stamping-out policy will not lead to the loss of many more stock on infected properties than the disease itself would cause. A large outbreak in a dairy area would affect the viability of dairy factories and may result in short-term shortages of dairy products.

Legislation in the United States currently prohibits the importation of beef from countries in which rinderpest is present, and all meat exports to the United States could therefore cease. It is possible that other countries could also place a ban on imports, at least in the short term. As the international export of meat is likely to be greatly reduced, at least in the short term, meat would only be directed to the domestic market. Prices are likely to fall. If an area supplying

milk to a major population centre is affected, milk shortages and consequent higher prices could be expected if the outbreak is large in scale. In dairying areas, however, the disease is even more amenable to eradication than in extensive grazing areas, so large-scale outbreaks should not occur (AUSVETPLAN).

4.1.2.8.2. Peste des petits ruminants

An outbreak of PPR in Australia would be expected to cause high mortality on the infected properties. The implementation of a slaughter-out policy may not lead to the loss of many more stock on infected properties than from the disease itself. There is no national agreement to pay compensation for stock destroyed and affected properties would suffer large financial losses.

If PPR became endemic, there would be continuing costs and losses due to animal mortalities, stamping out and the cost of preventative vaccination. Some industries not directly affected by PPR, such as the cattle industry, may also be affected by movement restrictions.

An outbreak of PPR will affect both local and export markets. Australia would lose its export market of live sheep and goats and their products at least in the short term until disease free zones have been well defined. If the disease spreads then greater losses will be involved. Not all products may be prohibited by our trading partners (AUSVETPLAN).

4.1.2.9. Feasibility of stamping out in Australia

Rinderpest and PPR are considered to be relatively easy diseases to control, provided adequate movement restrictions can be enforced. Rinderpest has been eradicated from Europe and southern Africa by slaughter of all infected and in-contact animals, and eradication campaigns in current endemic areas are aiming at eradicating rinderpest by vaccination and movement control globally by the year 2010. It is highly likely that these diseases would be quickly eradicated from Australia (AUSVETPLAN).

4.1.2.10. OIE recommended safety precautions

Article 2.1.4.12.

When importing from rinderpest free countries, Veterinary Administrations should require, for fresh meat or meat products of domestic ruminants and pigs, the presentation of an international sanitary certificate attesting that the entire consignment comes from animals

1) which have been kept in the country since birth, or have been imported from a rinderpest free country;

2) slaughtered in an abattoir and found to be healthy before and after slaughter.

Article 2.1.4.13.

When importing from countries considered infected with rinderpest, Veterinary Administrations should require, for meat products of domestic ruminants and pigs, the presentation of an international sanitary certificate attesting that the:

1) entire consignment of meat products comes from animals slaughtered in an abattoir and found to be healthy before and after slaughter;

2) meat products have been processed to ensure the destruction of rinderpest virus;

3) necessary precautions were taken after processing to avoid contact of the meat with any source of rinderpest virus.

Similar requirements are listed in Chapter 2.1.4 of the OIE Animal Health Code in relation to the importation of meat and meat products from small ruminants, with respect to the risk of introduction of pest de petits ruminants.

4.1.2.11. Recommendation

Although the risk of infection of Australian animals with Rinderpest or PPR as a result of the importation of sausage casings would appear to be low, the potential effects of the disease are extremely serious, especially in the short term. Therefore it is recommended that imports of casings should not be permitted from countries where rinderpest or PPR are present unless the casings are effectively treated under AQIS quarantine control.

4.1.3. Swine Vesicular disease

4.1.3.1. Description and aetiology

Swine vesicular disease (SVD) is a contagious viral disease of pigs which is indistinguishable in the field from foot and mouth disease, vesicular stomatitis and vesicular exanthema. SVD virus is a member of the enterovirus genus of the family Picornaviradae. Only one serotype is

recognised. SVD virus is very closely related to Coxsackie B5 virus of humans (Geering *et al* 1995).

4.1.3.2. World distribution

The disease has been reported from Italy, Hong Kong, the United Kingdom, France, Germany, Poland, Holland, Belgium, Switzerland, Japan, Malta and Greece. The disease is not present in North, Central or South America, although it may be present but not confirmed in some countries in Asia. It has never occurred in Australia (Geering *et al* 1995).

4.1.3.3. Natural and experimental hosts

Pigs are the only livestock species affected. Coxsackie B-virus like symptoms have been reported in some laboratory workers and animal attendants in contact with the virus (Geering *et al* 1995).

4.1.3.4. Pathology

Gross lesions of SVD are restricted to the formation and resolution of vesicles and are indistinguishable, either grossly or by histopathology, from those of FMD.

4.1.3.5. Pathogen inactivation

SVD virus is relatively resistant to inactivation. It is relatively stable over a pH range of 2-12, depending on temperature and time. It is more resistant to heating and desiccation than FMD virus. It can retain infectivity in contaminated pig faeces for 138 days. It is not destroyed by acidification in pig meat and retains infectivity in frozen pig carcasses for very long periods. In processed products such as salami it retains infectivity for more than 100 days (Geering *et al* 1995).

SVD virus has also been reported to survive in dried salami and pepperoni sausages for at least 400 days and in processed intestinal casings for at least 780 days. SVD virus is inactivated in intestinal casings if exposed to 0.5% citric acid for 24 hours, (McKercher *et al* 1980). Sodium hydroxide (0.5M, 2%), sodium hypochlorite (0.005M, 0.04% and formaldehyde (2.6M, 8%) completely inactivated SVD virus within 2 minutes exposure (Blackwell *et al* 1975).

4.1.3.6. Disease transmission

All tissues of infected pigs can contain virus and can therefore be a vehicle for transmission. In particular, SVD virus can survive for at least 780 days in salted pig casings (Loxam and Hedger, 1983). Tissues of infected pigs can contain large amounts of virus before the clinical signs become apparent. The disease is sometimes very mild, and as a result of this, the disease may not always be recognised and reported correctly. It is therefore possible that affected pigs may be slaughtered, and casings collected from them for human consumption. Contaminated pig meat can find its way into the food chain. However, time of slaughter in relation to infection with SVD virus is critical. Virus levels in infected pigs are highest 2-3 days

after infection and drop rapidly as antibodies develop. The amount of virus in pork products would be very small unless they were prepared from a herd early in an epidemic. The dose of SVD virus required to produce infection in pigs is approximately $7X10^5$ ID_{50} Titres of virus in infected pig meat are round 10^3-$10^{4.5}$ In order for a pig to become infected, it must eat approximately 22-700 gm of infected meat. While this is possible the likelihood of it happening is small, especially given the current bans on swill feeding of commercial pigs.

Because of the resistance of the virus to inactivation, both indirect spread by fomites and the recrudescence of the disease when farms are restocked after inadequate disinfection, are common.

4.1.3.7. Establishment requirements

In order for SVD to become established in Australia, a susceptible pig must come into contact with an infectious dose of the virus. Although this would appear to be unlikely in the case of sausage casings, it is possible. The disease is also readily spread by fomites, and therefore hides and skins, and pig hair bristles, must be considered to pose a risk of introduction of the disease.

4.1.3.8. Potential effects of disease establishment

A major part of the economic importance of this disease stems from its similarity to FMD. All exports of livestock and livestock products would cease immediately an outbreak was notified, and would not recommence until after laboratory confirmation that the disease was not FMD. Once this confirmation had been received, the export of pigs would be restricted until 6 months after the disease was stamped out. MacDiarmid (1991) expressed the opinion that sheep exports may also be affected, as sheep may be experimentally infected, although they show no signs of disease as a result. This is considered to be an unlikely outcome.

The extent of the social and economic effects of SVD would depend 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. Any confusion with FMD, if reported internationally, is likely to affect cattle, sheep and goat export industries at least in the short term.

If the disease is not eradicated new outbreaks will result in a vesicular disease investigation each time and perhaps confusion in international markets. SVD could affect the viability of some producers due to lost markets. However, the overall effect on the pig industry and on the national economy would be minor compared to a confirmed outbreak of FMD. The clinical effects of SVD are of little economic importance. (AUSVETPLAN).

4.1.3.9. Feasibility of stamping out in Australia

Conventional stamping-out procedures have been shown to eradicate SVD in the United Kingdom although this was with difficulty. Eradication is difficult due to resistance of the virus to inactivation, its capacity to be spread by fomites and also within the food chain and the fact that many cases of the disease are mild enough to go unnoticed initially allowing movement and spread of the virus. (AUSVETPLAN)

The disease may be difficult or impossible to eradicate if it became well established in the feral pig population of Australia (Geering 1990).

4.1.3.10. OIE recommended safety precautions

Article 2.1.3.3.

Veterinary Administrations of SVD free countries may prohibit importation or transit through their territory, directly or indirectly, from countries considered infected with SVD of:

3) fresh meat of domestic and wild pigs;

4) meat products of domestic and wild pigs which have not been processed to ensure the destruction of SVD virus;

Article 2.1.3.10.

When importing from SVD free countries, Veterinary Administrations should require, for fresh meat of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in an SVD free country since birth or for at least the past 28 days;

2) slaughtered in an abattoir and found to be healthy before and after slaughter.

Article 2.1.3.11.

When importing from countries considered infected with SVD, Veterinary Administrations should require, for fresh meat of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have not been kept in an SVD infected zone;

2) slaughtered in an abattoir not situated in an SVD infected zone and found to be healthy before and after slaughter.

Article 2.1.3.12.

When importing from countries considered infected with SVD, Veterinary Administrations should require, for meat products of pigs, the presentation of an international sanitary certificate attesting that the:

1) entire consignment of meat products comes from animals slaughtered in an abattoir and found to be healthy before and after slaughter;

2) meat products have been processed to ensure the destruction of SVD virus;

3) necessary precautions were taken after processing to avoid contact of the meat with any source of SVD virus.

4.1.3.11. Recommendation

Because of the serious nature of the disease and its resistance to inactivation, importation of products derived from pigs should not be permitted from countries where SVD is present unless the casings are effectively treated under AQIS quarantine control.

4.1.4. African swine fever

4.1.4.1. Description and aetiology

African Swine Fever (ASF) is probably the most serious viral disease threatening the world's pig producing industries (Fenner *et al* 1993). The disease affects only pigs. It can cause extremely high mortalities in outbreaks. Up to 100% mortality has been recorded (Wilkinson 1989; Geering *et al* 1995).

4.1.4.2. World distribution

ASF is present in most of sub-Saharan Africa. In 1957, the disease spread to Portugal, where it was eradicated, but it re-appeared in 1960 and quickly spread to Spain. ASF remains endemic in the Iberian peninsula and this region has been a major source for spread to other countries. ASF is now under good control in both Spain and Portugal and mainly restricted to feral pigs in only small areas of both countries. The disease spread to France, Italy (now endemic in Sardinia), Malta, Belgium, Holland, Cuba, Brazil, Dominican Republic and Haiti. In most cases it has been eradicated. In the cases of Malta and the Dominican Republic, ASF was eradicated by the total elimination of pigs from these countries (Geering *et al* 1995). There have been no occurrences in Australia (AUSVETPLAN).

4.1.4.3. Natural and experimental hosts

Domestic and feral pigs (*Sus scrofa*) are the only species susceptible in Australia. In Africa, the African wart-hog (*Phacochoerus aethiopicus*), African bush pig (*Potamochoerus porcus*) and possibly the African giant forest hog (*Hylochoerus meinertzhageni*) are susceptible to infection but do not show clinical signs. They are important in the epidemiology of the disease in Africa (AUSVETPLAN).

4.1.4.4. Pathology

4.1.4.4.1. Gross lesions

4.1.4.4.1.1. Acute form:

· lymph nodes are enlarged and haemorrhagic often resembling blood clots — the gastrohepatic, renal, mesenteric and submandibular lymph nodes are most often affected;

· spleen may be enlarged (2–3 times normal size), dark, friable or pulpy;

· haemorrhages can occur in almost any organ. They are most commonly seen on serosal membranes and in kidneys (as subcapsular petechiae), heart, urinary bladder, lung and gall bladder;

· septal oedema of lungs resulting in prominent interlobular septa; and

· fluid in body cavities.

4.1.4.4.1.2. Subacute form:

· findings are more variable than for the acute form;

· lymph node and renal haemorrhage;

· enlarged but not congested spleen;

· lobular consolidation of cranial lung lobes; and

· haemorrhage of the intestinal lining, lymph nodes and kidney.

4.1.4.4.1.3. Chronic form:

· enlarged lymph nodes;

· fibrinous pericarditis and pleurisy;

· lobular consolidation of lungs — may progress to lobular necrosis;

· small hard nodular white masses in lungs;

· arthritis;

· cutaneous ulcers;

· poor body condition.

4.1.4.4.2. Microscopic lesions (histopathology)

Extensive necrosis of lymphatic tissue is common, particularly in lymph nodes with karyorrhexis (nucleus fragmentation in degenerating granular leucocytes) of lymphocytes, and this may be accompanied by haemorrhage. Necrosis is more severe and frequent with ASF than classical swine fever (CSF). There is vasculitis, with degeneration of endothelium and fibrinoid degeneration of artery walls in all organs. There is non-purulent inflammation of the brain, spinal cord and spinal nerves with necrosis of mononuclear cell cuffs around affected vessels (AUSVETPLAN).

4.1.4.5. Pathogen inactivation

4.1.4.5.1. General properties/environment

ASF virus survives for prolonged periods under most environmental conditions and is resistant to many treatments that readily inactivate other pathogens (McDaniel 1980). It is not inactivated by freezing and thawing. ASF virus can be inactivated in liquid media by heating at 60° C for 30 minutes (Blaha 1989, Pensaert 1989). ASF virus is stable in a wide range of pH (pH 4–10). However, the intact virus is very sensitive to lipid solvents and detergents and also to oxidising agents such as hypochlorite as well as substituted phenols (Plowright *et al* 1994).

4.1.4.5.2. Animal products and by-products

The virus may survive for many months in raw, unprocessed frozen meat. ASF virus has been recovered after 150 days from infected meat kept at 4° C, after 104 days from meat kept at –4° C and after 188 days from bone marrow stored at –4° C (Fenner *et al* 1993).

The virus has been recovered from putrefied serum stored at room temperature for 15 weeks and from blood stored at 4° C for 18 months to 6 years. It has also been recovered from processed hams after 5 months of storage and from the bone marrow of such hams stored for 6 months (McDaniel 1980).

In the Belgian outbreak the European Union required that pigmeat produced in the infected area be sealed in hermetically closed containers and held at a temperature of at least 60° C for a period of four hours and for at least 30 minutes of this period above 70° C.

On one farm 115 pigs were exhumed 3 months after they died. No virus was detected in their tissues, however, lesions were present and antibodies were detected (AUSVETPLAN).

4.1.4.6. Disease transmission

ASF virus is resistant to pH changes which accompany *rigor mortis* and it is not inactivated by freezing and thawing (Fenner *et al* 1993; Blaha 1989; Wilkinson 1989). Brining was not sufficient to inactivate ASF virus in hams, and virus has been recovered from brined hams for up to six months (Blackwell, 1984).

The virus is relatively heat stable, but is inactivated in liquid media by heating at 60°C for 30 minutes (Blaha 1989, Wilkinson 1989). Cooked or canned products are safe provided they have been heated throughout to 70°C (Blackwell 1984, Wilkinson 1989).

It is not known how long the virus may persist in recovered animals, but outbreaks have been attributed to "carrier" pigs. There is no experimental evidence, however, to indicate that recovered animals can transmit the virus for more than a few weeks after infection (Wilkinson, 1989). However, the possibility of an asymptomatic carrier state can not be discounted. Requiring that slaughter pigs be free of clinical signs of ASF will not therefore provide sufficient safeguard against the presence of ASF virus in sausage casings.

4.1.4.7. Establishment requirements

Possible methods of introduction of ASF virus into Australia include illegally imported pig meats and other pig products, garbage from international aircraft and ships, biological products and illegally imported pigs and boar semen.

Feral pigs would be an important reservoir if they became infected (AUSVETPLAN).

4.1.4.8. Potential effects of disease establishment

Losses caused by ASF include mortalities, which can be very high, and loss of income from reduction of meat production and increased feed costs. An uncontrolled outbreak in Australia would result in severe losses and unemployment at the farm, processor and retail levels. Prices of other animal products might rise. If eradication can be undertaken quickly and effectively there may be no lasting damage to the pig industry provided it could recover its market share.

Those producers whose pigs escaped infection might attract a premium price for their produce but they might not benefit from the misfortunes of other producers. In Belgium, pigmeat consumption fell by 25% during the 1985 outbreak despite assurances that it was perfectly safe for human consumption.

Prolonged loss of income for producers whose herds are destroyed and subjected to quarantine controls would be substantial; there would be reduced market opportunities and changes to management practices. The stamping-out strategy may cause the destruction of some genetically important herds even though special efforts would be taken by their owners to protect them (AUSVETPLAN).

4.1.4.9. Feasibility of control in Australia

Eradication of ASF is extremely difficult and involves considerable resources. In major outbreaks of the disease, eradication has only been achieved by total national depopulation of pigs. If feral pig populations in Australia became infected then eradication may be impossible (AUSVETPLAN).

4.1.4.10. OIE recommended safety precautions

Article 2.1.12.10.

When importing from ASF free zones in ASF infected countries, Veterinary Administrations should require, for fresh meat of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in an ASF free country or free zone since birth;

2) slaughtered in an abattoir situated in an ASF free country or free zone and which only receives animals from a free country or free zone;

3) found to be healthy before and after slaughter.

Article 2.1.12.11.

When importing from ASF free zones in ASF infected countries, Veterinary Administrations should require, for meat products of pigs, the presentation of an international sanitary certificate attesting that these products:

1) have been processed from meat complying with provisions referred to in Article 2.1.12.10.;

2) have been processed in meat processing plants situated in an ASF free country or free zone, and in which only meat of animals from an ASF free country or free zone is processed.

Article 2.1.12.12.

When importing from countries considered infected with ASF, Veterinary Administrations should require, for meat products of pigs, the presentation of an international sanitary certificate attesting that the

1) entire consignment of meat products comes from animals slaughtered in an abattoir and found to be healthy before and after slaughter;

2) meat products have been processed to ensure the destruction of ASF virus;

3) necessary precautions were taken after processing to avoid contact of the meat with any source of ASF virus.

It appears clear from the context that Articles 2.1.12.10 and 2.1.12.11 are intended to refer also to imports from ASF free countries, as well as ASF free zones within infected countries.

4.1.4.11. Recommendation

Because of the extremely serious consequences of an outbreak of ASF in Australia, importation of products derived from pigs should not be permitted from countries where ASF is present unless the casings are effectively treated under AQIS quarantine control.

4.1.5. Hog cholera (Classical swine fever)

4.1.5.1. Description and aetiology

Hog cholera (Classical Swine Fever - CSF) is a highly infectious viral disease of pigs. It is characterised by rapid spread, septicaemia, haemorrhage and high mortality, which approaches 100% in susceptible populations (Leman *et al* 1981, Somerville, 1988). Worldwide, this is probably the most economically significant disease of swine (Fenner *et al* 1987, Somerville, 1988).

4.1.5.2. World distribution

CSF is present throughout Europe with the exception of Ireland, Iceland, Switzerland and the Scandinavian countries. Outbreaks occurred in the United Kingdom in 1986, but it has since been eradicated. It is also present in East and Central Africa, the Indian subcontinent, China, East and Southeast Asia, Mexico and most other countries in Central America, and throughout most of South America.

Outbreaks of CSF occurred in Australia in 1903, 1927–28, 1942–43 and 1960–61. In each case the disease was eradicated. The first three outbreaks were of virulent disease. They resulted from either imported pig meat or food refuse from ships being swill-fed to pigs.

The origin of the 1960–61 outbreak is unknown, but probably similar. This outbreak was caused by a strain of low virulence, and only came to official attention as a result of a higher than normal condemnation rate for 'septicaemia' of pig carcasses in abattoirs (Geering *et al* 1995).

4.1.5.3. Natural and experimental hosts

Pigs are the only species affected by Hog cholera, according to Fenner *et al.* (1993), Geering *et al.* (1995), and Terpstra (1994). However, Parsonson (1992) quotes work by Collett, *et al.* (1989) who suggest that cross infection between animal species is well documented and infection from cattle to sheep and pigs has been shown to occur.

4.1.5.4. Pathology

The incubation period is 3 to 8 days and persistent sub-clinical infection has been reported in carrier sows (Geering *et al* 1995), so it is possible that infected pigs could be slaughtered for human consumption, and pass both *ante mortem* and *post mortem* inspection.

4.1.5.5. Pathogen inactivation

Hog cholera virus is relatively stable and can survive 50^{o}C for 3 days, 37^{o}C for 7 to 15 days, and -70^{o}C for years. It survives for several years in frozen pork, and for months in chilled meat and bone marrow. The virus is stable within the pH range 3.0-13.0. (Blaha 1989). Salted and smoked products may still harbour virus, so the likelihood of the virus being present in salted casings is high (Blaha 1989). The virus is susceptable to a range of disinfectants including detergents. Recommended disinfectants include sodium hypochlorite (2.3% available chlorine), alkali wash and 4% lysol (Geering 1979).

4.1.5.6. Disease transmission

4.1.5.6.1. Live animals

Spread is by direct contact with infected pigs or by ingestion of products from infected pigs. Movement of infected pigs is the most important method of spread. Pigs incubating the acute form of the disease can shed virus before showing clinical signs. Chronic carriers (pregnant carrier sows and immunotolerant pigs born to carrier sows) are particularly important in the

epidemiology of an outbreak as they are clinically normal. In infected herds up to 43% of pregnant sows may be carriers. Breeding stock sales have been important in the spread of CSF overseas. However, there are very few movements of pregnant sows from one farm to another in Australia.

CSF virus is excreted in the highest concentration in oral fluid, with smaller quantities in urine, faeces and nasal and lachrymal fluids. Large quantities of virus may be disseminated when carrier sows farrow (AUSVETPLAN).

4.1.5.6.2. Animal products and by-products

The ingestion by pigs of pigmeat or pigmeat products infected with the virus is an important method of spread of CSF, especially in the first outbreak in a country. The unlicensed feeding of swill (food scraps containing material of placental mammal origin) is illegal in Australia.

Processed intestinal casings produced hog cholera lesions and death when inoculated into pigs after 147 days storage (McKercher *et al* 1978).

Processed pigmeat products from an European Community country infected with CSF were considered to be the most likely source of CSF virus in the 1986 English outbreak (AUSVETPLAN). About 60% of outbreaks of Hog Cholera in previously free territories arise from the feeding of food scraps of contaminated imported meats to pigs (Blaha 1989).

4.1.5.7. Establishment requirements

Pigmeat products, introduced legally or illegally, and ship's garbage are believed to have been the source of most of the incursions of CSF into Australia. Introduction of pig products from endemic countries is still the greatest risk however the swill feeding ban has reduced this risk considerably. Entry could be by smuggled meat products through airports or the mail, or garbage from ships, aircraft, yachts, etc. Fishing vessels from some Asian countries may constitute a risk. Feral pigs in contact with rubbish tips and food refuse from ships, on beaches and so on, remains a significant risk especially if contact or mating with domestic pigs occurs.

4.1.5.8. Potential effects of disease establishment

Losses caused by CSF include mortalities, which can be very high, and loss of income from reduction of meat production and increased feed costs. An uncontrolled outbreak in Australia would result in severe losses and unemployment at the farm, processor and retail levels. If eradication were achieved there is unlikely to be continuing damage to the industry beyond the need to recover its market share.

Prolonged loss of income for producers whose herds are destroyed will have a serious social and economic effect on them and their families. Movement controls will cause severe disruptions to the marketing of slaughter-weight pigs and breeding stock. There is no compensation for lost market opportunities for uninfected farms included in a control area.

The stamping-out strategy may cause the destruction of some genetically important herds even though special efforts would be taken by their owners to protect them (AUSVETPLAN).

4.1.5.9. Feasibility of control in Australia

If a low virulence strain of CSF were found to be widespread in Australia careful consideration would need to be given to formulating an appropriate control program. This could include voluntary accreditation of CSF-free herds and active dissemination of control information to the industry.

CSF has been eradicated from Australia previously by traditional stamping-out procedures of slaughter, disinfection, quarantine and movement controls. It is however considered that CSF would be very difficult to eradicate and would require considerable resources. It may be impossible to eradicate in some circumstances, for example if there was feral animal involvement (AAHL 1990) (AUSVETPLAN).

4.1.5.10. OIE recommended safety precautions

Article 2.1.13.3.

Veterinary Administrations of CSF free countries may prohibit importation or transit through their territory, directly or indirectly, from countries considered infected with CSF of:

4) fresh meat of domestic and wild pigs;

5) meat products of domestic and wild pigs which have not been processed to ensure the destruction of CSF virus;

Article 2.1.13.10.

When importing from CSF free countries, Veterinary Administrations should require, for fresh meat of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in a CSF free country since birth or for at least the past 40 days;

2) slaughtered in an abattoir and found to be healthy before and after slaughter.

4.1.5.11. Recommendation

Because of the extremely serious consequences of an outbreak of CSF in Australia, importation of products derived from pigs should not be permitted from countries where CSF is present unless the casings are effectively treated under AQIS quarantine control.

4.2. OIE List B diseases

4.2.1. Aujeszky's Disease (Pseudorabies Virus)

4.2.1.1. Description and aetiology

Pseudorabies virus is a member of the Herpesvirus family.

4.2.1.2. World distribution

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

AD has never been diagnosed in Australia (AUSVETPLAN).

4.2.1.3. Natural and experimental hosts

It primarily infects pigs, which are the only known virus reservoir. Other species such as cattle, sheep, dogs, and cats may be infected, but there is no evidence of these species transmitting virus. In all species other than swine, the disease is rapidly fatal. Humans are not susceptible. (Pensaert and Kluge 1989).

4.2.1.4. Pathology

Necrotizing enteritis has been reported in piglets inoculated intranasally. Intranuclear inclusion bodies may be found in degenerating crypt epithelial cells on microscopic examination. This, combined with the reported ability for virus to be shed by recovered pigs, suggests that it would be possible for sausage casings derived from slaughtered pigs to contain AD virus.

Generally, pigs will excrete virus during the two-four weeks following primary infection, but virus persistence with continuous excretion has been reported (Pensaert and Kluge 1989). Thawley *et al* (1980) reported that AD Virus had been detected in pigs up to 6 months after recovery.

4.2.1.5. Pathogen inactivation

The virus is generally not resistant to disinfectants, and is extremely sensitive to lipid solvents such as ethyl ether, acetone, chloroform and alcohol (Pensaert and Kluge 1989). The effect of disinfectants on inactivation of virus varies with the concentration of the disinfectant, the temperature and the pH range (Pensaert and Kluge 1989).

AD virus was rapidly inactivated (in seconds) by 0.5% sodium hypochlorite, but relatively resistant to sodium hydroxide at 0.8% and 1.6% (> 6 hours) 3% phenolic derivatives inactivated the virus within 10 minutes, and 0.6% formaldehyde within 1 hour (Pensaert and Kluge 1989).

4.2.1.6. Disease transmission

Cases of AD in dogs, cats, farmed mink and ferrets, and wild rats have been attributed to the consumption of meat from infected pigs. Pigs have been infected through eating carcasses of infected rats. Pigs can possibly be infected through consumption of infected pork scraps, but this is considered unlikely (MacDiarmid and Thompson 1997).

4.2.1.7. Establishment requirements

Since AD is rapidly fatal in sheep and goats, these are not likely to be a source of infected casings. Pig casings may be infected, but in order for disease establishment to occur, these must be fed to pigs, and transmission by this method is considered unlikely. Infected pig casings fed to dogs and cats could transmit the disease, but dogs and cats are dead end hosts, so the disease cannot become established in Australia as a result of this method of infection (MacDiarmid and Thompson 1997). Rats and wildlife may have some role as reservoirs and vectors (Geering *et al* 1995) Therefore it is possible that rats which gain access to imported casings or wastes derived from them, may become infected and transfer the infection to feral pigs.

4.2.1.8. Potential effects of disease establishment

Losses to individual producers and to the industry as a whole could be substantial if the disease is allowed to proceed uncontrolled over the long term. The cost of endemic disease has justified the undertaking of eradication in a number of countries. The strategy to act quickly when the disease is detected in Australia and the likely slow spread between herds in this country gives a high likelihood of succeeding with an eradication plan. This is likely to be the case even if the disease goes undetected for some time but losses would be greater.

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

4.2.1.9. Feasibility of control in Australia

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

4.2.1.10. OIE recommended safety precautions

Article 3.1.2.2.

Veterinary Administrations of importing countries should require, for fresh meat and meat products of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals slaughtered in an abattoir and found to be healthy before and after slaughter.

4.2.1.11. Recommendations

No specific restrictions should apply in relation to this disease.

4.2.2. Transmissible spongiform encephalopathies

The transmissible spongiform encephalopathies (TSEs) or prion diseases, are a group of highly unusual diseases of humans and animals. The TSEs are characterised by prolonged incubation period, spongiform degeneration of neuronal tissue, and an invariably fatal course (Geering *et al* 1995). The current list of recognised TSEs is shown in Table 5.

Table 5: Transmissible spongiform encephalopathies of animals and humans.

Disease	**Host**
Scrapie	Sheep, goats, mouflon
Bovine spongiform encephalopathy	Cattle, some antelope species
Creuzfeldt-Jakob Disease	Humans
Kuru	Humans
Gerstmann-Sträussler-Scheinker syndrome	Humans
Fatal familial insomnia	Humans
Transmissible mink encephalopathy	Mink
Chronic wasting disease	Deer
Feline spongiform encephalopathy	Cats, cheetahs

The nature of the causal agents of the TSEs is not clearly understood. No conventional infective agents have been visualised, nor have any nucleic acids been clearly associated with them. The causal agents are remarkably resistant to inactivation, and retain infectivity after

chemical and physical treatments which would denature nucleic acids (Geering *et al* 1995). Prions, which appear to consist of PrP, or protease resistant protein, have been identified as being consistently present in the brains of affected animals. The exact mechanism of reproduction of prions is not understood. A number of hypotheses have been proposed to explain the observed 'replication' of prion protein, and the observed strain variation that occurs with scrapie, and possibly bovine spongiform encephalopathy (BSE) (Geering *et al* 1995).

Of the TSEs, only BSE and scrapie are of concern when considering the import of sausage casings.

4.2.2.1. Bovine spongiform encephalopathy

Australia has a policy on the import of cattle and products derived from cattle, which was prepared as a result of a specific import risk analysis on BSE. The policy defines specific risk materials, which includes intestines. This policy provides, in relation to intestines and protein products derived from them, as follows:

> **"Specified risk materials**
>
> 1 From *countries* or *zones* with a *high incidence of BSE*.
>
> Bovine brains, eyes, spinal cord, tonsils, thymus, spleen, intestine, dorsal root ganglia, trigeminal ganglia, bones, and derived protein products from cattle over six months of age, may not be imported.
>
> 2 From *countries* or *zones with a low incidence of BSE*.
>
> Bovine brains, eyes, spinal cord and distal ileum, and derived protein products from cattle over six months of age, and born before the ban on the feeding of ruminant-derived *meat meal* to ruminants, may only be imported if the raw material was treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents.
>
> 3 From *BSE provisionally free countries* or *zones*
>
> Bovine brains and spinal cord and derived protein products from cattle over 30 months of age, and born before the ban on the feeding of ruminant-derived *meat meal* to ruminants, may be imported if the raw material was treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents."

The OIE Animal Health Code, Article 4.3.3.1 provides as follows:

> "APPENDIX 4.3.3.1.
>
> MEAT-AND-BONE MEAL

For the inactivation of transmissible spongiform encephalopathy agents for the production of meat-and-bone meal containing ruminant proteins, the following process (under study) should be used:

1) The raw material should be reduced to a maximum particle size of 50 mm before heating.

2) The raw material should be heated to a temperature of not less than 133°C for a minimum of 20 minutes at an absolute pressure of 3 bar."

As the requirements of Article 4.3.3.1 of the Code are inconsistent with final use of the product as sausage casings, this policy requires that casings derived from bovine animals may only be imported from

a) countries free, or provisionally free of BSE; or

b) from animals less than 6 months of age.

4.2.2.2. Scrapie

Australia has a draft policy (due for implementation on 12 April 1999) on the import of meat and meat products, which makes provisions relating to the import of meat products derived from sheep and goats, which is designed to address the risk of introduction of scrapie. was prepared as a result of a specific import risk analysis on BSE. The policy defines specific risk materials, which includes intestines. This policy provides as follows:

"SCRAPIE

[*Note: These conditions apply to ovine and caprine (sheep and goat)* meat *and* meat products *only*.]

The consignment does not contain offals (skulls including brains and eyes, spinal cord, tonsils, thymus, spleen, distal ileum, proximal colon, lymph nodes, adrenal gland, pancreas, liver or bone marrow), and protein products derived from them, from sheep and goats over 12 months of age originating from *countries* or *zones* not considered free from scrapie."

In accordance with this policy, casings derived from sheep or goats over 12 months of age may not be imported from countries not considered free of scrapie.

4.2.3. Transmissible gastro-enteritis

4.2.3.1. Description and aetiology

Transmissible gastro-enteritis (TGE) is a virulent coronavirus which causes severe diarrhoea and mortality in baby pigs (Bohl 1989). Recently, a closely related but antigenically distinct variant has appeared. This variant does not cause diarrhoea, but replicates in the respiratory tract and spreads by the aerogenic route (Pensaert 1989).

4.2.3.2. World distribution

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 (AUSVETPLAN).

4.2.3.3. Natural and experimental hosts

The classical variant can affect pigs of all ages, but young pigs suffer the most severe disease. In susceptible new born pigs, mortality may approach 100%, but this declines with increasing age, being very low in pigs over 5 weeks of age (Bohl 1989).

4.2.3.4. Pathology

Infected pigs have a brief viraemia only (Blaha 1989) and virus is recovered from few normal-appearing pigs at slaughter. MacDiarmid (1991) reports the findings of 2 studies into isolation of TGE in swabs from clinically normal pigs at slaughter, where 1.5% and 0.8% respectively were found to be infected.

4.2.3.5. Pathogen inactivation

TGE virus is stable when stored frozen, but somewhat labile at room temperature or above (Bohl 1989). Storage at 37^{o}C for 4 days resulted in a total loss of infectivity (Harada *et al* 1968). Due to the preservation of casings by salt, and storage at room temperature, it is unlikely that TGE would be transmitted in processed casings. However, no direct evidence that this was the case could be found. TGE virus is sensitive to a wide variety of disinfectants, including sodium hypochlorite, sodium hydroxide, formaldehyde solution (including vapour), iodine, phenolic, and quaternary ammonium compounds (Brown, 1981).

4.2.3.6. Disease 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.

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

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).

Forman (1991) demonstrated the possibility that TGE could be transmitted to susceptible pigs by ingestion of material derived from infected pigs. However, the disease could only be demonstrated in 4 of 12 one-week-old piglets orally inoculated with a tissue homogenate prepared from pigs slaughtered after direct exposure to infected piglets. Of 12 three-week-old piglets fed 1.5 kg of infected tissue daily for 5 days, none showed sign of disease although seroconversion did occur. These pigs were in close contact with the younger animals which did show signs of illness.

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. This work also demonstrated infection of very young pigs (6 days old at first dose), when dosed orally on 4 consecutive days with an homogenate derived from infected tissues.

While the work of Forman (1991) and Cook *et al* (1991) demonstrate the theoretical possibility that transmission in infected casings can occur, this probability must be considered to be extremely low. This is based on the low probability of infection in slaughter age pigs, the apparently high dose of infected material required to produce infection, the increased susceptibility of young pigs when compared with adult pigs, and the ban on swill feeding in Australia.

4.2.3.7. Establishment requirements

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. Swill feeding is illegal in Australia (AUSVETPLAN).

4.2.3.8. Potential effects of disease establishment

The introduction of TGE into Australia would have serious consequences for the pig industry.

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 infected properties, 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 infected property.

Movement controls will be largely restricted to the infected properties 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 (AUSVETPLAN).

4.2.3.9. 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 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 infected property 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 (AUSVETPLAN).

4.2.3.10. OIE recommended safety precautions

The OIE Code makes no specific recommendations relating to the importation of sausage casings, with respect to the risk of introduction of transmissible gastroenteritis.

4.2.3.11. Recommendation

On the basis of the information presented above, it is considered that the importation of sausage casings derived from pigs does not represent a high risk of the introduction of this disease to Australia. No specific safeguards are required.

4.2.4. Trichinellosis (trichinosis)

4.2.4.1. Description and aetiology

This disease is caused by infestation with *Trichinella spiralis*, a parasite infecting pigs, dogs, cats, rats, and humans. In humans, it is a serious zoonosis, and can cause mortalities as high as 40%. The main importance of trichinellosis lies in the danger posed to humans (Geering *et al 1995*).

4.2.4.2. World distribution

Trichinellosis is found more commonly in temperate than in tropical areas. It is present in North America, Argentina and Chile, northern and eastern Europe and Spain, the former Soviet Union, Lebanon, Nepal, Thailand, Indonesia Egypt, Kenya and the north island of New Zealand. It does not occur in Australia (Geering *et al 1995*).

4.2.4.3. Natural and experimental hosts

All mammals are susceptible, but infestation is most common in carnivores and omnivores. Pigs, dogs and cats are the major domestic species affected, although the incidence in horses is increasing. Several wild animal species have also been recorded as being susceptible (Geering *et al 1995*).

4.2.4.4. Pathology

The adult parasite lives in the small intestine. Larvae produced by adult females penetrate the intestinal wall and enter the circulatory system. They are transported to striated muscles, where they lodge in the most active muscle groups. Cysts in muscle may remain viable for years.

4.2.4.5. Pathogen inactivation

The trichinae are inactivated by cooking, generally to a core temperature of 60^{o}C. For swill feeding to pigs, a temperature of 100^{o}C for 30 minutes is recommended. The trichinae are also destroyed by freezing. Recommended time/temperatures for destruction of trichinae in frozen meat are as follows:

-15°C for 20 days

-23°C for 10 days

-25°C for 20 days, if >15 cm thick

-25°C for 10 days, if< 15 cm thick

-30°C for 6 days

-35°C for 40 minutes.

Since sausage casings are not routinely preserved by freezing but by salting, it is unlikely that this method of inactivation of trichinae will be used. Cooking of sausage casings is also unlikely to be acceptable. However, since *T. spiralis* larvae encyst in striated muscle, it is unlikely that sausage casings of porcine origin will pose a risk.

4.2.4.6. Disease transmission

Infestation is spread when encysted larvae in muscle are eaten by carnivores or omnivores. Occasionally, infestation may be spread by larvae passed in faeces of an infested host. Pigs usually become infested by feeding of uncooked food scraps.

Human infestation generally follows the eating of infested pig meat, but infestation has been reported after eating bear (and other game meat), marine mammal meat, and horse meat.

4.2.4.7. Establishment requirements

The disease could become established if underprocessed pork meat was eaten by susceptible pigs in Australia. Bans on swill feeding, and the predilection for striated muscle, mean that this is unlikely to occur from importation of sausage casings.

4.2.4.8. Potential effects of disease establishment

The major effects of the introduction of trichinellosis arises from the potential for human disease should the organism become established.

4.2.4.9. OIE recommended safety precautions

Article 3.5.3.1.

Veterinary Administrations of importing countries should require, for fresh meat of swine (domestic and wild), the presentation of an international sanitary certificate attesting that the entire consignment of meat:

1) comes from domestic swine slaughtered and inspected in an abattoir or wild swine which have been inspected; and

2) showed a negative response to a testing procedure for trichinellosis; or

3) comes from domestic swine which were born and bred in a country or part of the territory of a country free from trichinellosis in domestic swine; or

4) has been processed to ensure the destruction of all the larvae of the parasite.

4.2.4.10. Recommendation

No specific safeguards are required in relation to the possible introduction of trichinellosis in sausage casings.

4.2.5. Porcine polioencephalomyelitis / Enterovirus encephalomyelitis (Teschen/Talfan Disease)

4.2.5.1. Description and aetiology

Polioencephalomyelitis caused by enteroviruses occurs sporadically in many parts of the world. The milder form (Talfan disease or sporadic porcine encephalomyelitis) occurs in Australia. Teschen disease is a more virulent manifestation of porcine polioencephalomyelitis with a high morbidity and mortality. It is a disease of high morbidity and mortality, and is associated with major economic losses (Derbyshire 1986). This has never been reported in Australia. Both are caused by type 1 porcine enteroviruses, of the family Picornaviridae (Geering *et al* 1995).

Porcine enteroviruses are gut inhabitants that cause systemic infection and invade the nervous system.

4.2.5.2. World distribution

Teschen disease is found in Central and Eastern Europe, and in Madagascar. Teschen has never been reported in Australia, but the milder Talfan disease (sporadic porcine encephalomyelitis) which is also caused by a porcine enterovirus serotype 1, is present (Geering *et al* 1995).

4.2.5.3. Natural and experimental hosts

Pigs are the only natural host of the disease (Geering *et al* 1995).

4.2.5.4. Pathology

There are no characteristic gross lesions. Histologically, lesions are confined to the central nervous system. Lesions involve the whole of the cerebrospinal axis.

4.2.5.5. Pathogen inactivation

The viruses are relatively heat resistant, surviving for 15 minutes at 60^{o}C and for longer periods at 56^{o}C. Between pH 2.8 and 9.5, the virus is stable, although rapidly inactivated at more extreme values. Sodium hypochlorite and ethyl alcohol were the only commonly used

disinfectants that were effective in inactivating Teschen virus (Derbyshire, 1989). The virus is also highly resistant to the environment, and it has been shown to survive for more than 168 days at 15°C (Derbyshire 1986).

4.2.5.6. Disease transmission

The disease is highly contagious, and can cause outbreaks with extremely high morbidity and mortality rates. Infected pigs excrete virus in faeces and oral secretions. Transmission of the virus is most commonly by the faecal/oral route (Derbyshire 1986). Clinically recovered pigs can continue to excrete virus in faeces for up to seven weeks (Geering *et al* 1995). It is therefore possible that virus may be present in clinically normal pigs at slaughter, and could therefore infect casings prepared from the carcasses. Swill feeding has been reported as a source of spread of the virus (Geering *et al* 1995). Indirect transmission by fomites is likely, since the virus is relatively resistant (Derbyshire 1986).

4.2.5.7. Establishment requirements

In order for Teschen disease to become established in Australia as a result of the importation of sausage casings, infected casings would have to be fed to pigs. This is an unlikely occurrence due to the ban on swill feeding, but is not impossible.

4.2.5.8. Potential effects of disease establishment

Introduction of Teschen disease virus to Australia would be likely to cause major economic losses to the pig industry. Because of the high morbidity and mortality, it is likely that infection in a commercial herd would be rapidly diagnosed, and could therefore be readily eradicated by stamping out. However, the major risk of disease introduction would appear to be through the possibility of feral pigs being exposed to waste material on garbage dumps, or possibly through the contamination of the environment by waste from casings processing plants.

4.2.5.9. OIE recommended safety precautions

Article 3.5.4.3.

Veterinary Administrations of enterovirus encephalomyelitis free countries may prohibit importation or transit through their territory, directly or indirectly, from countries considered infected with enterovirus encephalomyelitis of fresh meat of domestic and wild pigs.

Article 3.5.4.10.

When importing from enterovirus encephalomyelitis free countries, Veterinary Administrations should require, for fresh meat of pigs, the presentation of an international sanitary certificate attesting that the entire consignment of meat comes from animals:

1) which have been kept in a country free from enterovirus encephalomyelitis since birth;

2) slaughtered in an abattoir and found to be healthy before and after slaughter.

4.2.5.10. Recommendation

Sausage casings derived from pigs should not be imported from countries where Teschen disease occurs unless the casings are effectively treated under AQIS quarantine control.

4.2.6. Porcine Reproductive and Respiratory Syndrome (PRRS)

4.2.6.1. Description and aetiology

Porcine Reproductive and Respiratory Syndrome (PRRS) is a new disease that initially spread as a pandemic, and has become endemic in a number of countries throughout the world. It has been known by a number of names, including porcine epidemic abortion and respiratory disease (PEARS) and blue-eared pig disease. Clinical effects are extremely variable, and sub-clinical infections are common. The reproductive effects of the disease include stillborn, mummified or decomposing piglets, as well as infertility, including delayed returns to oestrus, persistent re-breeding, and persistent anoestrus. In boars, reproductive effects include loss of libido and reduction in semen quality. Systemic signs include anorexia, pyrexia, agalactia, lethargy and skin discolouration. Respiratory signs include laboured breathing and coughing (Done *at al* 1996).

4.2.6.2. World distribution

The disease was first recorded in the USA in the 1980s and has since been reported in Canada, Germany, The Netherlands, Belgium, Britain, Spain and other European and Far Eastern countries (Done *at al* 1996). It does not occur in Australia.

4.2.6.3. Natural and experimental hosts

PRRS is not known to occur in any other reservoir species. A preliminary report has suggested that migratory fowl can become infected and are therefore possible vectors (Done *at al* 1996).

4.2.6.4. Pathology

Pathological changes are variable, and it appears likely that less pathogenic strains of the virus as seen in Britain, cause less severe changes than more pathogenic strains from other parts of the world. There is no evidence of pathological change in the intestinal tract, although the systemic nature of the disease suggests that all tissues of infected pigs could contain virus (Done *at al* 1996). Skin discolouration suggests the potential presence of virus in hides.

4.2.6.5. Pathogen inactivation

PRRS virus survives relatively poorly in the external environment. Virus can survive for several years in deep frozen tissues, but only 1 month at 4^{o}C, 48hr at 37^{o}C, and less than 45 min at 56^{o}C. The half life for virus survival is reduced at a pH below 5 or above 7, but live virus may be recovered from carcass meat stored at 4^{o}C or below for 48 hours (Done *at al* 1996). Storage of casings at room temperature is therefore likely to lead to inactivation of the virus within a reasonable period of time.

4.2.6.6. Disease transmission

Transmission between pigs may be by nose-to-nose contact, or by aerosols. Virus may enter new herds by movement of pigs, and probably by air borne spread. Virus has been isolated from faecal samples, urine, and from the semen of experimentally infected boars, and it is possible that the disease may be spread by artificial insemination (Done *at al* 1996). Sub-clinical herd infections are common, and there is prolonged circulation of virus within infected herds (Done *et al* 1996).

A number of researchers have investigated the possibility of transmission of the virus in meat. All tissues assayed by Bloemraad *et al.* (1994) had comparatively low titres, with the maximum tire being $10^{5.0}$ $TCID_{50}$/gm in tonsil. No virus assays were conducted on intestine, but mesenteric lymph nodes had titres of approx $10^{3.0}$ $TCID_{50}$/gm in pigs killed 5 and 10 days after experimental infection. Bloemraad *et al.* consider that the sporadic recovery of virus from muscle tissues is most likely from residual blood plasma in capillary vessels.

Frey *et al* (1993) report a series of experiments in which they infectd pigs with both European and North American strains of PRRS virus, and assayed various tissues for the presence of virus. No virus was able to be recovered from poolled samples of ileum and large intestine.

Mengeling *et al*. (1995) infected 21 6-week old pigs with one of three strains of the virus and examined various tissues for the presence of virus after euthanasia at 3, 7, 14, 22, 35, 49 and 70 days post-infection. Virus was recovered from only one animal, and was found in the ham muscle of a pig slaughtered 7 days after infection. These authors also considered the virus recovered from muscle to be blood associated, rather than in muscle tissue.

After checking tissues from experimentally infected pigs at 7 and 14 days post-infection, Magar *et al* (1995) concluded that the chance of PRRS virus being transmitted through pork was low. Again, the virus was considered to be associated with residual blood in the tissues.

Frey *et al*. (1995) reported that virus was able to be recovered from only six of 1049 sample pools taken from commercially slaughtered pigs. The level of virus was judged very low, because most isolates were obtained only after multiple cell culture passages and re-isolation was not always successful.

Larochelle and Magar (1997) collected samples representative of 73 days slaughterings over a 7 month period, from four separate processing plants receiving pigs from herds where seropositivity was high. All samples were negative when tested for live virus and by PCR.

Despite the fact that an oral infectious dose has not been established, the very low virus titres in muscle tissues and the likelihood that virus is associated with blood, mean that the chance of virus persisting in stripped and treated sausage casings of porcine origin is extremely low.

4.2.6.7. Establishment requirements

The disease has spread rapidly throughout many parts of the world, where intensive pig husbandry is practised. There is no reason to doubt that it would easily become established in Australia, should susceptible pigs be exposed and become infected. The incidence of sub-

clinical herd infection, prolonged virus circulation, and probable aerogenic spread, have combined to make eradication difficult in other countries once the disease has become established.

4.2.6.8. Potential effects of disease establishment

Losses as a result of PRRS are extremely variable, both in extent and duration. It is important to distinguish between losses due to the disease in the epidemic and endemic phases of the disease. Major losses if the disease became endemic would relate to the on-going effects on reproductive efficiency.

4.2.6.9. OIE recommended safety precautions

The OIE Code makes no specific recommendations relating to the importation of sausage casings, with respect to the risk of introduction of PRRS.

4.2.6.10. Recommendation

There appears to be no record of the disease having been spread by meat or meat products. In addition, the literature reports that the virus is not present in pig intestinal tissues. This, combined with the belief that virus in meat is associated with residual blood in muscle, and the treatment applied to intestines in the preparation of casings, suggest that it is unlikely that the disease could be introduced into Australia by way of sausage casings. No particular restrictions appear to be warranted.

4.3. Other potentially serious exotic diseases.

4.3.1. Vesicular exanthema

The disease is important in the differential diagnosis of FMD in pigs. The causative agent is a calicivirus, and is closely related, or identical, to caliciviruses isolated from sea lions, whales and fish (Geering *et al* 1995). The disease has occurred mainly in the United States, and on one occasion in Iceland in 1955. The last recorded outbreak of the disease was in California in 1976 (Thomson 1994). Although the disease can be spread by ingestion of meat or meat products, the absence of the disease in recent times, and the greater controls on swill feeding of pigs, make the introduction of the disease to Australia in imported casings derived from pigs highly unlikely. No specific quarantine restrictions are warranted.

4.3.2. Porcine epidemic diarrhoea.

Porcine epidemic diarrhoea (PED) is a disease similar to transmissible gastroenteritis. It is caused by a coronavirus, closely related to TGE. The disease affects pigs of all ages, while TGE affects mainly young piglets. Mortality rates for young pigs can be up to 50% for piglets less than 10 days old, while TGE can have mortality approaching 100% in young animals. PED is however, more severe in older pigs than is TGE. Despite this, few older pigs die, although some remain unthrifty. The pathology of PED is less severe than for TGE.

Given the similarities between the epidemiology of TGE and PED, there would appear to be no reason to impose conditions on the import of sausage casings in respect of PED

4.3.3. Post Weaning multi-systemic wasting disease

Another recently recognised disease of quarantine concern to Australia is postweaning multi-systemic wasting syndrome (PMWS). Although no direct causal relationship has yet been proven, PMWS is associated with a porcine circovirus. Although a non-pathogenic circovirus is present as a contaminant of a pig kidney cell line, and has been shown to have infected pigs in many parts of the world, there is some evidence that the circovirus associated with PMWS differs from that virus. The precise aetiology of PMWS is unknown at present, and there may be a number of factors which contribute to the development of the disease. The disease mostly affects pigs of 4-6 weeks of age, although this can vary with the characteristics of the particular production unit (Harding and Clark 1997). Other estimates are that the age range for the disease is 6-14 weeks. Histopathological changes are found in the ileum, caecum and colon, indicating at least the possibility that virus may be present in casings derived from pigs, although there is to date no evidence of the disease being spread in meat or meat products. The age range of the disease being younger than the usual slaughter age for pigs, and the requirement for casings only to be derived from animals which have passed both ante- and post-mortem inspection, should be sufficient to guard against the introduction of this disease to Australia in casings derived from pigs.

5. Risk Management Strategies

5.1. Edible collagen casings (derived from hides and skins)

The extreme chemical and physical processes involved in the production of edible collagen casings derived from animal hides are sufficient to destroy all disease agents of concern, which are likely to be present in or on hides and skins. This risk assessment paper reveals no reason to change the existing conditions for the import of edible collagen casings, which are as follows:

> *A Quarantine Entry is required for all consignments <u>except</u> those which are imported as non-commercial consignments by mail or those which are imported as personal consignments with passenger's accompanied baggage.*
>
> *All consignments must be inspected on arrival and the following conditions apply.*
>
> *A Permit application is required prior to importation and can be approved by AQIS Regional Offices.*
>
> *Each consignment of collagen casings must be accompanied by a government veterinary certificate stating:*
>
> *1. The animal casings contain collagen material only and no intestinal derived material.*

2. The collagen used to make the casings is derived from hides and skins only and the casings have been commercially prepared.

5.2. Natural casings derived from intestines

As a general rule, imported sausage casings are considered to pose a relatively low risk of the introduction of exotic disease agents. In order to pose a risk of transmission through meat and meat products, the pathogen must be present in edible tissues, susceptible species must be carnivorous or omnivorous, and oral infection must be possible (MacDiarmid and Thompson 1997). Although the infective agent of a number of diseases of concern may potentially be present in imported sausage casings, it is unlikely that susceptible species will consume sufficient quantities of infected product to become infected. This risk is further reduced if the requirements recommended for individual diseases discussed above are followed.

The usual storage of sausage casings either heavily dry salted or in saturated brine means that casings do not present an attractive food source. Restrictions on swill feeding further reduce the likelihood of casings being eaten by susceptible species. It is possible that susceptible animals may be fed table scraps after incorporation of the casing into sausage. The relatively small proportion of casing in finished sausage means that it is unlikely that a susceptible animal would consume an infectious dose of disease agent through this pathway, especially after the processing involved in the sausage making and cooking process. The possibility of environmental contamination with waste from processing plants cannot be completely discounted. However, given the dilution factors operating in disposal of waste water and the controls on disposal of solid industrial waste, it would be unlikely for infection to be spread to susceptible Australian species via this pathway.

Despite the overall low risk of introduction of exotic disease through the importation of sausage casings, the potential effects of some of the diseases of concern are so devastating that some form of risk management procedures is warranted.

Risk management strategies that could be applied to the importation of casings are either:

- restricting the source of casings to countries where diseases of concern do not occur, and imposing restrictions on transport to ensure that contamination does not occur during storage or transport, which is the basis of the current system; or

- controlling treatment and use of casings after importation to ensure that any potential pathogens present in the imported material are effectively inactivated.

The latter system would have dual advantages. Firstly, it would allow access to casings from a wider range of countries, with potential savings in price. Secondly, it would help to reduce the incentive to "smuggle" these cheaper casings into Australia using falsified documents, while maintaining confidence in the safety of the imported product.

The major disadvantage associated with the latter system is the increased processing cost which would arise from the mandatory disinfection treatment. This would cause an unjustified increase in the cost of casings from countries free of the diseases of concern, and disadvantage

importers of these casings. By implementing a combination of the two options, Australia could obtain the advantages of the latter system, without detriment to existing trade arrangements.

Post-import treatment of sausage casings is undertaken by European Union countries, and by Japan. The European Union requires that imported sausage casings be treated with either NaCl for 30 days, bleached or scraped and dried. Salt and/or drying treatments are not sufficient to effectively eliminate all pathogens of quarantine concern to Australia. The Japanese system requires that casings are desalinated and soaked for 2 hours in 80 ppm chlorine solution. This system of decontamination has been used in Japan for 30 years, to process casings from countries with a range of exotic animal diseases. This treatment regime should be capable of removing the major exotic disease agents of concern to Australia.

Such a system could best be implemented through the use of quality assurance arrangements between AQIS and the sausage casing industry. Casings from countries where diseases of concern are present could be imported, subject to certification that the casings come only from animals which have passed both ante- and post-mortem inspection, and have not come from animals held in an area of the exporting country where any of the diseases of concern have occurred. The casings could then be consigned to AQIS approved treatment facilities and subject to appropriate treatment, under an Approved Quarantine Directive. The implementation of such a system could provide access to casings from a wider range of countries, while maintaining quarantine security.

6. Proposed Australian conditions for importation of casings.

Two sets of conditions follow. The first is based on the main risk control measure being the prohibition of import of casings from countries where diseases of concern are present. The second set of conditions depends on post-arrival treatment of imported casings to destroy disease agents of concern. AQIS will permit the importation of casings under either option subject to compliance with the requirements of that option. The operation of Option 2 would require that post-arrival treatment facilities be approved by AQIS, and operated by industry under Approved Quarantine Directive arrangements.

Desalination and soaking the casings in 80 ppm available chlorine solution should provide an effective treatment for all pathogens identified in this analysis as a significant quarantine risk.

6.1. Option 1.

These conditions apply to the importation of:

1. natural sausage casings derived from intestines of cattle, sheep, goats, and deer from countries which are free of foot and mouth disease, rinderpest and peste des petits ruminants; and

2. natural sausage casings derived from intestines of pigs from countries that are free of foot and mouth disease, rinderpest, classical swine fever, African swine fever, Teschen disease and swine vesicular disease.

1. DOCUMENTATION

1.1 Permission to import the product into Australia must be obtained in writing from the Australian Quarantine and Inspection Service (AQIS), prior to the product first being exported.

1.2 Each consignment must be accompanied by a Permit (or copy of a Permit) issued in Canberra and the prescribed certification in Section 3; and will require on arrival, a Quarantine Entry issued by AQIS at the port of entry.

1.3 Each application to the Director for permission to import must include the following details:

* country of export

* name and identification/veterinary control number of producing establishment

* species of origin

* product type

* full details of any process of manufacture the casings have been subjected to.

Product type exported must correspond exactly to the product shown on the import permit issued in relation to the consignment.

1.4 Each application will be assessed on the above criteria as well as any other criterion that is considered relevant by the Director. This may include a country's health status with regard to diseases not listed in these guidelines and standards of meat inspection services and export establishments.

1.5 Each consignment must be accompanied by a Sanitary Certificate that conforms to the Office International des Epizooties (OIE) International Animal Health Code *Model Certificate No. 4*. The certificate must be signed by an *Official Veterinarian* of the country of export and each page of the certificate stamped with an official stamp.

Under ***IV. Attestation of wholesomeness*** the Sanitary Certificate must contain detail of the certifications listed in Section 2 of this document**.**

2. CERTIFICATION

Each consignment must be accompanied by a Sanitary Certificate signed by an *Official Veterinarian*. The Sanitary Certificate must conform to the Office International des Epizooties (OIE) International Animal Health Code *Model Certificate No. 4*. and must attest, under ***IV. Attestation of wholesomeness***, that:-

2.1 The casings were derived from animals originating in and slaughtered in the exporting country.

2.2 The casings are of ovine, bovine, cervine, caprine or porcine origin*.

**[Note: Delete those not applicable.]*

2.3 The animals from which the casings were derived were slaughtered on the following dates.....

2.4 The animals from which the casings were derived were slaughtered at the following approved establishments:.....*

2.5 The casings for export were prepared and/or stored at the following approved establishments:.....*

*[*Note: Provide identification/veterinary control number(s) of the establishments.*]

2.6 The animals from which the casings were derived were subjected to ante and post-mortem veterinary inspection and were found to be free from contagious or infectious disease.

2.7 The country of origin of porcine casings is officially free from foot and mouth disease, rinderpest, African swine fever, classical swine fever, swine vesicular disease and Teschen disease.

2.8 The country of origin of bovine, ovine, caprine or cervine casings is officially free from foot and mouth disease, rinderpest and peste des petits ruminants.

2.9 The country/zone of origin of the meat is a ***BSE free country/zone.***

OR

The country/zone of origin of the meat is a ***BSE provisionally free country/zone,*** the meat and meat product is derived from cattle which have not been exposed to meat-and-bone meal imported from a *country* or *zone* with a high incidence of BSE, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants is banned, and

iii) if BSE has been reported in the country/zone the consignment

either

does not include bovine brains, spinal cords or protein derived from them from cattle over 30 months of age and born before the ban on feeding ruminant derived meat-and-bone meal to ruminants

or

has been treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents.

OR

The country/zone of origin of the meat is a ***country/zone with a low incidence of BSE***, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants has been banned, and

iii) either

the consignment does not include bovine brains, eyes, spinal cord and distal ileum, or protein products derived from them, from cattle over 6 months of age and born before the ban on feeding ruminant derived meat-and-bone meal to ruminants

or

has been treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents, and

OR

The country/zone of origin of the meat is a ***BSE high incidence country/zone***, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants has been banned, and

iii) the cattle from which the meat destined for export originates:

- were permanently identified enabling them to be traced back to the dam and herd of origin;

- were not the offspring of BSE suspect or confirmed females; and

either

were born after the date of the ban on feeding ruminant-derived meat-and-bone meal to ruminants;

or

were born and remained in herds in which no case of BSE had been confirmed during the preceding seven years.

iv) a system is in operation enabling the fresh meat and meat products destined for export to be traced back to the establishment from which they are derived;

v) the *meat* and *meat products* did not contain brains, eyes, spinal cords, tonsils, thymus, spleen, intestine, dorsal root ganglia, trigeminal ganglia, bones nor nervous and lymphatic tissue exposed during the deboning process, nor products derived from them, from cattle over 6 months of age.

2.10 The consignment does not contain casings derived from sheep and goats over 12 months of age originating from *countries* or *zones* not considered free from scrapie.

2.11 Each establishment at which the animals from which the casings were derived were slaughtered had current AQIS approval and met

a a standard of construction equivalent to that set down in the "Australian Standard for Construction of Premises Processing Animals for Human Consumption" (1995)

b a standard of hygienic production equivalent to that set down in the "Australian Standard for Hygienic Production of Meat for Human Consumption" (2nd edition) (AS4461:1997)

2.12 Each establishment where the casings were prepared and stored had current AQIS approval and met:

a a standard of construction equivalent to that set down in the "Australian Standard for Construction of Premises Processing Meat for Human Consumption" (1995)

b a standard of hygienic production equivalent to that set down in the "Australian Standard for Hygienic Production of Meat for Human Consumption" (2nd edition) (AS4461:1997)

2.13 The casings were packed:

a so that each packing container contains casings derived from a single species of animal only;

b so that they were not exposed to contamination before export;

c in clean, new or disinfected packing containers; and

d so that the identification/veterinary control number* of the establishment where the casings were packed was readily visible on the outer wrapping or package.

[*Note: Numbers must not be able to be removed without damage.*]

3 IMPORTERS/AGENTS RESPONSIBILITIES

3.1 The casings must be transported to Australia in clean, sealed containers.

3.2 The casings shall be stored for no less than thirty days after the slaughter of the animals from which they were derived before release from quarantine in Australia.

6.2. Option 2.

These conditions apply to the importation of natural sausage casings derived from intestines of cattle, sheep, goats, deer and pigs.

1. DOCUMENTATION

1.1 Permission to import the product into Australia must be obtained in writing from the Australian Quarantine and Inspection Service (AQIS), prior to the product first being exported.

1.2 Each consignment must be accompanied by a Permit (or copy of a Permit) issued in Canberra and the prescribed certification in Section 3; and will require on arrival, a Quarantine Entry issued by AQIS at the port of entry.

1.3 Each application to the Director for permission to import must include the following details:

* country of export;

* name and identification/veterinary control number of producing establishment;

* species of origin;

* product type; and

* full details of any process of manufacture the casings have been subjected to.

Product type exported must correspond exactly to the product shown on the import permit issued in relation to the consignment.

1.4 Each application will be assessed on the above criteria as well as any other criterion that is considered relevant by the Director. This may include a country's health status with regard to diseases not listed in these guidelines and standards of meat inspection services and export establishments.

1.5 Each consignment must be accompanied by a Sanitary Certificate that conforms to the Office International des Epizooties (OIE) International Animal Health Code *Model Certificate No. 4*. The certificate must be signed by an *Official Veterinarian* of the country of export and each page of the certificate stamped with an official stamp.

Under ***IV. Attestation of wholesomeness*** the Sanitary Certificate must contain detail of the certifications listed in Section 2 of this document.

2. CERTIFICATION

Each consignment must be accompanied by a Sanitary Certificate signed by an *Official Veterinarian*. The Sanitary Certificate must conform to the Office International des Epizooties (OIE) International Animal Health Code *Model Certificate No. 4*. and must attest, under ***IV. Attestation of wholesomeness***, that:-

2.1 The casings were derived from animals originating in and slaughtered in the exporting country.

2.2 The casings are of ovine, bovine, cervine, caprine or porcine origin*.

**[Note: Delete those not applicable.]*

2.3 The animals from which the casings were derived were slaughtered on the following dates.....

2.4 The animals from which the casings were derived were slaughtered at the following approved establishments:.....*

2.5 The casings for export were prepared and/or stored at the following approved establishments:.....*

*[*Note: Provide identification/veterinary control number(s) of the establishments.*]

2.6 The animals from which the casings were derived were subjected to ante and post-mortem veterinary inspection and were found to be free from contagious or infectious disease.

2.7 The country/zone of origin of the meat is a ***BSE free country/zone.***

OR

The country/zone of origin of the meat is a ***BSE provisionally free country/zone,*** the meat and meat product is derived from cattle which have not been exposed to meat-and-bone meal imported from a *country* or *zone* with a high incidence of BSE, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants is banned, and

iii) if BSE has been reported in the country/zone the consignment

either

does not include bovine brains, spinal cords or protein derived from them from cattle over 30 months of age and born before the ban on feeding ruminant derived meat-and-bone meal to ruminants

or

has been treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents.

OR

The country/zone of origin of the meat is a ***country/zone with a low incidence of BSE***, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants has been banned, and

iii) either

the consignment does not include bovine brains, eyes, spinal cord and distal ileum, or protein products derived from them, from cattle over 6 months of age and born before the ban on feeding ruminant derived meat-and-bone meal to ruminants

or

has been treated in accordance with *Code* (Appendix 4.3.3.1.) to inactivate BSE infective agents, and

OR

The country/zone of origin of the meat is a ***BSE high incidence country/zone***, and

i) affected animals and the last progeny of affected females born within 2 years prior to or after the onset of clinical symptoms, have been slaughtered and completely destroyed, and

ii) the feeding of ruminant-derived meat-and-bone meal to ruminants has been banned, and

iii) the cattle from which the meat destined for export originates:

- were permanently identified enabling them to be traced back to the dam and herd of origin;

- were not the offspring of BSE suspect or confirmed females; and

  either

  were born after the date of the ban on feeding ruminant-derived meat-and-bone meal to ruminants;

  or

  were born and remained in herds in which no case of BSE had been confirmed during the preceding seven years.

iv) a system is in operation enabling the fresh meat and meat products destined for export to be traced back to the establishment from which they are derived;

v) the *meat* and *meat products* did not contain brains, eyes, spinal cords, tonsils, thymus, spleen, intestine, dorsal root ganglia, trigeminal ganglia, bones nor nervous and lymphatic tissue exposed during the deboning process, nor products derived from them, from cattle over 6 months of age.

2.8 The consignment does not contain casings derived from sheep and goats over 12 months of age originating from *countries* or *zones* not considered free from scrapie.

2.9 Each establishment at which the animals from which the casings were derived were slaughtered had current AQIS approval and met

a a standard of construction equivalent to that set down in the "Australian Standard for Construction of Premises Processing Animals for Human Consumption" (1995)

b a standard of hygienic production equivalent to that set down in the "Australian Standard for Hygienic Production of Meat for Human Consumption" (2nd edition) (AS4461:1997)

2.10 Each establishment where the casings were prepared and stored had current AQIS approval and met

a a standard of construction equivalent to that set down in the "Australian Standard for Construction of Premises Processing Meat for Human Consumption" (1995)

b a standard of hygienic production equivalent to that set down in the "Australian Standard for Hygienic Production of Meat for Human Consumption" (2nd edition) (AS4461:1997).

2.11 The casings were packed:

a so that each packing container only contains casings derived from only a single species of animal;

b so that they were not exposed to contamination before export;

c in clean, new or disinfected packing containers; and

d so that the identification/veterinary control number* of the establishment where the casings were packed was readily visible on the outer wrapping or package.

[*Note: Numbers must not be able to be removed without damage.*]

3 IMPORTERS/AGENTS RESPONSIBILITIES

3.1 The casings must be transported to Australia in clean, sealed containers.

3.2 The casings shall be stored for no less than thirty days after the slaughter of the animals from which they were derived before release from quarantine in Australia.

3.3 Upon arrival in Australia, the casings shall be immediately removed to an approved post-arrival treatment facility, where they will be desalinated and soaked for 2 hours in chlorine solution maintained at a level of at least 80 ppm of free available chlorine.

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