Supplementary import risk analysis: Sausage casings of bovine and porcine origin

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Supplementary Import risk analysis: Sausage casings of bovine and porcine origin

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Approved for Public Consultation

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1. Executive summary

This import risk analysis examines the biosecurity risks associated with the international trade in natural sausage casings of bovine and porcine origin, that have been prepared by methods which are standard operating procedures in much of the international casing industry.

Natural sausage casings are made from the intestines of cattle and pigs that have passed ante- and post-mortem inspection. The intestines are subject to a number of processing steps that remove gut contents and different tissue layers and the casings are subsequently stored at room temperature for a minimum of 30 days in dry salt or saturated brine.

Hog casings produced from the small intestine consist of the submucosa tissue layer, with complete removal of all lymphoid tissue (Peyer's patches). Hog casings made from the large intestine contain at least some of the major intestinal tissue layers.

Beef casings also contain most of the intestinal tissue layers as only part of the mucosa and lymphoid tissue is removed during processing. From a technological viewpoint the only part of the intestinal tract that is not utilised is the ileum[1] due to its aberrant shape.

Disease-causing organisms associated with bovine and porcine intestines were identified from previously peer-reviewed Ministry for Primary Industries risk analyses. Of the 10 disease-causing organisms examined in this risk analysis, 9 were identified from a risk analysis of cattle and pig meat, with the other organism identified from a single species risk analysis of pig meat.

The 10 disease-causing organisms examined in this risk analysis are:

- African swine fever virus
- Aujeszky's disease virus
- Classical swine fever virus
- Foot and mouth disease virus
- Porcine reproductive and respiratory syndrome virus
- Swine vesicular disease virus
- *Bacillus anthracis*
- *Salmonella* spp.
- *Echinococcus granulosus*
- The agent of bovine spongiform encephalopathy

Of these 10 organisms, one is assessed to be a risk; classical swine fever virus (hog casings) and risk management options are presented.

[1] In addition to the technological exclusion of the bovine ileum for beef casing production, the OIE recommends not to trade the ileum if derived from animals originating from countries with a BSE controlled or undetermined risk. This is because the ileum could contain BSE prions that if consumed by humans may cause variant Creutzfeld-Jakob disease.
See OIE Article 11.4.14, http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_bse.htm

The following measures could be considered to effectively manage the risk in imported hog casings.

Option 1

Hog casings could be imported only from countries, zones or compartments recognised by New Zealand as free from CSFV.

Option 2

For the inactivation of CSFV in casings of pigs, the following procedures could be used: salting for at least 30 days either with phosphate supplemented dry salt or saturated brine (Aw < 0.80) containing 86.5% NaCl, 10.7% Na_2HPO_4 and 2.8% Na_3PO_4 (weight/weight/weight), and kept at a temperature of greater than 20 °C during this entire period.

2. Introduction

This import risk analysis examines the biosecurity risks associated with imports of sausage casings prepared from the intestines of cattle and pigs. Disease-causing organisms are identified from previously peer-reviewed Ministry for Primary Industries (MPI) risk analyses of cattle and pig meat (MPI 2006; MPI 2014). Only those disease-causing organisms requiring risk management measures are considered further. This risk analysis supplements MPI's previous risk analysis for small ruminant sausage casings (MPI 2010).

3. Scope and commodity definition

This document assesses the biosecurity risks associated with natural sausage casings of porcine and bovine origin, prepared by methods which are standard operating procedures in much of the international casings industry.

The commodities under consideration are hog casings consisting of the submucosal tissue layer of the small intestine and casings made from the large intestine which contain all tissue layers. For beef casings this consists of all intestinal layers of the entire intestinal tract with the exclusion of the ileum. Casings are produced from animals that have passed ante- and post-mortem inspection according to the OIE's *Terrestrial Animal Health Code* (hereafter referred to as the *Code*) chapter 6.2 (OIE 2013). After cleaning and scraping, the casings have been stored at room temperature in either dry salt or saturated brine. As an alternative treatment the dry salt or saturated brine may be supplemented with sodium phosphates (E339) for a minimum of 30 days. The pH of casing stored in dry salt and saturated brine is between 7.5 and 8, and if supplemented with phosphate the pH increases to approximately 10 (Wijnker *et al.* 2009).

The entire intestinal tract of pigs is used for the production of casings. With the small intestines (duodenum, jejunum, ileum), bung (caecum), large intestines (colon ascendens and transversum), after end (colon descendens) and fat end (rectum) being especially utilised (Wijnker 2009).

The intestinal tract of cattle is also used entirely for the production of beef casings with the exception of the ileum. Its shape differs too much from the jejunum to produce the classic beef rounds and is therefore removed prior to the cleaning process and destroyed. Beef casings are produced from the small intestines including the duodenum and jejunum (beef rounds) and the caecum and from the large intestines, including the entire colon (beef middles). Beef casings are also produced from the urinary bladder and the oesophagus (weasand) (Ockerman and Hansen, 2000; Wijnker *et al*. 2008; European Natural Sausage Casings Association 2013).

The basic anatomy and function of the intestinal tract is very similar across the species used for casing manufacture (Wijnker 2009). The intestinal wall is composed of four main layers (Figure 1). The outermost layer is the tunica serosa. The next layer, the tunica muscularis, consists of two layers of smooth muscle. Beneath these layers is the tunica submucosa, which is comprised of a network of collagen fibres, elastin and blood vessels. The innermost layer, which lines the lumen of the intestine, is the tunica mucosa. Lymphoid tissue is embedded in the mucosa. This lymphoid tissue is most prominent in the ileum, where it is aggregated into Peyer's patches (Wijnker 2009).

Once pulled from the abdominal cavity the intestines have their gut contents removed. The intestines are then processed through a number of steps, which involves equipment such as;

conveyor belts, holding (soak) tanks, water sprinklers, rollers and scrapers, which facilitate the removal of unwanted tissue. Water in the holding tanks is usually heated to a temperature of 40 °C to assist cleaning. After processing, the casings are placed in cold water or a salt brine tank to wash away any residual blood and prevent the growth of bacteria (European Natural Sausage Casings Association 2013).

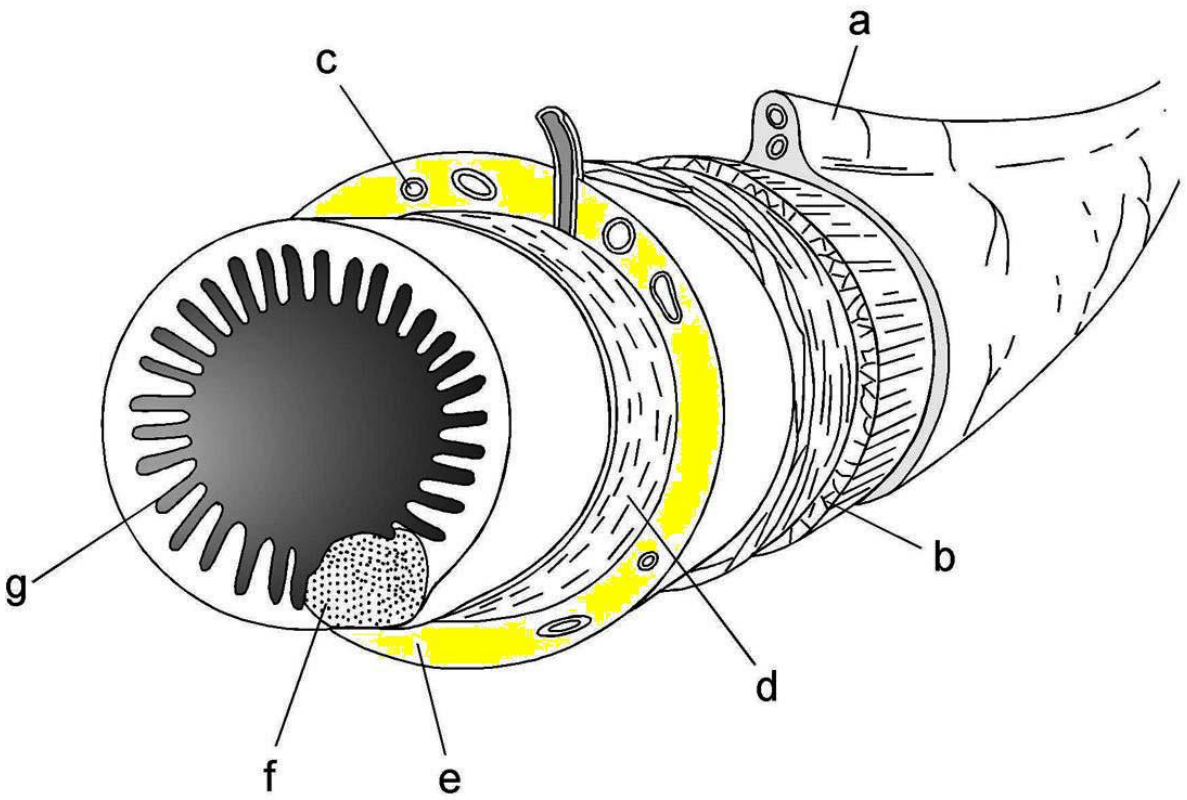
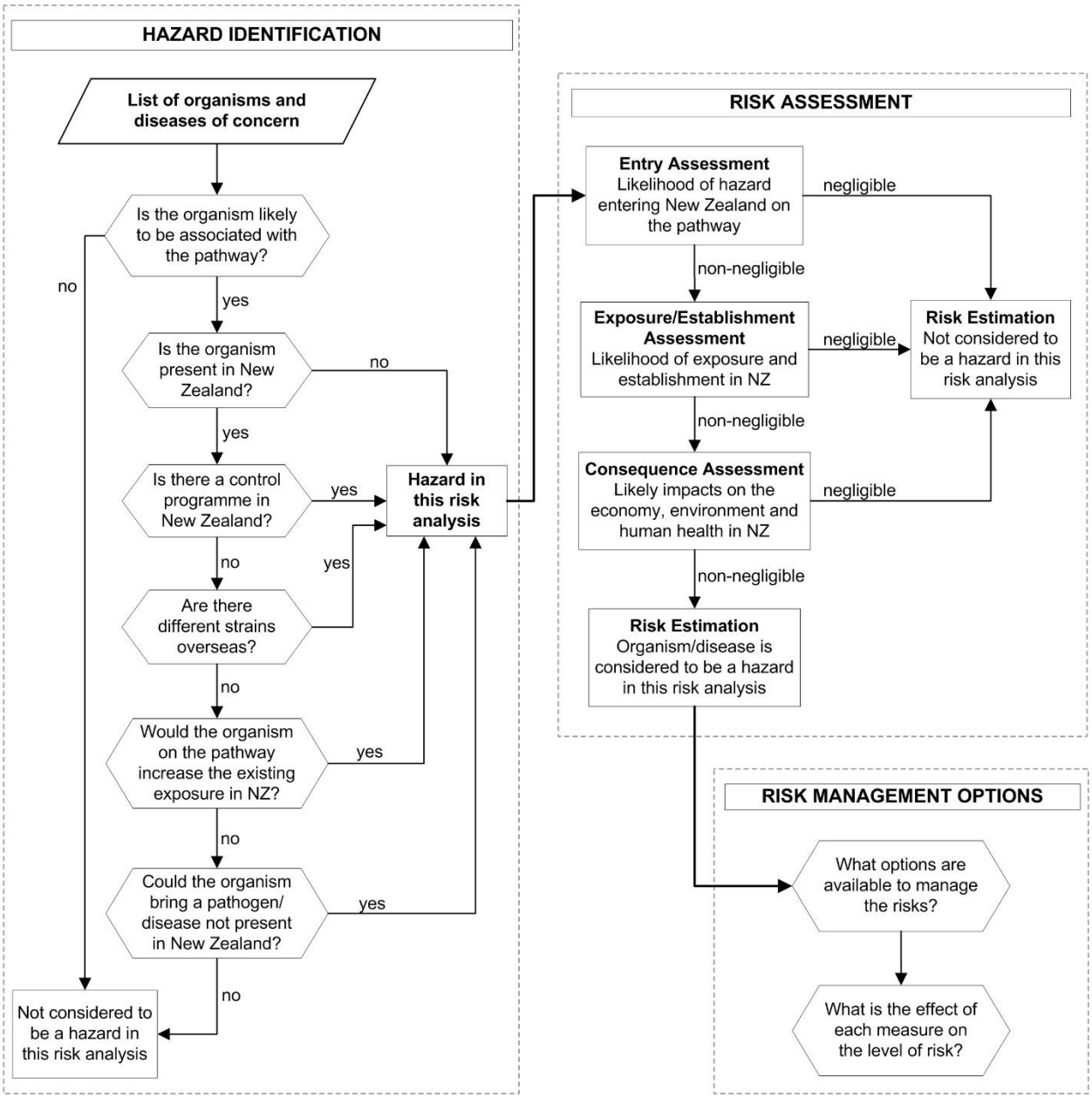


Figure 1: Schematic diagram of a small intestine showing **(a)** mesentery and serosa; **(b)** inner and outer muscle layers; **(c)** submucosal blood vessels; **(d)** muscularis mucosae; **(e)** submucosa; **(f)** lymphoid nodule (Peyer's patch); **(g)** tunica mucosa (villus and crypt layers). In hog casings the tunica mucosa, the muscularis, the serosa and Peyer's patches are removed during processing, so the natural casing consists of only the submucosa. In beef casings, all intestinal layers remain after cleaning (adapted from Koolmees *et al.* 2004).

4. Risk analysis methodology

The methodology used in this risk analysis follows the guidelines as described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1* (MPI 2006b) and in Section 2 of the *Code* (OIE 2013). The risk analysis process used by the *Code* and adopted by the Ministry for Primary Industries is summarised in Figure 2 (overleaf).

Figure 2. The risk analysis process.



4.1. PRELIMINARY LIST OF HAZARDS (ORGANISMS OF POTENTIAL CONCERN)

The first step in the risk analysis process is to compile a list of pathogens known to infect cattle and pigs. This list is subjected to the application of specific criteria to eliminate those that are not organisms of concern. The remaining organisms are then subjected to the process of hazard identification.

4.2. HAZARD IDENTIFICATION

Hazard identification includes formal identification of the organism, whether it is associated with an OIE listed disease, its New Zealand status, and a discussion on the relevant aspects of the epidemiology and characteristics of the organism. The hazard identification section is concluded by an assessment of whether or not the organism is identified as a hazard.

All hazards are subjected to risk assessment.

4.3. RISK ASSESSMENT

Risk assessment consists of:

a) *Entry assessment*: The likelihood of a hazard (pathogenic organism) being imported with the commodity.

b) *Exposure assessment*: Describes the biological pathway(s) necessary for exposure of susceptible animals or humans in New Zealand to the hazard. Further, a qualitative estimation of the probability of the exposure occurring is made.

c) *Consequence assessment*: Describes the likely potential consequences of entry, exposure and establishment or spread of an imported hazard.

d) *Risk estimation*: An estimation of the risk posed by the hazard associated with importing sausage casings. This is based on the entry, exposure and consequence assessments. If the risk estimate is assessed to be non-negligible, then the hazard is assessed to be a risk, and risk management measures may be justified to reduce the level of risk to an acceptable level.

Not all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of entry is negligible for a hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises when the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of susceptible species being exposed is negligible, or when both entry and exposure are non-negligible but the consequences of introduction are assessed to be negligible.

4.4. RISK MANAGEMENT OPTIONS

For each organism assessed to be a risk the options available for managing that risk are presented. Where the *Code* lists recommendations for the management of a risk, these are described alongside options of similar, lesser or greater stringency, where available. In addition to the options presented, unrestricted entry or prohibition may also be considered. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted.

As obliged under Article 3.1 of the World Trade Organization's Agreement on the Application of Sanitary and Phytosanitary (SPS) measures, the measures adopted in this IHS will be based on international standards, guidelines and recommendations where they exist except as otherwise provided for under Article 3.3. That is, measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate.

4.5. RISK COMMUNICATION

In drafting an IHS, MPI analyses the options available and proposes measures for the effective management of identified risks. These are then presented in a draft IHS that is released together with a risk management proposal that summarises the options analysis, the rationale for the proposed measures and a link to the risk analysis. The package of documents is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to these documents are reviewed before a final IHS is issued.

5. Preliminary list of hazards

The first step in the risk analysis process is the identification of agents of concern and the collation of these into a list of pathogens that may be associated with the commodity under consideration.

Following the completion of the *Import risk analysis: Meat and meat products from ruminants and pigs* (MPI 2014), only disease-causing organisms associated with cattle and pigs that required risk management measures are to be assessed. This is because the commodity definition of meat used in this previous risk analysis was "all edible parts of an animal". Therefore, the identification of disease-causing agents associated with intestinal tissues has been completed. In addition to these identified pathogens, porcine reproductive and respiratory syndrome virus will also be included in the list, as it was assessed in a separate single species risk analysis (MPI 2006).

Organisms in Table 1 are classified as requiring further assessment if they are:

- Exotic to New Zealand; and,
- Require risk management measures as described in MPI (2006; 2014).

Organisms listed in Table 1 do not require further assessment if they are:

- Not assessed as requiring risk management measures as described in MPI (2014); or,
- The organism is not associated with the intestinal tract.

Table 1: Exotic organisms of concern that may be present in beef and hog casings (adapted from MPI 2014).

Organism	OIE List	Zoonotic	Relevant species infected	Transmission	Assessed to be a risk in edible tissue[2]	Requires further assessment
Viruses						
Akabane and related simbu viruses	No	No	Cattle	Vector-borne	No	No
Adenovirus	No	No	Pigs	Direct contact	No	No
African swine fever virus	Yes	No	Pigs	Direct contact, ingestion, vector-borne	Yes	Yes
Alcelaphine herpesvirus 1 (malignant catarrhal fever)	No	No	Cattle	Close contact	No	No
Aujeszky's disease virus	Yes	No	Pigs and cattle	Direct oral-nasal contact, ingestion	Yes	Yes
Blue eye disease virus	No	No	Pigs	Direct contact	No	No
Bluetongue virus	Yes	No	Cattle	Vector-borne	No	No
Borna disease virus	No	Yes	Exceptionally cattle	Direct contact	No	No
Bovine ephemeral fever virus	No	No	Cattle	Vector-borne	No	No
Bovine herpesvirus 5, 1.1 and 1.2a strains (IBR/IPV)	Yes	No	Cattle	Direct contact	No	No
Bovine parvovirus	No	No	Cattle	Faecal-oral	No	No
Bovine rhinovirus	No	No	Cattle	Direct contact	No	No
Bovine viral diarrhoea virus	Yes	No	Cattle, pigs	Direct contact, ingestion (pigs)	No	No
Bungowannah virus	No	No	Pigs	Direct contact, ingestion?	No	No
Classical swine fever virus (hog cholera)	Yes	No	Pigs	Direct contact, ingestion	Yes	Yes
Epizootic haemorrhagic disease (EHD) virus (including Ibaraki virus)	Yes	No	Cattle	Vector-borne	No	No
Equine encephalitis viruses (eastern, western, Venezuelan)	Yes	Yes	Pigs	Vector-borne	No	No
Foot and mouth disease virus	Yes	No	Pigs	Close contact, aerosol, ingestion	Yes	Yes

[2] See MPI's 2014 *Import risk analysis*: Meat and meat products from ruminants and pigs. Draft for public consultation. [Online] Available from: http://www.biosecurity.govt.nz/files/regs/imports/risk/meat-and-meat-products-from-ruminants-and-pigs-feb-2014.pdf [Accessed 3th March 2014].

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Assessed to be a risk in edible tissue	Requires further assessment
Viruses (continued)						
Hendra virus	No	Yes	Pigs (experimentally)	Direct contact with fruit bats	No	No
Influenza viruses	No	No	Pigs	Close contact	No	No
Japanese encephalitis virus	Yes	Yes	Pigs	Vector-borne	No	No
Jembrana disease virus	No	No	Cattle	Mechanical by biting insects	No	No
Kunjin virus	No	Yes	Pigs	Vector-borne	No	No
Louping ill and related viruses (tick borne encephalitis)	No	Yes	Cattle and pigs	Vector-borne	No	No
Lumpy skin disease virus	Yes	No	Cattle	Mechanical by biting insects	No	No
Menangle virus	No	Yes	Pigs	Contact with fruit bats	No	No
Murray Valley encephalitis virus	No	Yes	Pigs	Vector-borne	No	No
Nipah virus	Yes	Yes	Pigs	Exposure to fruit bats, direct contact, ingestion	No	No
Porcine haemagglutinating encephalomyelitis virus	No	No	Pigs	Direct contact	No	No
Porcine epidemic diarrhoea virus	No	No	Pigs	Faecal-oral	No	No
Porcine respiratory coronavirus	No	No	Pigs	Direct contact, ingestion?	No	No
Porcine reproductive and respiratory syndrome virus	Yes	No	Pigs	Direct contact, ingestion (experimentally)	Yes[3]	Yes
Rabies virus	Yes	Yes	Cattle and pigs	Direct contact (bite), ingestion?	No	No
Rift Valley fever virus	Yes	Yes	Cattle	Vector-borne	No	No
Ross River and Barmah Forest viruses	No	Yes	Cattle and pigs	Vector-borne	No	No
Swine pox virus	No	No	Pigs	Mechanical arthropod vectors	No	No
Swine vesicular disease virus	No	No	Pigs	Close contact, ingestion	Yes	Yes

[3]See MPI (2006). *Import risk analysis*: Porcine reproductive and respiratory syndrome (PRRS) virus in pig meat. [Online] Available from: http://www.biosecurity.govt.nz/files/regs/imports/risk/prrs-risk-analysis.pdf [Accessed 4th December 2013].

Teschovirus serotype 1	No	No	Pigs	Ingestion, aerosol	No	No

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Assessed to be a risk in edible tissue	Requires further assessment
Viruses (continued)						
Transmissible gastroenteritis virus	Yes	No	Pigs	Ingestion	No	No
Vesicular exanthema of swine virus	No	No	Pigs	Ingestion	No	No
Vesicular stomatitis virus	Yes	Yes	Cattle and pigs	Vector-borne, direct contact	No	No
West Nile fever virus	Yes	Yes	Cattle and pigs	Vector-borne	No	No
Bacteria including *Mycoplasma spp.*						
Bacillus anthracis	Yes	Yes	Cattle and pigs	Ingestion of spores	Yes	Yes
Borrelia burgdorferi	No	Yes	Cattle	Vector-borne	No	No
Borrelia theileri	No	No	Cattle	Vector-borne	No	No
Brucella melitensis, B. abortus, B. suis	Yes	Yes	Cattle and pigs	Direct contact, ingestion	Yes	No[4]
Burkholderia pseudomallei	No	Yes	Cattle and pigs	Contact with contaminated environment	No	No
Exotic Salmonella spp. e.g. Salmonella abortus ovis, S. Dublin, S. Typhimurium DT 104	No	Yes	Cattle	Faecal-oral	Yes	Yes
Francisella tularensis	Yes	Yes	Cattle and pigs	Vector-borne, ingestion	No	No
Leptospira spp.	No	Yes	Cattle	Ingestion, cut skin	No	No
Mycobacterium bovis	Yes	Yes	Cattle	Direct contact, ingestion	No	No
Mycoplasma hyosynoviae	No	No	Pigs	Direct contact	No	No
Mycoplasma mycoides Subsp. *mycoides SC*	Yes	No	Cattle	Direct contact, ingestion of lung tissue?	No	No
Mycoplasma bovis (and other exotic Mollicutes of cattle)	No	Various	Cattle	Direct contact, ingestion (milk)	No	No
Pasteurella multocida B and E	Yes	No	Cattle and pigs	Close contact	No	No
Protozoal parasites						
Babesia bovis, B. bigemina	Yes	No	Cattle	Tick-borne	No	No

[4] See OIE Article 8.4.2. Which lists the digestive tract as a safe commodity with regard to *Brucella* spp. http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_bovine_brucellosis.htm

Besnoitia besnoiti, B. caprae	No	No	Cattle	Haematophagous insects, ingestion	No	No

Table 1 (continued)

Organism	OIE List	Zoonotic	Species infected	Transmission	Assessed to be a risk in edible tissue	Requires further assessment
Protozoal parasites (continued)						
Theileria spp. (sheep, deer and cattle species)	No	No	Cattle	Tick-borne	No	No
Sarcocystis hominis	No	Yes	Cattle	Ingestion	No	No
Sarcocystis suihominis	No	Yes	Cattle	Ingestion	No	No
Trypanosoma spp. (Tsetse transmitted)	Yes	No	Cattle	Vector-borne	No	No
Trypanosoma evansi	Yes	No	Cattle	Haematophagous insects	No	No
Rickettsial and Chlamydial organisms						
Anaplasma marginale, A. centrale, A. caudatum	Yes	No	Cattle	Tick-borne	No	No
Chlamydophila abortus	Yes	Yes	Cattle	Ingestion (foetal membranes and fluid)	No	No
Coxiella burnetii	Yes	Yes	Cattle	Aerosol, vector-borne, ingestion	No	No
Ehrlichia ruminantum	Yes	No	Cattle	Vector-borne	No	No
Other *Ehrlichia* spp. e.g. *E. chaffeensis*	No	Yes	Cattle	Vector-borne	No	No
Arthropods						
Warble fly	No	No	Cattle	Fly	No	No
Internal parasites						
Echinococcus granulosus	Yes	Yes	Cattle, pigs	Ingestion	Yes	Yes
Exotic *Trichinella* species	No	Yes	Cattle, pigs	Ingestion	Yes	No[5]
Cysticercus cellulosae	Yes	Yes	Pigs	Ingestion	No	No
Cysticercus bovis	No	Yes	Cattle	Ingestion	No	No
Coenurus cerebralis	No	Yes	Cattle, pigs	Ingestion of viable cyst	Yes	No[6]
Prions						

[5] Infectious larvae only develop in striated muscle of infected animals (Gottstein *et al.* 2009). The infectious stage of this organism's lifecycle is not associated with the commodity.

[6] The infectious cysts (coenurus) can only develop and survive in the brain and spinal cord of cattle and pigs (Radostits *et al.* 2007). The infectious stage of this organism's lifecycle is not associated with the commodity.

Bovine spongiform encephalopathy	Yes	Yes	Cattle	Ingestion	Yes	Yes

Based on the above, the following organisms are identified as requiring further assessment.

Viruses

African swine fever virus

Aujeszky's disease virus

Classical swine fever virus

Foot and mouth disease virus

Porcine reproductive and respiratory syndrome virus

Swine vesicular disease virus

Bacteria

Bacillus anthracis

Salmonella spp.

Internal parasites

Echinococcus granulosus

Prions

The agent of bovine spongiform encephalopathy

The listed disease-causing organisms will be assessed in groups determined by the species they infect. Those that infect both pigs and cattle will be assessed first, followed by those that infected only pigs and then only cattle.

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6. Bacillus anthracis

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Bacillus anthracis is a bacterium belonging to the family *Bacillaceae* and genus *Bacillus* (De Vos and Turnbull 2004).

6.1.2. OIE list

Anthrax is an OIE listed disease of multiple species.

6.1.3. New Zealand status

Anthrax is listed as an exotic notifiable disease (Unwanted Organisms Register 2014). The first reported case of Anthrax in New Zealand was in 1895 and it was last diagnosed in 1954 (Gill 1993).

6.1.4. Epidemiology

Anthrax is a bacterial disease primarily of herbivores, although all mammals, including humans and some avian species are susceptible (OIE 2012). Domesticated animals have varying susceptibility to disease with sheep being the most susceptible followed by cattle, goats, horses, pigs, dogs and cats (MacDiarmid and Thompson 1997).

Natural infection of animals usually occurs by the ingestion of spores in meat or soil and other routes of infection include spores entering the body through cutaneous lesions, insect bites and, rarely, inhalation (De Vos and Turnbull 2004).

Once within the body the most common course of disease is septicaemia followed by death (MacDiarmid and Thompson 1997). Vegetative cells of *B. anthracis* do not survive well outside their host. In contrast, spores that form following exposure to oxygen are very resistant and can survive for a considerable time in the environment (De Vos and Turnbull 2004).

Pigs are more resistant to anthrax compared to herbivores and large quantities of infected meat need to be consumed to initiate infection. Although, because of the feeding behaviour of pigs, they can be exposed to much higher levels of anthrax compared to herbivores and mass mortalities have been recorded (De Vos and Turnbull 2004).

The OIE (2014) considers that "there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs".

6.1.5. Hazard identification conclusion

Anthrax is a zoonotic disease that is present in a number of countries. It is capable of causing severe disease and can be transmitted to susceptible mammal species through the ingestion of contaminated animal products. As anthrax is capable of forming spores, and as other spore forming bacteria have been shown to survive commercial processing (Wijnker *et al*. 2006), the disease is identified as a hazard in beef and hog casings.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

Cattle or pigs infected with anthrax and displaying clinical signs will not pass ante- and post-mortem inspections and casings will not be made from these animals. The likelihood of entry is assessed as negligible.

6.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk is assessed to be negligible.

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7. Echinococcus granulosus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Echinococcus granulosus is a cestode parasite within the family *Taeniidae*. Ten genotypes are recognised (G1-G10) within this species complex (Youssefi *et al.* 2013).

7.1.2. OIE list

The OIE lists infection with *Echinococcus granulosus* and *E. multilocularis* as diseases of multiple species.

7.1.3. New Zealand status

In 2002, New Zealand was declared provisionally free from hydatids infection (Pharo 2002) and no new cases have been identified since then. *Echinococcus* spp. are listed as unwanted notifiable organisms (Unwanted Organism Register 2014).

7.1.4. Epidemiology

E. granulosus has a worldwide distribution and only a small number of mainly island countries are free from this organism (World Animal Health Database 2014).

Echinococcosis is a parasitic tapeworm infection occurring in dogs (and some species of wild canids) which are the primary host and several species of herbivores, and pigs are intermediate hosts (MacDiarmid and Thompson 1997; World Health Organisation 2001). Dogs become infected by consuming raw infected tissue (predominately lungs and liver) that contains viable cysts. Once a cyst is ingested, a tapeworm may develop within the dog's intestine and once mature it will produce eggs that are passed in faeces. Intermediate hosts can then opportunistically consume these eggs and the resultant larvae migrate primarily to the lungs, but also the liver, and develop into a fluid filled cyst. Other sites of infection may include the central nervous system, skeletal muscles and the marrow cavity of bones (World Health Organisation 2001).

Human ingestion of eggs excreted in dog faeces can cause infection leading to death in extreme cases. Humans do not perpetuate the lifecycle of this parasite and are considered accidental hosts (World Health Organisation 2001).

Cysts are rarely viable in cattle, except in countries where the cattle genotype (G5) is present (World Health Organisation 2001).

The majority of cysts are located in offal (lungs and liver) (MacDiarmid and Thompson 1997), although there is a small likelihood of cysts being found in other body tissue (i.e. intestines). If this were to occur, the cysts would not remain associated with casings due to commercial processing (MPI 2010). Accordingly, the *Code* lists casings as a safe commodity that requires no risk management measures.

7.1.5. Hazard identification conclusion

The commercial processing of casings will eliminate any cysts associated with bovine or porcine intestinal tissue. Hence, *E. granulosus* is not identified as a hazard in beef and hog casings.

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8. Foot and mouth disease virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Foot and mouth disease virus (FMDV) is a member of the family *Picornaviridae* and genus *Aphthovirus*. There are seven distinct serotypes; A, O, C, SAT1, SAT2, SAT3, and Asia1 (OIE 2012).

8.1.2. OIE list

Foot and mouth disease (FMD) is an OIE listed disease.

8.1.3. New Zealand status

This virus has never been found in New Zealand (Pharo 2002) and is listed as an exotic notifiable organism (Unwanted Organisms Register 2014).

8.1.4. Epidemiology

FMD is a highly contagious, economically significant disease of cloven-hoofed animals, including pigs and cattle (OIE 2012).

FMD is distributed widely across the world and is endemic in large areas of Africa, Asia, the Middle East and South America (World Animal Health Information Database Interface 2014).

All secretions and excretions from an infected individual can contain infectious virus and viral shedding can occur for up to four days before the onset of clinical signs (OIE 2009). The pattern of excretion is similar for pigs and cattle with a sharp decline occurring at approximately 4-5 days after signs of clinical disease and all secretions and excretions being free from detectable virus 10-14 days post-infection (Alexandersen *et al*. 2012). Transmission to susceptible species can occur by a variety of routes, including direct contact, indirect contact via infected fomites, airborne infection caused by the inhalation of aerosols, and by the consumption of contaminated meat (Thomson and Bastos 2004).

Cattle can become carriers of FMDV for up to 3 years, whereas pigs do not become carriers and do not harbour infectious virus for more than 28 days (OIE 2012). Cattle that are in a carrier state may be able to transmit FMDV if they come into close contact with other susceptible species. However, the importance of this route for transmission is controversial (Thomson and Bastos 2004).

The severity of clinical signs is dependent on the strain of virus, exposure dose, age and breed of animal, host species, and degree of host immunity. Mortality is lower in older animals (1-5%) and is generally higher in young calves and piglets (≥20%). Clinical signs in cattle include vesicles in the mouth and on the feet, pyrexia, anorexia, shivering, and a reduction in milk production for 2-3 days. Clinical disease is usually severe in pigs and can include pyrexia, development of vesicles at pressure points on limbs, and severe foot lesions, which can cause lameness (OIE 2009; Alexandersen *et al*. 2012).

Recent outbreaks of FMD in countries considered free from the disease have probably been caused by contaminated untreated food waste that has been feed to livestock. The economic cost of such an outbreak was estimated at $US 15 billion for the United Kingdom incursion which occurred in 2001 (Alexandersen *et al*. 2012).

FMDV has been isolated from the intestines of experimentally infected cattle and pigs (Wijnker *et al.* 2007; 2012). McKercher *et al.* (1978) state in their paper that FMDV can survive in processed casings for 250 days, although Wijnker *et al* (2007) highlights that no experimental data is available to support this suggestion.

8.1.5. Hazard identification conclusion

FMDV is a highly contagious, economically devastating disease, which can be transmitted to susceptible species through the consumption of infected animal products.

As FMDV has been isolated from intestinal tissue, it is identified as a hazard in hog and beef casings.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

FMDV can be present in both cattle and pig intestines.

Clinical disease is usually severe in pigs and they do not become carriers as they only harbour infectious virus for up to 28 days. Hence, there is a lower likelihood that infected pigs would pass ante- and post-mortem inspections. Cattle, however, can be carriers of this virus for up to 3.5 years and some breeds of cattle (for example Zebu (*Bos indicus*)) often exhibit less severe or no clinical signs compared to European breeds of cattle (*Bos taurus*) (Alexandersen *et al*. 2012). FMDV persists in carrier cattle in the mucosa of the soft palate, pharynx and cranial oesophagus (Thompson and Bastos 2004) and is unlikely to be present in the intestines. Infected cattle that are carriers of FMDV are more likely to pass ante- and post-mortem inspections compared to pigs.

FMDV has been shown to be inactivated in pig and cattle intestines that were stored in dry salt or dry phosphate supplemented salt for 30 days at 20 °C (Wijnker *et al*. 2007; Wijnker *et al.* 2012). Another study exposed FMDV within a 3D collagen matrix model to a salt solution or a phosphate supplemented salt solution and found that inactivation occurred after 30 days at all tested temperatures (Wieringa-Jelsma *et al*. 2011).

For the inactivation of FMDV in casings of ruminants and pigs, the *Code* (Article 8.6.41) recommends that the following procedure should be used; salting for at least 30 days either with dry salt (NaCl) or with saturated brine (water activity; $a_w < 0.80$), or with phosphate supplemented dry salt (containing 86.5 percent NaCl, 10.7 percent Na_2HPO_4 and 2.8 percent Na_3PO_4, weight/weight/weight), and kept at a temperature of greater than 12 °C during this entire period (OIE 2013).

The standard manufacturing procedure for the international casings industry includes salting for 30 days at room temperature (see commodity definition Section 3).

For beef and hog casings complying with the commodity definition, the likelihood that FMDV would enter New Zealand is assessed to be negligible.

8.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk is estimated to be negligible.

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9. Exotic *Salmonella* spp.

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Salmonella spp. are Gram-negative bacteria which belong to the family *Enterobacteriaceae*. There are approximately 2500 serovars in the *Salmonella* genus and most of these belong to the species *enterica* and subspecies *enteric*. For simplicity, only the genus name followed by the serovar will be written e.g. *Salmonella* Dublin.

Multiple different strains are present in each serovar, which can be differentiated by phage typing and identified by the notation DT and a number. The strain *Salmonella* Typhimurium DT104 is important as it has developed resistance to multiple types of antibiotics and is widely distributed around the world (MPI 2010).

9.1.2. OIE list

Salmonellosis of sheep and goats associated with *Salmonella* Abortusovis is an OIE listed disease. In poultry, fowl typhoid and pullorum disease (*Salmonella* Gallinarum-Pullorum) are listed by the OIE.

9.1.3. New Zealand status

Salmonella Dublin is listed as an unwanted notifiable organisms and was first isolated from a sheep in 1966 and since then no new isolates have been reported (Clark 1999). *Salmonella* Typhimurium is endemic in New Zealand, although, *S.* Typhimurium DT 104 has been rarely isolated from humans and, in New Zealand, it has only been recorded once in three dogs in a household in which the owner was suffering from diarrhoea after returning from overseas (Julian 2002). It is classified in the category of "other exotic organism" and is unwanted (Unwanted Organisms Register 2014).

9.1.4. Epidemiology

Salmonella spp. have been isolated from virtually all species of vertebrates that have been tested and this genus of bacteria can contaminate the environment for a considerable period of time (Penrith *et al.* 2004).

Transmission of *Salmonella* spp. occurs mainly by the faecal oral route. Oral infection results in the multiplication of the organism in the intestine and invasion of the intestinal mucosa, followed by replication in the intestinal lymphatic tissue. Bacterial replication may also occur in the lungs if the organism is introduced intranasally (Penrith *et al.* 2004).

The course of infection is dependent on the infectious dose, serovar virulence and the host's immune response. Clinical signs of disease may appear 38 to 48 hours after infection and intestinal salmonellosis usually lasts 2 to 7 days (Penrith *et al.* 2004).

If the infection is restricted to the intestinal tract clinical signs may present as watery diarrhoea and may progress to enterocolitis. Infection in the intestinal tract may also spread to other organs causing septicaemia or bacteraemia (Fenwick and Collet 2004).

Salmonellosis is a zoonosis and can be easily transmitted from animals to humans by ingestion or inhalation. Animal products especially raw milk, meat and eggs are the most common cause of infection in humans (Fenwick and Collet 2004).

In outbreaks of bovine salmonellosis, the morbidity may reach 50 per cent and the fatality rate in untreated animals can be 100 per cent (Fenwick and Collet 2004). In pigs that display clinical signs of disease mortality can also be high (Penrith *et al*. 2004).

Clinically infected animals excrete large numbers of bacteria, especially in faeces and adult animals can become carriers, which may then sporadically shed bacteria (Penrith *et al*. 2004).

Casings are cleaned in batches in water heated to 40 °C (see commodity definition section 3) and bacterial cross-contamination between casings may occur (Wijnker 2009).

9.1.5. Hazard identification conclusion

Salmonella spp. are associated with bovine and porcine intestines and faecal cross-contamination occurs during processing into casings. Exotic *Salmonella* spp. are identified as a hazard in beef and hog casings.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Casings are stored for 30 days in either a dry salt which has a water activity (a_w) of 0.75, or in saturated brine (a_w 0.75-0.80). Most bacterial species cannot survive a water activity of less than 0.91 (Wijnker *et al.* 2006). Houben (2005) sampled 214 consignments of internationally traded casings and found that none tested positive for *Salmonella* spp. This provides evidence that the commercial manufacturing process inactivates vegetative bacteria.

As the a_w level in commercially processed casings is below that at which vegetative bacteria can remain viable, the likelihood of entry for all exotic *Salmonella* spp. is assessed as negligible.

9.2.2. Risk estimation

Since the likelihood of entry is assessed as negligible, the risk is estimated to be negligible.

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10. African swine fever virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

African swine fever virus (ASFV) is the sole member of the family *Asfarviridae* and genus *Asfivirus* (International Committee on Taxonomy of Viruses 2012).

10.1.2. OIE list

African swine fever (ASF) is listed by the OIE as a disease of swine.

10.1.3. New Zealand status

ASFV has not been described in New Zealand and is listed as an unwanted notifiable organism (Unwanted Organisms Register 2014).

10.1.4. Epidemiology

ASF is a highly contagious viral disease of pigs, which is endemic in many countries in sub-Saharan Africa (OIE 2013). Within Europe ASFV has been eradicated from the Iberian Peninsula but is endemic in feral pigs in Sardinia, Italy. In 2007 ASFV was isolated from pigs in Georgia (ProMed 2007) and since then the virus has spread eastwards and northwest and is currently present in western Russia, Armenia, Azerbaijan, Ukraine, Belarus, Lithuania, Lativa, Poland and the latest country to report the disease as of the 29th of September 2014 is Estonia (ProMed 2014). The virus is maintained mostly in the wild boar population and they are the main source of infection for domestic pigs (ProMed 2014).

ASFV affects members of the pig family (Suidae). Domesticated and feral pigs and European wild boars are susceptible to this virus (Sanchez-Vizcaino and Neira 2012). Three African suid species are subclinical carriers and act as reservoirs: warthogs (*Phacochoerus africanus*), giant forest hogs (*Hylochoerus meinertzhageni*) and bush pigs (*Potamochoerus porcus*) (Sanchez-Vizcaino and Neira 2012).

The spread of ASFV can occur by direct contact with infected pigs, contact with contaminated fomites and through tick vectors (genus *Ornthidoros*). This virus has often spread to new areas by pigs consuming uncooked infected pork. Waste food from international transport carriers (e.g. aircraft) have been shown to be an important source of ASFV (European Food Safety Authority 2010). ASFV strains vary in virulence and the incubation period for this disease is 4-19 days. Virulent strains can cause death within 4-10 days and mortality rates within the herd can reach 100%. These virulent strains are characterised by high fever, loss of appetite, and haemorrhages in the skin and internal organs (OIE 2012). After the onset of clinical signs, ASFV is shed at high levels in all secretions and excretions (Sanchez-Vizcaino and Neira 2012).

Less virulent strains are characterised by a slight fever, reduced appetite and depression, and these general signs can lead to the misidentification of the disease. Low virulence strains can cause seroconversion and on occasion subclinical non-haemorrhagic infection (OIE 2012).

ASFV can remain viable for up to 140 days in salted dried hams (Mebus *et al.* 1993; Mebus *et al.* 1997) and for up to 30 days in smoked pepperoni and salami sausage (McKercher *et al.* 1978). McKercher *et al.* (1980) also reported ASFV surviving in natural hog casings that were stored for 97 days, although no data is provided in the paper to support this claim.

10.1.5. Hazard identification conclusion

ASFV is an environmentally stable, highly contagious OIE listed disease and is found throughout all tissues and body fluids of infected pigs.

ASFV is identified as a hazard in hog casings.

ASFV is not identified as a hazard in beef casings.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Pigs that are infected with low virulence strains of ASFV and those that have recovered from infection, but are chronic carriers, are most likely to be infectious at slaughter (MPI 2014). Ante- and post-mortem inspections may not exclude these infected pigs from slaughter due to the absence of observable clinical signs.

There have been no studies conducted on the inactivation of ASFV in swine casings, although a study by Wieringa-Jelsma *et al.* (2011) found that ASFV in a 3D collagen matrix model, developed to simulate the situation in natural casings, was inactivated in 21 days at all tested temperatures when stored in a saturated salt solution.

Due to the manufacturing steps used to produce casings it is likely that the titre of any ASFV present would be significantly reduced. AFSV in cell culture has been inactivated when heated to 56 °C for 70 minutes or 60 °C for 20 minutes (Plowright and Parker 1967) and it has been inactivated in pig slurry when heated to 65 °C for 1 minute (Turner and Williams 1999). During processing casings are placed in heated water (approximately 40 °C) for an unspecified period to assist with cleaning. This may reduce the titre of virus. Additionally, the storage conditions that are a part of the standard operating procedure used by the international casing industry (see commodity definition section 3) would be expected to inactive any residual virus, as shown by Wierenga-Jelsma *et al.* (2011), especially when the salting process is supplemented by the addition of phosphate.

The likelihood that ASFV would enter New Zealand in this commodity is assessed as negligible.

10.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk is estimated to be negligible.

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11. Aujeszky's disease virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

The causative agent of Aujesky's disease (also known as pseudorabies) is *suid herpesvirus type 1*, a member of the *Varicellovirus* genus within the subfamily *Alphaherpesvirinae* and family *Herpesviridae* (International Committee on Taxonomy of Viruses 2012).

11.1.2. OIE list

Aujesky's disease (AD) is an OIE listed disease.

11.1.3. New Zealand status

AD was first identified in domestic pigs in New Zealand in 1976. The source of the introduction is unknown but is suspected to have been by the importation of new pig breeds from Britain. Disease surveillance began in 1988 and all individuals that were serologically positive were culled. The last domestic pig that tested seropositive was in 1995 and New Zealand is now regarded as being free from this disease in both domestic and feral pig populations (Motha 1998; MacDiarmid 2000).

Aujesky's disease virus (ADV) is an unwanted notifiable organism (Unwanted Organisms Register 2014).

11.1.4. Epidemiology

AD is widespread throughout the world and is especially prevalent in areas that farm pigs at high densities. Several countries have successfully eradicated AD from their domestic herds and are now regarded as free from the disease. This includes a number of countries in Europe, and the United States. Feral pigs in these countries still harbour the disease and present a constant threat of reintroduction into AD free herds (Mettenleiter *et al.* 2012).

ADV is shed in pig secretions, excretions and aerosols and the incubation period is approximately 1-8 days. Virus shedding commences 1-2 days post infection but before the onset of viremia and clinical signs. Shedding peaks 2-5 days post infection and lasts for up to 17 days (Mettenleiter *et al.* 2012).

Pigs are the only natural host for AD, being the only species which can survive infection. However, cattle, sheep, cats, dogs and rats can also be naturally infected resulting in fatal disease (Mettenleiter *et al.* 2012). Cattle do not survive infection and the incubation period is usually a couple of days before the onset of severe neurological signs (Van Oirschot 2004). Humans are not susceptible to infection and all susceptible species except pigs are considered dead-end hosts (MPI 2014).

Clinical signs of disease are variable and are dependent on the age of the pig and strain of virus. In neonatal pigs the disease may result in sudden death with few, if any clinical signs. In 1-2 week old pigs, signs may include trembling, incoordination, convulsion, tremor, ataxia and paralysis and mortality can be 100%. Older animals aged between 3-6 weeks may exhibit similar signs and as they age the mortality rate decreases to less than 5% in pigs aged 5 months. Individuals who survive infection become latently infected and reactivation of the virus can occur if animals are stressed (Mettenleiter *et al.* 2012).

ADV is relatively resistant to environmental conditions and is stable at pH 4-12. At extreme pH values, below 2 or above 13.5 viral inactivation takes 2-4 hours. With regards to thermal

stability, ADV was found to survive at 25 °C, 15 °C, and 4 °C for approximately 6, 9 and 20 weeks respectively. At a temperature of 60 °C, inactivation takes about 60 minutes and at 100 °C about 1 minute (Mettenleiter *et al.* 2012). ADV is most stable at pH 7 and when stored in water heated to 25 °C inactivation of 99.99% of the virus occurred after 4 days (Van Oirschot 2004). Further, ADV has been found to be inactivated by ultraviolet light and dry conditions (Animal Health Australia 2012; Mettenleiter *et al.* 2012).

ADV is not very contagious; for example an infectious dose (ID) of $>1 \times 10^4$ tissue culture infective dose ($TCID_{50}$)[7] is needed to cause infection in animals. However, piglets are more susceptible to infection with an ID of 1×10^2 $TCID_{50}$ initiating infection. Larger doses of virus are needed to cause infection by the oral route compared to intranasal infection (Mettenleiter *et al.* 2012).

Transmission of ADV is primarily through the respiratory route from direct nose-to-nose contact and by contact with contaminated fomites, with the nasal and oral cavities being the main entry points for infection (Mettenleiter *et al.* 2012). Under favourable atmospheric conditions, when virus loads are high ADV may be disseminated short distances by the airborne route, although long distance airborne spread is disputed. Exposure to semen and vaginal mucosa can also transmit infection and AD appears to be predominantly transmitted by the venereal route in feral swine (Mettenleiter *et al.* 2012). Regarding the consumption of infective tissue, the *Code* does not consider meat or meat products that contain no offal to pose a risk to the importing country (OIE 2014). Offal containing head, thoracic or abdominal viscera, however, require risk management measures.

ADV has been isolated from Meissner's plexuses within submucosa tissue in both experimentally and naturally infected pigs (Narita *et al.* 1984; Ezura *et al.* 1995; Zhao 1996; Narita *et al.* 1998). There have been no studies on the inactivation of ADV virus in casings.

11.1.5. Hazard identification conclusion

ADV has been isolated from intestinal tissue from infected pigs. It is relatively stable, has been isolated from the submucosa, and the OIE has made recommendations for the trade in swine viscera. For these reasons, ADV is identified as a hazard in hog casings.

In cattle this disease is rapidly fatal so infected individuals are not likely to pass ante- and post-mortem inspections. ADV is not identified as a hazard in beef casings.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Pigs are the only species that can remain latently infected after recovering from this disease. Clinical signs may be minor or not apparent when animals are either incubating the virus or are latent carriers. Ante- and post-mortem inspections may subsequently not detect these animals. ADV in latently infected animals has been shown to be reactivated by stress, and handling and transport prior to slaughter may contribute to this reactivation.

Even though no studies have been conducted on ADV survival in swine casings there are factors that will likely lead to a substantial reduction in virus titre. Storage of ADV in water for four days at 25 °C resulted in a 99.99% reduction in virus titre (Van Oirschot 2004). During processing casings are placed in heated water (approximately 40 °C; see commodity definition

[7] Tissue culture infective dose ($TCID_{50}$): The dose of virus that will produce pathological changes in 50% of cell cultures inoculated.

section 3) and this will reduce the titre of virus. The standard operating procedure of storing casings in dry salt or saturated brine for a minimum of 30 days would be expected to further reduce the titre of virus.

The likelihood of entry is assessed as negligible.

11.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk estimate for ADV is negligible.

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12. Classical swine fever virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Classical swine fever virus (CSFV) is a member of the *Pestivirus* genus within the family *Flaviviridae* (International Committee on Taxonomy of Viruses 2012).

12.1.2. OIE list

Classical swine fever (CSF) is an OIE listed disease.

12.1.3. New Zealand status

New Zealand has been free from CSF since 1953 (Davidson 2002) and it is listed as an exotic notifiable organism.

12.1.4. Epidemiology

CSF is a highly contagious and economically important disease of swine, which includes all breeds of the species *Sus scrofa* that are the only natural host for this virus (OIE 2009).

In the last 15 years, more than 60 countries have notified the OIE of at least one outbreak of CSF. Presently, it is found in much of Asia, some African countries, Central and South America, and Russia and Latvia in Europe (United States Department of Agriculture 2013; World Animal Health Information Database Interface 2014).

There have been two CSF outbreaks in New Zealand (Wellington 1930, Auckland 1953). Eradication of the disease was achieved by culling infected herds (Anonymous 1991).

Transmission of CSF is mainly by the oral and oronasal routes, via direct and indirect contact. All bodily fluids of an infected individual can contain infectious virus and the most common pathways of introduction into new countries is by pigs being fed insufficiently cooked pork in food waste. Once CSF is present in a country, wild pigs can harbour the virus and reintroduce it to domestic populations. Shedding of the virus can occur for months from infected individuals (OIE 2009).

The incubation period for CSF is 2-14 days. Shedding of the virus may occur before the onset of clinical signs and the amount excreted within body fluids is dependent on the virulence of the strain with more virulent strains resulting in a higher concentration of virus (United States Department of Agriculture 2013).

Clinical signs of disease are variable and depend on the virus strain, the age, the health status and breed of the pigs (Kirkland *et al.* 2012). Acute CSF is caused by virulent strains and is the most devastating form of the disease, usually causing death within 1-3 weeks. Signs of acute disease include high fever, huddling, weakness, drowsiness, anorexia, conjunctivitis and constipation followed by diarrhoea. Mortality in young pigs may reach 100% (OIE 2009). In the chronic form of the disease, the same clinical signs may be observed and pigs usually survive for 2-3 months before dying (Kirkland *et al.* 2012).

McKercher *et al.* (1980) demonstrated that CSFV may survive in processed casings for up to 147 days when stored in saturated brine and held at a temperature of 4 °C, as pigs inoculated intramuscularly with these infected casings produced clinical signs of disease and died. Further, Depner *et al.* (1998) showed that CSFV can survive in casings that have been stored in saturated brine (pH 6.3) at 4 °C for 20 days. The mean half-life of CSFV has been shown to

be 50 hours when held in suspension and stored at a pH of 7 and temperature of 21 °C (Depner *et al.* 1992).

Recently, Wijnker *et al.* (2008) inoculated a pig with a highly virulent strain of CSFV ("Koslov"). The intestines from this animal were used to make casings and these casings were treated with phosphate-supplemented salt or citrate-supplemented salt and stored for 30 days at 4 or 20 °C. After the 30-day storage period, the casings were fed to susceptible pigs and disease occurred in those pigs which consumed casings that were stored in citrate-supplemented salted and held at a temperature of 4 °C. All other pigs did not become infected.

Reflecting these findings, the *Code* recommends that casings be stored in phosphate supplemented salt or brine for a minimum of 30 days at a temperature above 20 °C to enable the safe trade in hog casings.

12.1.5. Hazard identification conclusion

As the standard manufacturing procedure for casings does not include the addition of phosphate-supplemented salt there is the potential for CSFV to be present in hog casings and, therefore, CSFV is identified as a hazard.

CSFV is not identified as a hazard in beef casings.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Pigs infected with less virulent strains of CSFV may pass ante- and post-mortem inspection and hence infected intestines from such animals may be used for the production of casings.

The standard processing procedure for hog casings is to apply salt for a minimum of 30 days and this treatment may not inactive CSFV, especially if the temperature is less than 20 °C (Wijnker *et al.* 2008; Wieringa-Jelsma *et al.* 2011). Viable virus may remain and the likelihood of entry of CSF in the commodity is assessed as non-negligible.

12.2.2. Exposure assessment

Historically the main pathway of entry of CSF into countries has been attributed to the importation of contaminated meat that has been fed to pigs in food waste (Anonymous 1991).

In New Zealand the feeding of untreated meat or food waste is prohibited by legislation (Biosecurity Meat and Food Waste for Pigs Regulations 2005). Compliance with this regulation is thought to vary across the pig farming community, and is likely to be high in commercial operations but lower in small-scale backyard pig farms (MPI 2006).

If pigs were illegally fed with contaminated casings, infection could spread to other pigs and contaminate the environment.

The likelihood of exposure is assessed as non-negligible.

12.2.3. Consequence assessment

The consequences of introduction would be dependent on the strain of CSFV and its pathogenicity (MPI 2014). High mortalities (90-100%) could occur due to the naivety of New Zealand herds to this disease (Anonymous 1991). If the disease was allowed to spread, the economic cost could be high. Furthermore, feral pigs could become infected and be a persistent source of infection for domestic herds.

Control measures to limit the spread of CSF would likely consist of the quarantine of infected premises, trace back of pigs that could have been exposed, the slaughter of all pigs on the farm and thorough cleaning and disinfection of all the exposed premises with carcasses being disposed by burning or deep burial (MPI 2014).

Pigs are the only host for this virus so there are no consequences for human health or for other animals.

12.2.4. Risk estimation

Since the likelihood of entry and exposure, and the consequences are all assessed to be non-negligible, the risk estimation is also non-negligible. CSFV is assessed to be a risk in hog casings.

12.3. RISK MANAGEMENT

12.3.1. Options

The *Code* chapter for CSF states that the status of a country, zone or compartment can be determined after considering the criteria listed in Article 15.2.2. These criteria include that the disease should be notifiable in the whole territory (OIE 2014).

The *Code* sets out recommendations that allow for a country, zone or compartment to be considered free from CSF if there has been no outbreak of CSF and no vaccination has been carried out in the last 12 months, unless there are means, validated according to Chapter 2.8.3 of the OIE *Manual*, to distinguish between vaccinated and unvaccinated pigs. Recommendations on how to recover a free status after an outbreak has occurred in a previously free country or zone are provided in Article 15.2.6. Furthermore, Article 15.2.4 provides recommendations on the bilateral recognition of CSF free compartments and Article 15.2.5 details how a containment zone can be established in a previously CSF free country or zone. In May 2013, CSF was added to the list of diseases for which official OIE recognition of disease free status can be obtained. No countries, zones or compartments have been recognised as officially disease free and applications for freedom will start to be evaluated from May 2014 to May 2015.

The *Code* gives recommendations for the importation of swine casings that may originate from CSF infected countries or zone.

The relevant *Code* article is reproduced below:

Article 15.2.24. Procedures for the inactivation of the CSFV in casings of pigs

For the inactivation of CSFV in casings of pigs, the following procedures should be used: salting for at least 30 days either with phosphate supplemented dry salt or saturated brine (Aw < 0.80) containing 86.5% NaCl, 10.7% Na_2HPO_4 and 2.8% Na_3PO_4 (weight/weight/weight), and kept at a temperature of greater than 20 °C during this entire period.

CSFV is not identified as a hazard in beef casings so no risk management measures are justified for this commodity.

The following measures could be considered to effectively manage the risk in imported hog casings.

Option 1

Hog casings could be imported only from countries, zones or compartments recognised by New Zealand as free from CSFV.

Option 2

For the inactivation of CSFV in casings of pigs, the following procedures could be used: salting for at least 30 days either with phosphate supplemented dry salt or saturated brine (Aw < 0.80) containing 86.5% NaCl, 10.7% Na_2HPO_4 and 2.8% Na_3PO_4 (weight/weight/weight), and kept at a temperature of greater than 20 °C during this entire period.

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13. Porcine reproductive and respiratory syndrome virus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Porcine reproductive and respiratory syndrome virus (PRRSV) is a member of the family *Arteriviridae* within the genus *Arterivirus* (International Committee on Taxonomy of Viruses 2012).

13.1.2. OIE list

Porcine reproductive and respiratory syndrome (PRRS) is listed as a disease notifiable to the OIE.

13.1.3. New Zealand status

PRRS has not been described in New Zealand and is listed as an unwanted notifiable organism (Unwanted Organisms Register 2014).

13.1.4. Epidemiology

PRRS was first recognised in 1987 in the USA and within a few years it had been described in a number of pig producing countries (OIE 2010). Currently, it is found in many countries in Europe, the Americas, and Asia (World Animal Health Information Database Interface 2014). Pigs are the only host for this virus.

Arterviruses are characterised as having a high rate of mutation because of their mechanism of RNA replication. This makes PRRSV genetically unstable and isolates vary in both nucleic sequence and pathogenicity (MPI 2006). Differences in virulence between strains also exist and the basis for this is not yet fully understood (College of Veterinary Medicine 2013).

Clinical signs of disease are variable and influenced by a number of factors including the virulence of the virus, whether the virus is infecting a naïve population, the age at which the onset of infection occurs, whether other diseases are present in the population, the size of the herd, and farm management practices. Some herds may experience reproductive failure, with sows aborting or farrowing pre-term, the birth of stillborn or mummified piglets, sow deaths and pre-weaning mortality of piglets. Respiratory disease and death can also occur in weaners and fattening pigs. In contrast, due to the differences in virulence between strains, some herds may be relatively unaffected (MPI 2006; College of Veterinary Medicine 2013).

Previous studies have found that the highest titre of virus is located in macrophages, particularly those in the lungs and lymph nodes (MPI 2006). In addition, PRRSV has been isolated from the small and large intestine of experimentally infected pigs. The virus was mainly observed in the mucous membrane epithelium or glandular epithelium, located within the mucosa layer of the intestine (Li *et al.* 2012). In hog casings only a residual amount of mucosa tissue remains after processing (see commodity definition section 3).

Transmission can occur by a variety of routes including direct contact with an infected pig, contaminated semen, infected fomites, consumption of infected meat and aerosols (European Food Safety Authority 2005, MPI 2006). Several studies have shown that PRRS can be transmitted by the consumption of raw infected pig meat. However, no study has exposed meat to normal handling and commercial processing conditions before feeding to pigs and this would be expected to have a significant effect on the titre of virus in meat (Pharo and Cobb 2011).

Several studies have found that a high percentage of pigs presented for slaughter in countries where PRRS is established have tested seropositive (MPI 2006).

PRRSV persists relatively poorly in the external environment and this is due to the characteristics of the lipid envelope, which is susceptible to heat, pH, desiccation and time since slaughter (MPI 2006; Zimmerman *et al.* 2012). This virus has been shown to persist for 1-6 days at 20-21 °C, 2-24 hours at 37 °C and 6-20 minutes at 56 °C. At refrigeration temperature (4 °C), approximately 90% of PRRSV infectivity is lost within a week (Benfield *et al.* 1999). A study measuring the survival time of four PRRSV isolates held at ambient temperatures and a pH of 7.5 found that the mean half-life was 155.5 hours at 4 °C; 84.8 hours at 10 °C; 27.4 hours at 20 °C; and 1.6 hours at 30 °C (Jacobs *et al.* 2010). PRRSV is stable when the pH is 6.5 - 7.5 and infectivity is rapidly reduced when the pH is below 6 or above 7.5 (Zimmerman *et al.* 2006).

13.1.5. Hazard identification conclusion

PRRSV has been identified in the small and large intestines of experimentally infected swine and is identified as a hazard in hog casings.

PRRSV is not identified as a hazard in beef casings.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

PRRSV strains vary in pathogenicity and for some strains infection can remain inapparent in many herds (European Food Safety Authority 2005). Ante- and post-mortem inspections may not exclude these infected pigs from slaughter.

Viral persistence in pig tissue is influenced by the interaction between time since slaughter, storage temperature and pH.

PRRSV is heat liable and placing intestines into heated water (approximately 40 °C; see commodity definition section 3) which assists with their cleaning would reduce the titre of virus in infected mucosa tissue.

After cleaning, casings are stored for at least 30 days at room temperature (approximately 20 °C; European Food Safety Authority 2012). Jacobs *et al.* (2010) predict the half-life of PRRSV to be 27.4 hours at 20 °C. Storage of casings for 30 days at room temperature would, therefore, be associated with a reduction in viral titre equivalent to 26 half-lives; that is, a reduction of >99.9999% of the original virus titre will occur over this period.

As PRRSV is only found in mucosa tissue which is present in hog casings in residual amounts, and there would be a significant inactivation of virus during the 30 day storage period, the likelihood of entry is assessed as negligible.

13.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk is estimated to be negligible.

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14. Swine vesicular disease virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Swine vesicular disease virus (SVDV) is a variant of Enterovirus B that is a member of family *Picornaviridae* and genus *Enterovirus* (International Committee on Taxonomy of Viruses 2012). There is only one serotype with strain variation between isolates (OIE 2009).

14.1.2. OIE list

Swine vesicular disease (SVD) is not listed by the OIE.

14.1.3. New Zealand status

SVD has never been reported in New Zealand and is an exotic notifiable organism (Unwanted Organisms Register 2014).

14.1.4. Epidemiology

SVD was once found throughout Europe but has since been eradicated from many countries with recent outbreaks occurring in Portugal (2007) and Italy (2009) (Alexandersen *et al.* 2012). The disease is also likely to be present in some Asian countries and the last documented outbreak was in Taiwan in 2000 (European Food Safety Authority 2012).

Swine are the only host for SVDV and it is not a zoonotic disease (European Food Safety Authority 2012).

The result of infection with SVDV is variable and strain dependant. Vesicles may develop on the coronary band, at the junction with the heel, and interdigital spaces of the feet, and less often on the snout, tongue and lips (OIE 2009). Mortality does not usually occur, but morbidity may reach 100%. Recent outbreaks have been notable for displaying less severe or no clinical signs and infection has only been found when animals are tested in serosurveillance programmes or for export certification (OIE 2009). The clinical signs of SVD can be wrongly attributed to foot and mouth disease or vesicular stomatitis and any outbreak must be confirmed by enzyme-linked immunosorbent assay (OIE 2009; European Food Safety Authority 2012).

The most common way of introducing this disease to new areas is through the movement of subclinically infected animals. The incubation period for this virus is between 2-7 days although for trade purposes the *Code* considers it to be 28 days (OIE 2009). The virus can be excreted from the nose and mouth and in faeces for up to 48 hours before the onset of clinical signs. Virus shedding continues for up to 2 weeks from the nose and mouth, up to 3 months in faeces, and at least 7 days from vesicles, with the highest titre of virus being shed in the first 7 days post infection (PI) (OIE 2009; Alexandersen *et al.* 2012). All tissues also contain virus during the viraemic period, which can occur within 1 day PI, and lasts for approximately 2-3 days (OIE 2009; Alexandersen *et al.* 2012; European Food Safety Authority 2012). Not all infected animals develop viraemia (European Food Safety Authority 2012). Transmission occurs by exposure to contaminated excretions, faeces, fomites, direct contact and consumption of infected meat.

SVDV can survive for a prolonged period in the environment (Alexandersen *et al.* 2012). It is preserved by refrigeration, can withstand a wide range of pH (2.5-12) and is inactivated when heated to 56 °C for 1 hour (OIE 2009). It can also survive the commercial curing process for Serrano hams (Mebus 1993; Mebus *et al.* 1997).

A recent study has found that SVDV titre is reduced significantly when exposed to the conditions applied in the commercial production of casings (Wieringa-Jelsma *et al.* 2011).

The European Food Safety Authority (2012) has assessed that animal products are unlikely to contribute to the spread of SVDV.

14.1.5. Hazard identification conclusion

SVDV is an environmentally stable virus that has never been reported as occurring in New Zealand. SVDV is identified as a hazard in hog casings.

SVDV is not identified as a hazard in beef casings.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

As pigs may be subclinically infected with SVD and viraemic at the time of slaughter, ante- and post-mortem inspections may not always detect these infections, especially as recent SVD outbreaks have exhibited less severe or no clinical signs (OIE 2009). Casings derived from such animals may subsequently contain infectious virus.

Several steps used in the manufacturing process of casings will reduce the survival of SVDV. During processing casings are placed in heated water (approximately 40 °C; see commodity definition section 3) to assist with cleaning. This process may reduce the titre of any SVDV that might be present in casings, as it has been shown that a heat treatment of 56 °C one hour inactivates this virus (OIE 2009). After cleaning the casings are stored in dry salt or saturated brine for a minimum of 30 days and this has been experimentally shown to reduce the titre of SVDV in a 3D collagen matrix model (Wieringa-Jelsma *et al.* 2011). Moreover, experience from international trade has shown that SVDV does not appear to be a risk in hog casings.

The combination of these two processing steps will reduce the likelihood of entry of SVDV to a negligible level.

14.2.2. Risk estimation

Since the likelihood of entry is assessed to be negligible, the risk estimate for SVDV is negligible. SVDV is assessed not to be a risk in hog casings.

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15. The agent of bovine spongiform encephalopathy

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Bovine spongiform encephalopathy (BSE) is a transmissible spongiform encephalopathy (TSE) or prion disease (OIE 2010).

15.1.2. OIE list

BSE is an OIE listed disease of cattle.

15.1.3. New Zealand status

New Zealand is free from BSE and the OIE categorises New Zealand's risk status for this disease as negligible.

Variant Creutzfeldt-Jacob disease is the human manifestation of BSE and it has never been identified in New Zealand (Environmental Science and Research 2013).

15.1.4. Epidemiology

BSE is an invariably fatal disease of cattle that was first identified in the United Kingdom (UK) in 1986. Since then it has been reported in 27 other countries, the majority of which are within Europe (OIE 2014). In the UK the outbreak peaked in 1992 when 37 280 cases were recorded (MPI 2014) and in 2013 there were three cases. Outside of the UK in 2013, there were four cases, all of which occurred within Europe (2 in France, 1 in Ireland and 1 in Poland) (OIE 2014). Globally, BSE is disappearing rapidly.

BSE was spread by feeding cattle infected meat and bone meal of bovine origin. The disease originally spread from the UK most likely due to the export of infected cattle and infected meat and bone meal. There is no evidence for horizontal transmission of this disease and vertical transmission does not play a role in perpetuating epidemics (Radostits *et al*. 2007; OIE 2010). In July 1988, the UK enacted a ban prohibiting the feeding of ruminant proteins to ruminants and in 1994, this ban was extended to all forms of mammalian protein as cross contamination in feed mills was occurring resulting in further cases of BSE (Radostits *et al*. 2007; Scottish Government 2012). The result of these control measures is that the number of infected animals has significantly decreased and BSE has now become rare.

Pigs have been experimentally infected with BSE when inoculated simultaneously by the intracranial, intravenous and intraperitoneal routes. Infection by oral exposure to BSE, however, has not been demonstrated and no cases of TSE in pigs were found during the BSE epidemic in cattle in the UK (Wells *et al*. 2003). TSEs have also been found in eight species of ungulates in zoos or wildlife parks in the UK and it is likely that these animals were exposed to contaminated feed (Kirkwood and Cunningham 1994). Feline spongiform encephalopathy (FSE) was also identified in domestic cats and zoo felines in the UK, with this outbreak coinciding with the BSE outbreak in cattle. The infectious agent in these cases was shown to be the same in these species. Up to 2003, 89 cases of FSE had been identified (Radostits *et al*. 2007) but no cases have been reported since 2007, which suggests that the disease has disappeared (MPI 2014).

The incubation period for BSE is long, ranging from 2.5 to over 8 years and it could persist for the entire lifespan of cattle. Because of this, it is a disease that manifests predominately in adult animals. The risk of infection is highest in the first 6 months of life and adult cattle are

at a lower risk of acquiring infection. Infected animals display clinical signs which are variable but which include changes in temperament, posture, sensorium and movement. These clinical signs become progressively worse, with the course of the disease ranging from 1 to 6 months. There is no cure for BSE and clinical infection always results in mortality (Radostits *et al*. 2007).

BSE infection in humans causes a syndrome called variant Creutzfeldt-Jakob disease (vCJD). As of the 7th of July 2014, 177 cases (definite or probable) of vCJD had been identified in the UK of which all have resulted in death (National CJD Research and Surveillance Unit 2014). The most important route of transmission to humans is through the consumption of food products that contain infectious tissue. The number of infectious tissues in cattle is small and the majority is located within the central nervous system, with 60% of BSE infectivity in the brain, followed by 24% in the spinal cord, 9.6% in the ileum, 3.6% in the dorsal root ganglia and <0.1% in the tonsils (New Zealand Food Safety Authority 2006).

Initially it was uncertain how humans were becoming infected with the BSE agent until mechanically recovered meat (MRM) was investigated. MRM was processed from the vertebral column of cattle using high-pressure techniques. The spinal cord was removed before processing, however, residual spinal tissue and the two dorsal root ganglia that are attached to the spinal cord were incorporated into a meat paste, which was used as an ingredient in a number of foods, such as burgers, sausages, pies and baby food. Up to 2% of the meat paste could contain central nervous system tissue. For example, a sausage weighing 100 grams could have contained up to two grams of infectious tissue and in sheep it has been shown that the infectious oral dose is only 0.5 grams (MacDiarmid 2004). Cooper and Bird (2002) concluded that about 90% of the MRM paste was used in burgers and it is the consumption of this product that drove the dietary exposure to BSE. So, while it is true that BSE infectivity is found in the intestines it was not a significant route of exposure for the people in the UK who acquired the disease.

In response to this outbreak the European Union has classified the entire intestinal tract of cattle from the duodenum to the rectum as 'specified risk material' (Regulation EC, No 999/2001 of the European Parliament), which is excluded from processing, and must be destroyed (Wijnker *et al.* 2008). The *Code*, on the other hand, recommends the exclusion of the ileum only (OIE 2014).

The European Food Safety Authority (2014) has developed a BSE infectivity model of cattle intestines and mesenteries from the European Union. This model showed that the distribution of infectious tissue within the intestines and the titre of the BSE agent vary by age. In cattle aged 18 months or less the jejunum, ileum and caecum contain the majority of infectious tissue, which peaks at approximately 15 bovine oral infectious doses ($BoID_{50}$)[8] per animal. Infectivity declines as cattle age and in animals aged 60 months or more, less than one $BoID_{50}$ is present in intestines and mesenteries. The majority of infectious tissue in these older animals is present not within the intestine but in the mesenteric nerves and celiac and mesenteric ganglion complex, with the ileum and jejunum containing only a residual amount of the total infectivity (1% and 6% respectively).

After processing cattle intestines into casings it has been estimated that there is on average a 25% reduction in infectivity in the duodenum, jejunum, caecum and colon tissues. The greatest reduction in infectivity (approximately 3 $BoID_{50}$) for processed casings occurs in cattle slaughtered before 18 months of age and in cattle older than 120 months the mean reduction in infectivity is only 0.01 $BoID_{50}$ (European Food Safety Authority 2014a).

[8] Bovine oral infectious dose ($BoID_{50}$): The oral dose that infects 50% of bovine animals following experimental inoculation.

In addition, the European Food Safety Authority (2014a) has concluded that for any given situation the exclusion of the last 4 metres of the small intestine (which includes the ileum) and of the caecum from the food and animal feed pathway will decrease the infectivity associated with intestinal tissue by more than 90% in cattle aged 36 months old and below.

It has been estimated that the outbreak of BSE in the United Kingdom has cost 600 billion pounds. In response, many countries have developed active surveillance programmes to provide evidence of freedom from this disease. The detection of only a single case of BSE can result in export bans and significant economic losses (Radostits *et al*. 2007).

15.1.5. Hazard identification conclusion

BSE has never occurred in New Zealand. Cattle intestines have been found to harbour BSE and this pathogen is identified as a hazard in beef casings.

BSE is not identified as a hazard in hog casings.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

In 2013, the total number of cattle identified as infected worldwide with BSE was 7 (OIE 2014). In 2014 (up to the 1st of May), two cattle have been found infected in Germany (OIE 2014b). The number of cases worldwide has steadily declined and because BSE is now so rare, the likelihood of casings being prepared from an infected cow is extremely low.

The OIE categorises the BSE risk status of countries as "negligible", "controlled" or "undetermined". The likelihood of BSE entering in any commodity from a country with a negligible BSE risk status would be negligible.

BSE infected animals will pass ante- and post-mortem inspection as there are no gross pathological changes detectable in an infected animal, and there are no preclinical tests available to diagnose infection. For countries with a controlled risk or undetermined risk status, the *Code* (Article 11.4.14) recommends that the ileum should not be traded. The likelihood that BSE would enter New Zealand in beef casings from countries with a controlled or undetermined risk status that are certified to comply with the commodity definition (exclusion of the ileum from international trade) is assessed as negligible.

15.2.2. Risk estimation

Since the likelihood of entry, is assessed to be negligible, the risk is assessed as negligible. BSE is assessed not to be a risk.

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