Import risk analysis: Sausage Casings from Small Ruminants

Rapid Risk Assessment

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Approved for general release

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Executive summary

This analysis examines the biosecurity risks posed by sausage casings of ovine or caprine origin, prepared by methods which are standard operating procedures in much of the international casings industry.

The sausage casings under consideration are processed from the intestines of animals which have passed ante- and post-mortem inspection. In making sausage casings from the intestines of sheep and goats, only the small intestines are used, and the only tissue which remains after processing is the submucosal layer. After cleaning and scraping, the casings are stored at room temperature in either dry salt or saturated brine for a minimum of 30 days, which is standard industry practice.

Once pulled from the abdominal cavity, the sheep or goat intestines have the gut contents stripped from them before being placed in cool storage overnight during which time the mucosa will degrade for easier removal. The mucosa is crushed in various steps and the outer layers (tunica serosa and tunica muscularis) are scraped off. The casings are cleaned in batches in warm water and studies have demonstrated that no lymphoid tissue (Peyer's patches) remains after cleaning.

The 15 diseases considered in this import risk analysis are those identified in a previously-published peer-reviewed assessment of the potential risks to animal health from sheep and goat meat products.

This import risk analysis concludes that sausage casings processed, salted and stored according to practices standard in the international casings industry pose no animal health risk to New Zealand and could be imported safely without any disease-specific measures being required.

1. Introduction

This import risk analysis examines the animal health risks posed by sausage casings prepared from the intestines of sheep and goats. The diseases assessed are those identified in a peer-reviewed assessment of the potential risks to animal health from sheep and goat meat published by the OIE (MacDiarmid and Thompson 1997).

2. Scope and commodity definition

This risk analysis deals only with sausage casings of ovine or caprine origin, prepared by methods which are standard operating procedures in much of the international casings industry.

The commodity under consideration comprises the submucosal layer of the small intestines of sheep and goats which have passed ante- and post-mortem inspection according the the OIE's *Terrestrial Animal Health Code*[1] (Chapter 6.2.). After cleaning and scraping, the casings have been stored at room temperature in either salt or saturated brine for a minimum of 30 days.

In making sausage casings from the intestines of sheep and goats, only the small intestines are used (Wijnker 2009). Further, in the European Union, the ileum of sheep and goats is designated as 'specified risk material' (SRM) and is excluded from processing and must be destroyed (Ruffing et al 2007; Wijnker 2009).

The intestinal wall comprises four 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). Essentially only the submucosa remains when sheeps' intestines are processed into casings (Wijnker 2009)[2]. Although occasional traces of serosa, mucosa and muscularis may remain, Peyer's patches are completely removed (Koolmees et al 2004).

Once pulled from the abdominal cavity, the sheeps' intestines have the gut contents stripped from them before being placed in cool storage overnight during which time the mucosa will degrade for easier removal. The mucosa is crushed in various steps and the outer layers (tunica serosa and tunica muscularis) are scraped off. The casings are cleaned in batches in water around 40°C (Wijnker 2009). A comparative histological study of casings produced from sheeps' intestines showed that no significant differences exist between those cleaned by manual or mechanical techniques and no lymphoid tissue (Peyer's patches) remains after cleaning (Koolmees et al 2004).

[1] http://www.oie.int/eng/normes/mcode/a_summry.htm

[2] While casings processed from sheeps' intestines comprise submucosa only, casings made from beef intestines retain all the original layers (Wijnker 2009).

After cleaning, **casings are stored for a minimum of 30 days at room temperature in either dry salt** (a_w 0.75) **or saturated brine** (a_w between 0.75 and 0.80) (Wijnker et al 2006; Wijnker 2009).

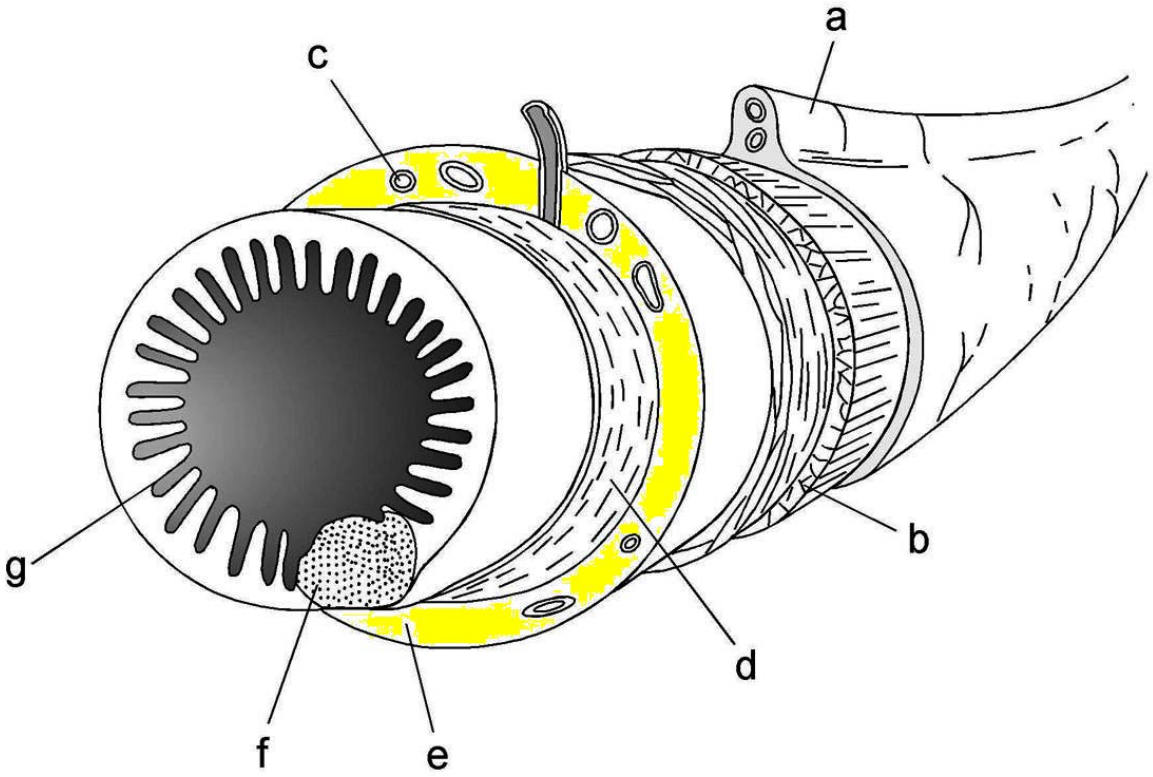


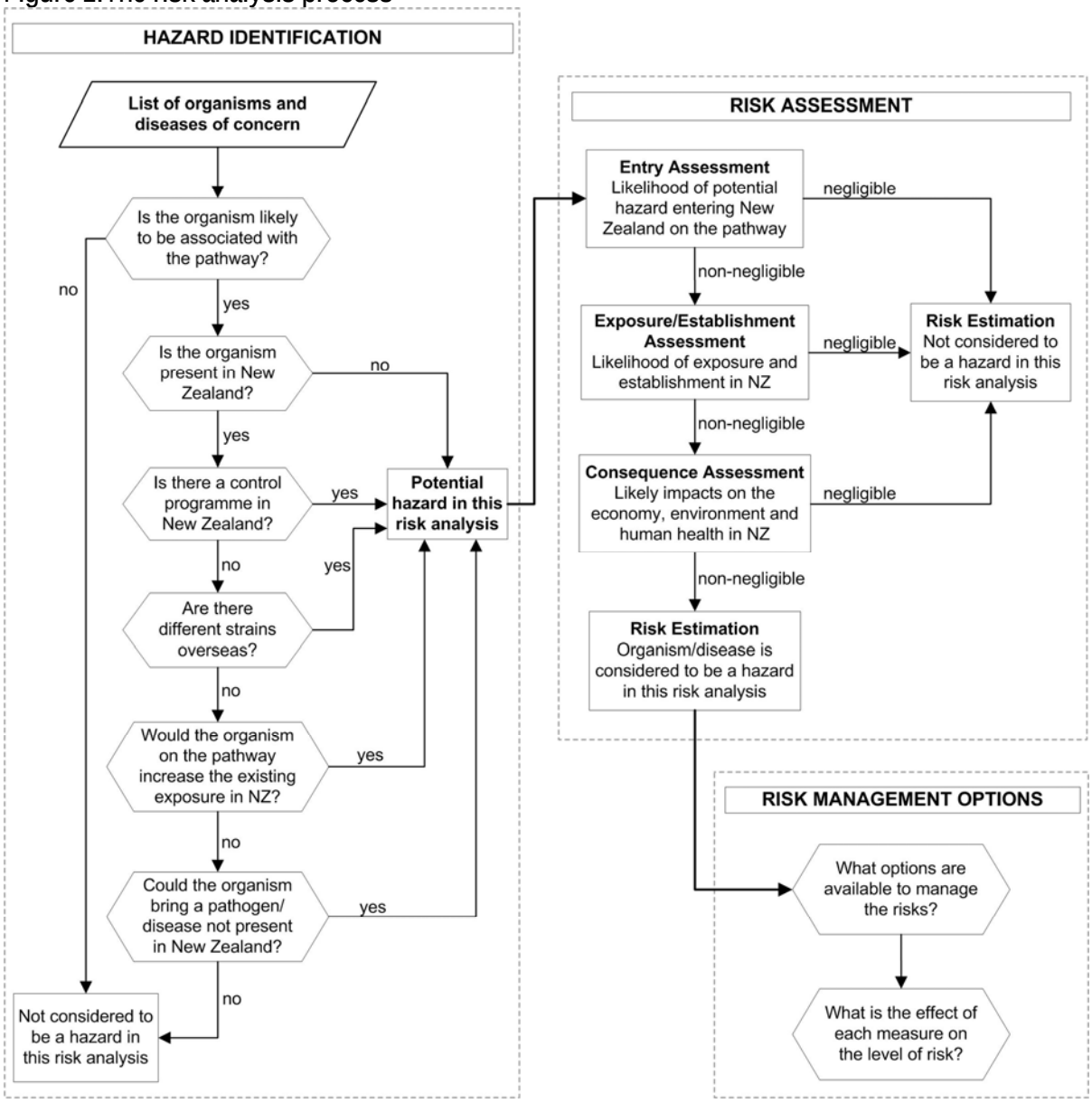
Figure 1: Schematic diagram of sheep small intestine showing (a) mesentery and serosa; (b) inner and outer muscle layers; (c) submucosal blood vessels; (d) muscularis mucosae; (e) submucosa; (f) lymphoid nodule (Peyer's patch); (g) tunica mucosa (villus and crypt layers). The tunica mucosa, the muscularis, the serosa and Peyer's patches are removed during processing, so the natural casing consists of only the submucosa (e). (Adapted from Wijnker 2009).

3. Risk analysis methodology

The methodology used in this risk analysis follows the 2006 MAF Biosecurity New Zealand *Risk Analysis Procedures-Version 1*. These procedures combine the guidelines in the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE) and International Plant Protection Convention guidelines. The procedures provide a framework which adheres to the requirements of the World Trade Organization's *Agreement on the Application of Sanitary and Phytosanitary Measures* (the *SPS Agreement*) and of the Biosecurity Act 1993.

The process followed is shown in Figure 2.

Figure 2:The risk analysis process



3.1. PRELIMINARY HAZARD LIST

The first step in the risk analysis process is to use text books and other sources to draw up a list of pathogens known to infect the species in question. This list of organisms of concern is subjected to the application of specific criteria to eliminate those that clearly are not hazards. The remaining organisms are considered to be 'preliminary hazards' and are then subjected to the process of hazard identification.

3.2. HAZARD IDENTIFICATION

Each organism in the preliminary hazard list is subjected to a hazard identification step that includes formal identification of the organism, whether it is an OIE listed disease, its New Zealand status, and particularly the epidemiology and relevant characteristics of the disease it causes. The hazard identification section is concluded by an assessment of whether the organism is a potential hazard or not. All potential hazards are subjected to risk assessment.

3.3. RISK ASSESSMENT

Risk assessment consists of:

1. *Entry assessment*: The likelihood of a pathogenic organism being imported with the commodity.
2. *Exposure assessment*: The likelihood of animals or humans in New Zealand being exposed to the potential hazard.
3. *Consequence assessment*: The consequences of entry, establishment or spread of an imported organism.
4. *Risk estimation*: An estimation of the risk posed by the biological products based on the entry, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is a risk and risk management measures are justified to reduce the level of risk posed by the importation of the commodity 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, 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 exposure to susceptible species in the importing country is negligible, or when both entry and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

3.4. RISK MANAGEMENT

For each organism classified as a risk, a risk management step is carried out to identify the available options. Where the *Code* makes recommendations for the management of a risk, these are described alongside options of similar, lesser or greater stringency, if available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all risks. 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 IHS is drafted.

As obliged under Article 3.1 of the *SPS Agreement*, the measures adopted in an IHS should be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where 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 following a risk assessment).

3.5. SPECIAL CONSIDERATIONS

Wijnker (2009) has pointed out that there have been very few studies conducted on the possible association of pathogens with casings. He goes on to say that opinions offered on their potential disease risk have been based on studies carried on other tissues and products and their relevance is questionable.

For a meat product to serve as a vehicle for the introduction of animal disease a number of criteria must be met (MacDiarmid and Thompson 1997; MacDiarmid and Thompson 2004).

- The disease must be present in the country of origin.
- The disease must be present in the particular animal slaughtered (or the tissues must have become contaminated during the butchering process).
- The diseased tissues must pass inspection procedures.
- The pathogen in the tissues must survive storage and processing and be present at an infectious dose.
- The pathogen must be present in the tissues traded.
- The pathogen must be able to establish infection by the oral route.
- Scraps of the meat product must find their way into a susceptible animal of the appropriate species in the importing country.

The likelihood of each of these criteria being met will be different for each pathogen and country of origin.

One further criterion must be fulfilled before a disease poses a risk to the livestock industry of the importing country. This is;

- Should the pathogen establish infection in a susceptible host in the importing country, local conditions must be such that the disease could spread and become endemic.

This criterion is particularly apt for those diseases whose transmission is dependent on arthropod vectors which are not present in the importing country.

With the exception of pathogens which can survive the processes involved in the manufacture of meat and bone meal, for a disease carried in sheep or goat meat to be introduced into an importing country, the pathogen must be one that can infect a pig, dog, cat or other meat-eating animal. **Sheep and goats are extremely unlikely to be infected directly by pathogens carried in meat.**

3.6. COUNTRY FREEDOM STATEMENTS

For some disease agents it may be concluded that the exporting country must be free from the disease for there to be no risk attached to the importation. In such cases, a veterinary certificate should be required confirming country freedom on the date of shipment of the commodity.

3.7. PRELIMINARY HAZARD LIST

The first step in the risk analysis is the identification of agents of concern and the collation of these agents into a preliminary hazard list of pathogens that might be associated with the commodity under consideration. The list of organisms of concern in this risk analysis (Table 1) is taken from a peer-reviewed assessment of the potential risks to animal health from sheep and goat meat published by the OIE (MacDiarmid and Thompson 1997).

Organisms in Table 1 are classified as preliminary hazards (Column 5) if they are:

- Exotic to New Zealand and or there is uncertainty about their status.
- Organisms that occur in New Zealand for which there are known subspecies or strains or host associations that do not occur here but do occur in an exporting country.
- Organisms that occur in New Zealand and the exporting country and for which a Pest Management Strategy under the Biosecurity Act is in place. In this case, measures taken to prevent entry of the organism must not be more stringent than the measures applied in the Pest Management Strategy.

Mycobacterium bovis (bovine tuberculosis) is the only endemic organism classified as a preliminary hazard since a Pest Management Strategy is in place for the disease in cattle and deer.

Disease agents listed in Table 1 are not preliminary hazards if they are:

- Known to occur in New Zealand and do not meet the criteria defined above.
- Organisms transmitted by arthropod vectors

Table 1: List of organisms of concern, adapted from MacDiarmid and Thompson (1997).

Organism	OIE list	Zoonotic	NZ status	Preliminary hazard
Metazoan parasites				
Echinococcus granulosus	Yes	Yes	Exotic	Yes
Viruses				
Aujeszky's disease virus	Yes	No	Exotic	Yes
Foot and mouth disease virus	Yes	No	Exotic	Yes
Maedi-visna virus	Yes	No	Exotic	Yes
Peste des petits ruminants and rinderpest viruses	Yes	No	Exotic	Yes
Capripoxviruses	Yes	No	Exotic	Yes
Bacteria including spirochaetes and rickettsias				
Bacillus anthracis	Yes	Yes	Exotic	Yes
Brucella spp.	Yes	Yes	Exotic	Yes
Coxiella burnetii	Yes	Yes	Exotic	Yes
Leptospira spp.	Yes	Yes	Some serovars exotic	
Mycobacterium bovis	Yes	Yes	Endemic, control programme	
Mycobacterium avium subsp. *Paratuberculosis*	Yes	Probably not	Endemic	No
Mycoplasma agalactiae and *M. capricolum* subsp. *Capripneumoniae*	Yes	No	Exotic	Yes
Salmonella spp.	No	Yes	Some species/serovars exotic	Yes
Prions				
Scrapie	Yes	No	Exotic	Yes
Bovine spongiform encephalopathy	Yes	Yes	Exotic	Yes

Organisms found to be preliminary hazards are listed below:

Metazoan parasites
Echinococcus granulosus

Viruses
Aujeszky's disease virus
Foot and mouth disease virus
Maedi-visna virus
Peste des petits ruminants and rinderpest viruses
Capripoxviruses

Bacteria including spirochaetes and rickettsias
Bacillus anthracis
Brucella spp.
Coxiella burnetii
Leptospira spp.
Mycobacterium bovis
Mycoplasma agalactiae and *M. capricolum* subsp. *capripneumoniae*
Salmonella spp.

Prions
Scrapie
Bovine spongiform encephalopathy

References

Koolmees PA, Tersteeg MHG, Keizer G, van den Broek J, Bradley R (2004). Comparative histological studies of mechanically versus manually processed sheep intestines used to make natural sausage casings. *Journal of Food Protection* 67: 2747-2755.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

MacDiarmid SC, Thompson EJ (2004). Animal health risk analysis. In Jensen WK, Devine C, Dikeman M (eds.) *Encyclopedia of Meat Sciences,* Volume 1. Elsevier Academic Press, Amsterdam: 27-31.

Ruffing M, Windemann H, Schaefer J (2007). Prevention of prion diseases in the production of medicinal products, medical devices and cosmetics. In Hörnlimann B, Riesner D, Kretzschmar H (eds.) *Prions in Humans and Animals.* De Gruyter, Berlin; 529-545.

Wijnker JJ (2009). *Aspects of Quality Assurance in Processing Natural Sausage Casings*. PhD Thesis, Utrecht University: Pp 114. Http:// igitur-archive.library.uu.nl/dissertations/2009-0108-201005/wijnker.pdf. Accessed 14 June 2010.

4. *Echinococcus granulosus*

4.1. HAZARD IDENTIFICATION

4.1.1. Aetiological agent

The cestode *Echinococcus granulosus*.

4.1.2. OIE list

Listed.

4.1.3. New Zealand status

Eradicated from New Zealand. An unwanted, notifiable organism.

4.1.4. Relevant epidemiology

Echinococcosis (hydatids) is a parasitic infestation involving dogs (and some species of wild canids) as primary hosts of the tapeworm *Echinococcus granulosus*, and sheep, goats and other herbivores as the secondary hosts of the cystic larval stages of the same tapeworm (MacDiarmid and Thompson 1997).

Tapeworm eggs are produced by mature adults in the intestine of the primary host, usually the dog, and are ingested by the herbivorous secondary host. Larvae migrate in the body of the secondary host before forming the hydatid cyst. The lifecycle is completed when the carnivorous primary host ingests the cystic stage in the tissues of the secondary host (MacDiarmid and Thompson 1997).

Echinococcosis is a serious zoonosis. Humans may become infested by the cystic stages following ingestion of eggs passed in the faeces of infested dogs (MacDiarmid and Thompson 1997).

The life cycle of *Echinococcus* is dependent on meat serving as a vehicle by which the cystic larval stage (protoscolices) are transmitted to the carnivorous primary host. Most hydatid cysts are found in offal and are only rarely located in muscle tissue (MacDiarmid and Thompson 1997).

The larval stage of *E. granulosus* is the fluid-filled hydatid cyst, the wall of which comprises two layers derived from the parasite surrounded by a dense layer of host-derived connective tissue. Hydatid cysts are found mostly in the liver (approximately two thirds) and the lungs (about a quarter). On rare occasions hydatid cysts may be located in some other organ such as kidney, spleen, bones or brain (Acha and Szyfres 2003). While one cannot completely exclude the possibility that a hydatid cyst could occur in the serosa and muscularis of a sheep's small intestine, this is not very likely and any cyst in these layers would be stripped away in the process of making casings.

4.1.5. Hazard identification conclusion

It is inconceivable that an intact hydatid cyst would remain in processed sausage casings. *Echinococcus granulosus* is not, therefore, a hazard in the commodity.

References

Acha PN, Szyfres B (2003). *Zoonoses and Communicable Diseases Common to Man and Animals.* Third edition. Volume III. Parasitoses. Pan American Health Organization, Washington DC: Pp 395.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

5. Aujeszky's disease virus

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family: *Herpesviridae*; Subfamily: *Alphaherpesvirinae*; Genus: *Varicellovirus*; Species: Suid herpesvirus 1 (Aujeszky's disease virus, pseudorabies virus).

5.1.2. OIE list

Listed.

5.1.3. New Zealand status

Notifiable organism, eradicated from New Zealand.

5.1.4. Relevant epidemiology

Aujeszky's disease, or pseudorabies, is a viral disease which may affect all species of domestic livestock and many species of birds, including poultry. In all species except swine Aujeszky's disease is rapidly fatal.

Cases of Aujeszky's disease in dogs, cats, farmed mink and ferrets, and wild rats have been attributed to the eating of meat from infected swine. Outbreaks in pigs have been attributed to their eating the carcasses of rats dying of the disease. While it is possible that pigs can be infected via pork scraps in swill the lack of prominence given this possibility by most writers suggests that it is not very likely. As Aujeszky's disease is rapidly fatal in sheep and goats it is unlikely that infected animals would be processed for meat (MacDiarmid and Thompson 1997).

When non-porcine hosts such as sheep and goats are infected with Aujeszky's disease virus, the virus is taken up by peripheral sensory nerves and is transported to related ganglia. From there, virus spreads to the spinal cord and brain (Van Oirshot 2004). The small intestine is thus not involved in the pathogenesis of Aujeszky's disease is sheep, nor does viraemia occur.

5.1.5. Hazard identification conclusion

Aujeszky's disease is rapidly fatal in sheep and goats and it is highly unlikely that infected animals would pass ante-mortem inspection if presented for slaughter. The virus is not present in either the blood or small intestine of sheep and goats dying from Aujeszky's disease. Aujeszky's disease virus is not, therefore, a hazard in the commodity.

References

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Van Oirschot JT (2004). Pseudorabies. In Coetzer JAW, Tustin RC. *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 909-918.

6. Foot and mouth disease virus

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: *Picornaviridae*; Genus: *Aphthovirus*, Species: *Foot and mouth disease virus*.

6.1.2. OIE list

Listed.

6.1.3. New Zealand status

An exotic notifiable disease.

6.1.4. Relevant epidemiology

Foot and mouth disease (FMD) is one of the most contagious diseases of domestic animals. It is an acute viral disease which affects a wide variety of domesticated and wild cloven-hoofed animals including cattle, sheep, goats and pigs. FMD is characterised by the formation of vesicles and erosions of the mouth, nose, feet and teats, but infections in sheep and goats are often subclinical.

Many outbreaks of FMD have been traced to waste food being fed to pigs (MacDiarmid and Thompson 1997).

6.1.5. Hazard identification conclusion

Restrictions have been placed on the international trade in sausage casings because of fears that they could spread FMD. However, as Wijnker (2009) points out, there have been very few studies conducted on casings and opinions offered on their potential disease risk have been based on studies carried on other tissues and products and their relevance is questionable. Wijnker et al (2007) are critical of some risk assessments which have accorded a high FMD risk to casings, because those assessments were based on inappropriate inferences drawn from studies conducted on tissues and processes not relevant to casings.

Nevertheless, low titres of FMD virus are found in the cleaned casings prepared from infected animals, most probably originating from the very small amount of blood remaining after processing (Wijnker et al 2007; Wijnker 2009).

Foot and mouth disease virus is, therefore, a potential hazard in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

Low titres of FMD virus are found in the cleaned casings prepared from infected animals, most probably originating from the very small amount of blood remaining after processing

(Wijnker et al 2007; Wijnker 2009). Wijnker cites McKercher as stating that FMD virus remains infectious in untreated processed natural casings for as long as 250 days. However, McKercher provided neither reference to the original studies on casings nor information on the processing and storage conditions (temperature, pH, salt) (Wijnker et al 2007; Wijnker 2009).

However, recent studies have demonstrated that intestines originating from sheep infected with FMD can be effectively treated to remove all infectivity. Casings were rendered free from FMD virus contamination by storage at room temperature (20 °C) for 30 days in salt or in salt supplemented with phosphate (Wijnker et al 2007; Wijnker 2009).

The OIE's *Terrestrial Animal Health Code* (Article 8.5.39.) recommends that casings of small ruminants can be rendered free from FMD virus through salting for at least 30 days with either dry salt (NaCl) or with saturated brine (a_w <0.80), or with phosphate salts/sodium salts mixture, kept at a temperature of about 20°C.

These storage conditions, shown to inactivate all FMD virus in salted casings, are already part of the standard operating procedures of the international casings industry (Wijnker et al 2007; Wijnker 2009) and are incorporated into the commodity definition (see Section 2).

The likelihood of FMD virus would enter New Zealand in the commodity as defined is thus negligible and additional risk management measures are unjustifiable.

References

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Wijnker JJ (2009). *Aspects of Quality Assurance in Processing Natural Sausage Casings.* PhD Thesis, Utrecht University: Pp 114. Http:// igitur-archive.library.uu.nl/dissertations/2009-0108-201005/wijnker.pdf. Accessed 14 June 2010.

Wijnker JJ, Haas B, Berends BR (2007). Removal of foot-and-mouth disease virus infectivity in salted natural casings by minor adaptation of standardized industrial procedures. *International Journal of Food Microbiology* 115: 214-219.

7. Maedi-visna virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family Retroviridae, Genus *Lentivirus*, Maedi-visna virus.

7.1.2. OIE list

Listed.

7.1.3. New Zealand status

Exotic notifiable organism.

7.1.4. Relevant epidemiology

Maedi is a slowly progressive interstitial pneumonia of sheep, and visna is a slowly progressive leucoencephalomyelitis. Both syndromes are caused by the same retrovirus. It is unusual for more than a small proportion of infected animals to develop clinical signs.

Maedi-visna is not highly contagious and prolonged contact appears necessary for transmission of infection. The virus is cell-associated, in monocytes, and is spread mainly through infective droplets during close contact and through colostrum.

It is highly unlikely that meat could serve as a vehicle for the transmission of maedi-visna.

The virus is host specific and carnivores do not become infected (MacDiarmid and Thompson 1997).

7.1.5. Hazard identification conclusion

It is inconceivable that sausage casings could serve as a vehicle for maedi-visna virus; it is not, therefore, a hazard in the commodity.

References

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

8. Peste des petits ruminants and rinderpest viruses

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family Paramyxoviridae, Genus *Morbillivirus*, peste des petits ruminants virus and rinderpest virus.

8.1.2. OIE list

Listed.

8.1.3. New Zealand status

Exotic notifiable organisms.

8.1.4. Relevant epidemiology

Rinderpest is an acute, highly contagious viral disease of cattle and, secondarily, sheep, goats and all cloven-hoofed animals. Swine may become infected and spread the disease. The disease in cattle is characterised by high fever, necrotic stomatitis and gastroenteritis. Mortality in epizootics of rinderpest can be very high; 90% to 100% in naive populations and up to 30% to 50% in enzootic situations (MacDiarmid and Thompson 1997).

Peste des petits ruminants (PPR) is a similar acute disease of sheep and goats caused by a virus closely related to, but distinct from, that of rinderpest. Explosive outbreaks of PPR occur, with mortalities of around 90% (MacDiarmid and Thompson 1997).

In Africa, sheep and goats are said to play a secondary role in maintaining rinderpest. The situation in India is different, with strains of rinderpest distinguishable from PPR virus having become established in sheep and goats (MacDiarmid and Thompson 1997).

The transmission of rinderpest is nearly always dependent on close contact between animals. However, under certain circumstances the virus may persist in meat and infect swine fed on scraps of such meat. Pigs then spread the virus to cattle, sheep, goats and other susceptible ruminants. Such indirect spread is, however, unusual. PPR is said not to spread in this way. Close contact between infected and susceptible live animals is required although pigs can be infected subclinically by experimental inoculation (MacDiarmid and Thompson 1997).

Small ruminants are insufficiently susceptible to maintain rinderpest in the absence of cattle. The clinical signs of rinderpest in sheep and goats vary from the very mild to the severe gastrointestinal disease seen in cattle. The lesions of rinderpest are the direct result of virus-induced cytopathology and virus replication, and hence lesions, are mostly seen in lymphoid tissues and epithelial tissues, especially in the alimentary tract. Infectivity is closely associated with mononuclear leucocytes and is not readily detected in plasma and other body fluids (Rossiter 2004).

8.1.5. Hazard identification conclusion

Because, under certain circumstances, rinderpest virus may persist in meat and infect swine fed on scraps of such meat, it is considered to be a potential hazard in the commodity. PPR virus, which is believed not to spread via meat scraps, is not considered to be a hazard in the commodity.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

On rare occasions rinderpest virus may persist in meat at a titre sufficient to infect swine fed on such meat. The rinderpest virus is fragile and fresh meat poses minimal risk. Frozen meat is more of a risk because virus persists longer. However, virtually all outbreaks of rinderpest in hitherto-free areas have been due to importation of live animals with perhaps only a single outbreak being attributable to importation of meat products (Rossiter 2004).

In the infected animal, rinderpest virus is associated mainly with leucocytes, lymphoid tissue and epithelial tissue. Should an infected sheep or goat pass ante-and post-mortem inspection and its small intestine be harvested for casings, these tissues would be stripped away in the process of casings production.

Animals with clinical signs mild enough to escape post-mortem inspection would have very low titres of rinderpest virus. Once the intestines have been processed into casings, with all mucosa and lymphoid tissues stripped away (Wijnker 2009), any residual virus associated with the product would be in the form of traces of blood. The fragility of rinderpest virus means that infectivity persists for a short time only outside the body (a few hours to as long as four days) (Rossiter 2004) and it is highly probable that it would be destroyed by the 30 day storage period required in the commodity definition, much as Wijnker has demonstrated for FMD virus (Wijnker et al 2007; Wijnker 2009). When kept at the pH optimum for survival, around pH 7.0[3], and a temperature of 25°C, rinderpest virus is inactivated in a few hours (De Boer and Barber 1964; Plowright 1962).

The likelihood that rinderpest virus would enter New Zealand in sheep or goat sausage casings is assessed to be negligible. The imposition of risk management measures is thus unjustifiable.

References

De Boer CJ, Barber TL (1964). pH and thermal stability of rinderpest virus. *Archiv für die gesamte Virusforschung* 15: 98-108.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Plowright W (1962). Rinderpest virus. *Annals of the New York Academy of Sciences.* 10: 548-563.

Rossiter PB (2004). Rinderpest. In Coetzer JAW, Tustin RC. *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 629-659.

[3] The pH of casings during storage is close to neutral; that is, pH 7.0 (Wijnker 2009).

Wijnker JJ (2009). *Aspects of Quality Assurance in Processing Natural Sausage Casings*. PhD Thesis, Utrecht University: Pp 114. Http:// igitur-archive.library.uu.nl/dissertations/2009-0108-201005/wijnker.pdf. Accessed 14 June 2010.

Wijnker JJ, Haas B, Berends BR (2007). Removal of foot-and-mouth disease virus infectivity in salted natural casings by minor adaptation of standardized industrial procedures. *International Journal of Food Microbiology* 115: 214-219.

9. Capripoxviruses

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family Poxviridae, Genus Capripox, sheeppox virus and goatpox virus.

9.1.2. OIE list

Listed

9.1.3. New Zealand status

Exotic notifiable organism.

9.1.4. Relevant epidemiology

Sheeppox and goatpox are acutely progressing diseases characterised by a rise in body temperature and lesions on those parts of the skin devoid of hair covering. The disease may also affect the mucous membranes of the respiratory and gastrointestinal tracts. Sheeppox is probably the most economically serious pox disease of livestock. Goatpox tends to be milder (MacDiarmid and Thompson 1997).

The main routes by which the capripox viruses are transmitted are direct contact with infected animals or contaminated materials, and by the respiratory route (Kitching 2004). It has not been possible to infect animals experimentally by the oral route (MacDiarmid and Thompson 1997).

Most strains of sheeppox and goatpox are highly host specific and pigs, dogs or cats do not appear to be susceptible (MacDiarmid and Thompson 1997).

9.1.5. Hazard identification conclusion

MacDiarmid and Thompson (1997) concluded that sheepox virus and goatpox viruses are unlikely to be introduced in imported meat. Given the processing and storage requirements to produce casings for international trade, there is little likelihood that these viruses would be present in the commodity. Further, as capripox viruses cannot be transmitted by the oral route it is concluded that they are not a hazard in the commodity.

References

Kitching RP (2004). Sheeppox and goatpox. In Coetzer JAW, Tustin RC. *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 1277-1281.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

10. *Bacillus anthracis*

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

The bacterium *Bacillus anthracis.*

10.1.2. OIE list

Listed.

10.1.3. New Zealand status

Exotic notifiable organism.

10.1.4. Relevant epidemiology

Anthrax is an acute, non-contagious bacterial disease of mammals caused by the spore-forming bacterium *Bacillus anthracis*. In its commonest form it is a septicaemia characterised by a rapidly fatal course. While all mammals are susceptible to anthrax, the degree of susceptibility varies. Common domesticated animals may be ranked in order of decreasing susceptibility as; sheep, cattle, goats, horses, pigs, dogs and cats. The course of the disease is rapid in herbivores. Birds are generally more resistant than mammals, but carrion-eating birds may become infected and/or spread anthrax after feeding on infected tissue. Most cases of anthrax result from animals ingesting spores present on feedstuffs but cases may also occur in pigs, dogs and cats following ingestion of meat from animals dying from the disease (MacDiarmid and Thompson 1997).

Pigs and carnivores are highly resistant to anthrax and infection requires the ingestion of large numbers of *B. anthracis* in infected meat (De Vos 2004).

Spores of other bacteria (*Clostridium* spp.) have been found in sheep casings traded internationally (Houben 2005). The salting and storage steps, which are a normal part of casings production, are not sufficient to inactivate the spores of *Clostridium perfringens* which is a natural bowel inhabitant in many animals (Wijnker et al 2006; Wijnker 2009).

10.1.5. Hazard identification conclusion

Because the spores of some bacterial species have been shown to survive in casings traded internationally, *B. anthracis* is considered to be a potential hazard in the commodity.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Sheep or goats infected with anthrax would not pass ante- and post-mortem inspection and so their intestines would not be processed into casings. The likelihood that spores of *B.*

anthracis would enter New Zealand in the commodity is thus negligible and specific measures are unjustifiable.

References

De Vos V, Turnbull PCB (2004). Anthrax. . In Coetzer JAW, Tustin RC. *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 1788-1818.

Houben JH (2005). A survey of dry-salted natural casings for the presence of *Salmonella* spp., *Listeria monocytogenes* and sulphite-reducing *Clostridium* spores. *Food Microbiology* 22: 221-225.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

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Wijnker JJ, Koop G, Lipman LJA (2006). Antimicrobial properties of salt (NaCl) used for the preservation of natural casings. *Food Microbiology* 23: 657-662.

11. *Brucella* spp.

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

The bacteria *Brucella melitensis* and *B. abortus*.

11.1.2. OIE list

Listed.

11.1.3. New Zealand status

Brucella species are exotic, notifiable organisms.

11.1.4. Relevant epidemiology

Brucellosis is a chronic infection caused by bacteria of the genus *Brucella*. The species which primarily affects sheep and goats is *B. melitensis*, although these animals may occasionally be infected with *B. abortus*. *Brucella melitensis* and *B. abortus* may infect pigs and dogs but cats are resistant to infection (MacDiarmid and Thompson 1997).

When animals first become infected with one of the *Brucella* species there is a bacteraemia. Subsequently the organisms localise in certain tissues such as lymph nodes. Pigs and dogs have been reported as becoming infected after eating meat from animals infected *B. suis* and MacDiarmid and Thompson (1997) suggested that, under certain circumstances, meat products might serve as a vehicle for *Brucella* species although, they concluded, this is unlikely with sheep and goat meat products.

11.1.5. Hazard identification conclusion

Because *Brucella* species can cause a bacteraemia in small ruminants and localise in lymphoid tissues they are considered to be a potential hazard in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Because all lymphoid tissue is stripped away during the production of casings (Koolmees et al 2004; Wijnker 2009), any possible *Brucella* contamination of the cleaned casings before salting and storage would have to be from the very small amount of blood remaining after processing, as Wijnker (2009) reported for FMD virus.

However, most bacterial species, including the ones that may be present in the gastrointestinal tract of ruminants, cannot survive a water activity (a_w) less than 0.91 (Wijnker 2009). The casings industry uses either dry salt (a_w 0.75) or saturated brine (a_w between 0.75 and 0.80) for preservation and the normal storage period of 30 days at 20°C

exceeds the minimum period required to eliminate all vegetative bacteria that might be present in the sheeps' casings (Wijnker, Koop and Lipman 2006; Wijnker 2009).

While *Brucella* spp. were not amongst the bacteria specifically studied by Wijnker (Wijnker et al 2006; Wijnker 2009), his conclusions almost certainly apply, as other studies have demonstrated that growth of *B. abortus* and *B. melitensis* is completely inhibited at a concentration of NaCl of 4% (Mitscherlich and Marth 1984), far less than the saturated brine used to store casings[4]. Further, *B. abortus* and *B. melitensis* have been shown not to survive beyond 25 days in sterilized seawater[5] (Mitscherlich and Marth 1984), the salt concentration of which is also less than that used to store casings.

The likelihood that *Brucella* species could enter New Zealand in sheep or goat casings is thus assessed to be negligible.

References

Houben JH (2005). A survey of dry-salted natural casings for the presence of *Salmonella* spp., *Listeria monocytogenes* and sulphite-reducing *Clostridium* spores. *Food Microbiology* 22: 221-225.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Mitscherlich E, Marth EH (1984). *Microbial Survival in the Environment*. Springer-Verlag, Berlin: Pp 802.

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Wijnker JJ, Koop G, Lipman LJA (2006). Antimicrobial properties of salt (NaCl) used for the preservation of natural casings. *Food Microbiology* 23: 657-662.

[4] In Wijnker's studies of the antimicrobial properties of NaCl in casings, the concentrations used ranged between 16% and 36% (Wijnker, Koop and Lipman 2006). In the survey conducted by Houben (2005), concentrations of NaCl in casings traded internationally were between 10% and 40%.

[5] On average, seawater has a salinity of about 3.5%. (http://en.wikipedia.org/wiki/Seawater)

12. *Coxiella burnetii*

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

The rickettsial organism *Coxiella burnetii*.

12.1.2. OIE list

Listed.

12.1.3. New Zealand status

Exotic notifiable organism.

12.1.4. Relevant epidemiology

Coxiella burnetii is the causative agent of Q fever. It is found in most countries (Acha and Szyfries 2003; Kelly 2004) and serological evidence of infection is very common in sheep and goats in some countries (Kelly 2004). Infection in animals is usually inapparent, but clinical disease involving fever, conjunctivitis, arthritis, mastitis, abortions and reproductive disorders is seen occasionally. Cattle, sheep and goats are the species most likely to show clinical signs of Q fever. The disease is a zoonosis. *Coxiella burnetii* is maintained in a wild-life reservoir involving especially rodents and birds. Infection is transmitted to domestic animals, particularly sheep and cattle, by ticks (MacDiarmid and Thompson 1997).

During the bacteraemic phase of the disease, *C. burnetii* is carried to all organ systems and in some animals may persist for months in liver, kidney, muscles and lymph (MacDiarmid and Thompson 1997). Large numbers of *C. burnetii* may be shed in the faeces of infected animals (Kelly 2004).

In experimentally contaminated meat stored under refrigeration, the organism may survive up to 30 days (Mitscherlich and Marth 1984). Humans may, occasionally, become infected by eating infected foodstuffs but this route of infection is uncommon (Acha and Szyfres 2003). MacDiarmid and Thompson (1997) found no reference to meat serving as a vehicle for *C. burnetii* and concluded that this is because it is only milk that serves as a vehicle for oral infection. MacDiarmid (1991) commented that meat meat products are not regarded as a vehicle for *C. burnetii*, as evidenced by the lack of reports of its presence in meat.

12.1.5. Hazard identification conclusion

Coxiella burnetii is found in the faeces of infected animals. In experimentally contaminated meat stored under refrigeration, the organism may survive up to 30 days. Pigs, cats and dogs are susceptible to *C. burnetii*. Infection by the oral route is rare but has been reported. *Coxiella burnetii* is considered a potential hazard in the commodity.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Coxiella burnetii may be present in blood, lymphoid tissue and faeces at various times during the course of infection. However, during the processing of intestines into casings, intestinal contents are discarded, the intestines washed, and lymphoid tissues stripped away. Any *C. burnetii* associated with the product would be in the form of traces of blood.

Meat products have not been regarded as a vehicle for *C. burnetii* and so, naturally, virtually no research has been conducted on the organism's survival in meat products. There is a single report of the organism surviving up to 30 days in experimentally contaminated meat stored under refrigeration. Casings, however, are routinely stored for at least 30 days at around 20°C (Wijnker 2009), rather than at refrigeration temperatures. Survival would be significantly reduced at this higher temperature (see Mitscherlich and Marth 1984).

While *C. burnetii* was not amongst the bacteria specifically studied by Wijnker (Wijnker et al 2006; Wijnker 2009), his conclusions almost certainly apply. Most bacterial species, including the ones that may be present in the gastrointestinal tract of ruminants, cannot survive a water activity (a_w) less than 0.91. Casings are stored at a_w between 0.75 and 0.80 (Wijnker 2009). It is highly probable that the small number of *C. burnetii* that might be present in any traces of blood that might remain when the casings are salted would be completely eliminated during the 30 days storage period.

The likelihood of *C. burnetii* entering New Zealand in the commodity is assessed to be negligible and specific measures are unjustifiable.

References

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MacDiarmid SC (1991). *The Importation into New Zealand of Meat and Meat Products: A Review of the Risks to Animal Health.* Ministry of Agriculture and Fisheries, Wellington.: Pp 180.

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13. *Leptospira* spp.

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Bacteria of the genus *Leptospira.* There are over 200 serovars of *Leptospira*, classified into 23 serogroups.

13.1.2. OIE list

Listed.

13.1.3. New Zealand status

A number of serovars of *Leptopira* are present in New Zealand. Those which are not here are classified by MAF as 'other exotic organisms'.

13.1.4. Relevant epidemiology

Leptopira are fragile organisms which are destroyed rapidly by heating, drying or extremes of pH. However, it is possible that infection may occasionally be spread to carnivores in slaughter scraps from the urinary system. However, because of the short survival time of *Leptopira* in most tissues, kidneys are the only meat products which might serve to introduce new leptospiral serovars (MacDiarmid and Thompson 1997).

Leptospira survive for a few hours only in salt solution (seawater) (Mitscherlich and Marth 1984).

13.1.5. Hazard identification conclusion

Leptospira spp. are not a hazard in the commodity and specific measures are unjustifiable.

References

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Mitscherlich E, Marth EH (1984). *Microbial Survival in the Environment.* Springer-Verlag, Berlin: Pp 802.

14. *Mycobacterium bovis*

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Mycobacterium bovis.

14.1.2. OIE list

Listed.

14.1.3. New Zealand status

Endemic but subject to a Pest Management Strategy under the Biosecurity Act 1993.

14.1.4. Relevant epidemiology

Bovine tuberculosis is a chronic, infectious disease caused by *Mycobacterium bovis* of which cattle are the principal host. Infection may occur in sheep and goats, and humans, dogs, and cats are also susceptible. Infection is rare in sheep (Allen 1988; Davidson et al 1981) and uncommon in goats (Allen 1987; Sanson 1988).

Meat is an unlikely vehicle for tuberculosis. However, transmission through the ingestion of raw meat of tuberculosis-infected animals is possible. Swine may contract tuberculosis after being fed improperly cooked or raw offal from tuberculous cattle (MacDiarmid and Thompson 1997). Francis (1973) stated "... The danger to [humans] from tubercle bacilli in or on flesh is very slight indeed if animals are in reasonably good health at the time of slaughter and good hygienic procedures are observed - this is still generally true even when cattle have quite severe lesions of tuberculosis."

Tubercle bacilli may be found in the lymphoid tissues of infected animals and tuberculous animals may have episodes of bacteraemia.

Palásek et al (1991) reported that approximately 0.3% of swine slaughtered in Czechoslovakia had tuberculous lesions[6] in the intestines. They demonstrated survival of *Mycobacteria* in salted “guts” for up to 7 months. However, the “guts” in the study were not comparable to casings in that only the mucosa had been removed.

14.1.5. Hazard identification conclusion

Although rare in sheep and goats, *Mycobacterium bovis* might be present in intestines of infected animals and so is considered a potential hazard in the commodity.

[6] These lesions were attributable to *Mycobacterium avium* and *M. intracellulare*, not *M. bovis*.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Tuberculosis is rare in sheep and uncommon in goats, so infected animals would seldom be presented for slaughter. Sausage casings, as described in the commodity definition (see above) must have been harvested from animals which have passed ante- and post-mortem inspection. Such inspection would disqualify animals with visible lesions.

Although Palásek et al (1991) reported the survival of *Mycobacteria* in salted pigs' "guts" for up to 7 months, these "guts" were not comparable to sausage casings as defined in that only the mucosa had been removed.

During the processing of intestines into casings, lymphoid tissues, where *Mycobacteria* might be expected to reside, are stripped away. Any residual *Mycobacteria* associated with the product would be in the form of traces of blood. Tuberculous animals may have bacteraemic episodes, although these occur infrequently and involve small numbers of bacilli only. Even in animals dying from miliary tuberculosis, the dose of bacilli present in muscle tissue is only 100-200 per gram (Francis 1973).

Because rare episodes of bacteraemia have been reported in animals with tuberculosis, and because Czech workers have reported the survival of *Mycobacteria* in pigs' "guts", the likelihood of entry is assessed to be extremely low.

14.2.2. Exposure assessment

Only the collagen submucosa remains when sheeps' intestines are processed into casings. These are stored in either saturated brine or salt (Wijnker 2009). It is improbable that a pig, dog or cat would gain access to such a commodity. It is improbable that the commodity would be palatable to these animals either.

There may be an extremely low likelihood that small numbers of *Mycobacteria* could be present in a shipment of casings, but it is stretching credibility to suggest that such contamination might comprise an oral infectious dose. The doses required to infect by mouth are very high, requiring several million bacilli (MacDiarmid and Thompson 1997).

The likelihood that animals in New Zealand could be exposed to an infectious dose of *Mycobacteria* in imported casings made from sheep or goat intestines is assessed to be negligible and so no specific measures are justifiable.

References

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Allen GM (1988). Tuberculosis in sheep-a very rare disease. *Surveillance,* 15(5): 8-9.

Davidson RM, Alley MR, Beatson NS (1981). Tuberculosis in a flock of sheep. *New Zealand Veterinary Journal,* 29: 1-2.

Francis J (1973). Very small public health risk from flesh of tuberculous cattle. *Australian Veterinary Journal,* 49: 496-497.

MacDiarmid SC, Thompson EJ (1997). The potential risks to animal health from imported sheep and goat meat. *Revue Scientifique et Technique de l'OIE* 16: 45-56.

Palásek J, Pavlas M, Kubů I (1991). Survival of salmonellae and Mycobacteria in salted and unsalted swine guts used as sausage casings and sausage emulsion of a hard salami. *Acta Veterinaria Brno*, 60: 375-381.

Sanson RL (1988). Tuberculosis in goats. *Surveillance* 15(2), 7-8: 7-8.

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Sanson RL (1988). Tuberculosis in goats. *Surveillance* 15(2), 7-8: 7-8.

15. *Mycoplasma* spp.

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Mycoplasma agalactiae and *M. capricolum* subsp. *capripneumoniae*, members of the Class Mollicutes.

15.1.2. OIE list

Listed.

15.1.3. New Zealand status

Exotic notifiable organism.

15.1.4. Relevant epidemiology

Contagious agalactia is a severe mastitic and arthritic condition of sheep and goats caused by *Mycoplasma agalactiae*. Contagious caprine pleuropneumonia is a highly contagious, frequently fatal pneumonic condition of goats caused by infection with *M. capricolum* subsp. *capripneumoniae*. *Mycoplasma agalactiae* spreads through being shed in milk, urine, nasal and lacrimal secretions. Contagious caprine pleuropneumonia spreads via the respiratory route and, possibly, also via urine, milk and genital secretions (MacDiarmid and Thompson 1997).

MacDiarmid and Thompson (1997) found no reference to *M. agalactiae* or *M. capricolum* subsp. *capripneumoniae* in meat products. They stated that *M. agalactiae* and *M. capricolum* subsp. *capripneumoniae* are host specific and will not infect pigs, dogs or cats and considered the likelihood of their being introduced through imports of meat products to be negligible.[7]

The mycoplasmas lack cell walls and are bound only by a plasma membrane; they are susceptible to lysis by osmotic shock (Coetzer and Tustin 2004). The prolonged storage in very high concentrations of salt, which has been shown to eliminate all vegetative bacteria that might be present in the sheeps' casings (Wijnker 2009), exerts its effect through osmotic shock and so will certainly eliminate any possible contamination by mycoplasmas (also see Mitscherlich and Marth 1984).

Mycoplasma spp. are not a hazard in the commodity and no measures are justifiable.

[7] MacDiarmid and Thompson (1997) referred to *M. capricolum* subsp. *Capripneumoniae* as *Mycoplasma* species F38 biotype. This designation is now obsolete (Lefèvre and Thiaucourt 2004).

References

Coetzer JAW, Tustin RC (2004). *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 2043-2044.

Lefèvre P-C, Thiaucourt F (2004). Contagious caprine pleuropneumonia. In Coetzer JAW, Tustin RC **(2004)**. *Infectious Diseases of Livestock.* Second edition. Oxford University Press, Oxford: 2060-2065.

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16. *Salmonella* spp.

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

There are approximately 2,500 known serovars in the *Salmonella* genus. Most of these belong to the species *enterica* and the subspecies *enterica*. The serovar names such as 'Dublin' and 'Typhimurium' should not be italicised. The correct name for the serovar Typhimurium is *Salmonella enterica* subsp. *enterica* serovar Typhimurium. However, in the following discussion, for the sake of simplicity names are abbreviated thus; *Salmonella* Typhimurium.

Within each serovar there are multiple strains which can be identified by phage typing. Phage types are identified by the notation DT and a number. *Salmonella* Typhimurium DT104 is of particular significance because it exhibits multiple resistance to the commonly used antibiotics. It is now widely distributed in the world

16.1.2. OIE list

Salmonella Abortusovis is listed. The other *Salmonella* are not.

16.1.3. New Zealand status

Salmonella Dublin is listed as an unwanted notifiable organism. *Salmonella* Typhimurium is endemic in New Zealand but DT104 has been isolated only rarely from humans and once from a dog. *Salmonella* Typhimurium DT104 is classified in the category of "other unwanted organisms". *Salmonella* spp. exotic to New Zealand are classified as other exotic species.

16.1.4. Relevant epidemiology

Salmonellosis is a bacterial infection of many wild and domestic animals as well as humans. It is caused by the numerous species (serotypes, serovars) of the genus *Salmonella*. All *Salmonella* species are pathogenic, with their virulence varying considerably between species. As a zoonosis, salmonellosis is seldom acquired directly; it is usually a foodborne infection (MacDiarmid and Thompson 1997).

Salmonella Abortusovis is a relatively uncommon cause of abortion in ewes. The reservoir of infection is said to be the carrier sheep and organisms are excreted in the faeces and vaginal mucus. Ingestion is probably the main route of infection. *Salmonella* Abortusovis is highly adapted to the sheep and is found only in that species, so pigs, dogs and cats would not be susceptible (MacDiarmid and Thompson 1997).

While almost any foodstuffs, whether vegetable or animal origin, may serve as a vehicle for those salmonellae which are not highly adapted to a particular host, *S.* Abortusovis is not a feedborne pathogen (MacDiarmid and Thompson 1997).

Animals, including sheep and goats, may harbour inapparent *Salmonella* infection, with intermittent shedding of the bacterium in faeces (Acha and Szyfres 2003). During the processing of intestines into casings, the casings are cleaned in batches in water around 40°C and microbiological cross-contamination with organisms such as *Salmonella* occurs (Wijnker 2009).

16.1.5. Hazard identification conclusion

Because intestinal carriage of *Salmonella* is common, and cross-contamination occurs during the processing of casings, the organism is considered a potential hazard in the commodity.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

The basic processing of natural casings involves the removal of faecal material and washing. The casings may be wet packed in saturated brine or packed dry with salt. Natural casings are decontaminated in two of the processing steps. First is the physical removal of bacteria through washing and the second is the destruction of bacteria by high concentrations of salt (Gabis and Silliker 1974).

Wijnker cites a number of studies which are unavailable in English but which showed that storage of casings in brine or dry salt results in the destruction of *Salmonella* within 7 to 30 days (Wijnker 2009). Wijnker's own studies have confirmed the complete elimination of *Salmonella* from casings by the normal salting and storage processes used by the international casings industry. The casings industry uses either dry salt (a_w 0.75) or saturated brine (a_w between 0.75 and 0.80) for preservation and the normal storage period exceeds the minimum 30 days required to eliminate all vegetative bacteria that might be present in the sheeps' casings (Wijnker et al 2006; Wijnker 2009). In an earlier study involving casings contaminated with *Salmonella*, Gabis and Silliker (1974) demonstrated that the organism was eliminated from sheeps' casings after 7 days of being packed in dry salt. They concluded that there would be no *Salmonella* risk in casings traded internationally because of the lengthy period of exposure to saturated brine solution (Gabis and Silliker 1974). This has been confirmed by a survey on internationally-traded dry-salted natural hog and sheep casings which did not detect *Salmonella* contamination in 214 consignments (Houben 2005).

The likelihood of *Salmonella* species entering in the commodity is assessed to be negligible and no specific measures are justifiable.

References

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17. Scrapie

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

The aetiological agent of scrapie is widely, but not universally, believed to be a prion. Prions are said to be agents which are clearly distinguishable from viruses, bacteria and other pathogens in that they are believed to be comprised solely of protein with no nucleic acid content. The prion is generally believed to be a misfolded isomer of PrP, a soluble protein found in cell membranes. The normal form is, by convention, referred to as PrP^{c} while the insoluble misfolded and proteinase resistant isomer is referred to as PrP^{sc}. According to the protein-only hypothesis, PrP^{sc} is the principal or sole component of the scrapie agent (Bradley and Verwoerd 2004a).

17.1.2. OIE list

'Classical' scrapie is listed as reportable to the OIE. So-called 'atypical' scrapie is not reportable to the OIE because it is clinically, pathologically, biochemically and epidemiologically unrelated to 'classical' scrapie, may not be contagious and may be a spontaneous degenerative condition of older sheep.

17.1.3. New Zealand status

Exotic, unwanted organism.

17.1.4. Relevant epidemiology

The epidemiology of scrapie has been thoroughly discussed in a recent import risk analysis conducted by MAF Biosecurity New Zealand (2009).

Scrapie is a transmissible, progressive and invariably fatal neurological disease of sheep and goats which occurs in most sheep-producing countries of the world with the exception of New Zealand, Australia, Argentina (Hörnlimann et al 2007) and South Africa (MAF Regulatory Authority 1999; Bradley and Verwoerd 2004b).

Scrapie is most often seen in sheep between 2 and 5 years of age. Infection is acquired by the oral route, at or shortly after birth, probably from the agent passed in placenta or birth fluids (Hörnlimann et al 2007), although transmission in the milk from infected ewes has been demonstrated (Konold et al 2008).

Following ingestion of the scrapie agent, infection is first established in the Peyer's patches of the small intestine (Bradley and Verwoerd 2004b; Hörnlimann et al 2007).

17.1.5. Hazard identification conclusion

Because scrapie is present in most sheep-producing countries and because the first site of scrapie agent replication is in the Peyer's patches of the small intestine, it is considered a potential hazard in the commodity.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

The scrapie agent is generally considered to be PrP^{sc}, and this is found in the cell membranes of the Peyer's patches of the small intestine. However, in the processing of sheep and goat intestines into casings, Peyer's patches are removed completely after cleaning (Wijnker 2009). A comparative histological study of casings produced from sheeps' intestines showed that no significant differences exist between those cleaned by manual or mechanical techniques and no lymphoid tissue (Peyer's patches) remain after cleaning (Koolmees et al 2004). Wijnker (2009) concluded that casings prepared from sheeps' intestines pose a negligible risk of infectivity from BSE which, like scrapie, may be associated with the lymphoid cells of the Peyer's patches.

The likelihood of scrapie entering New Zealand in sheep or goat casings is assessed to be negligible and no scrapie-specific measures are justifiable.

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18. Bovine spongiform encephalopathy

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

As is the case with the closely-related scrapie, the aetiological agent of bovine spongiform encephalopathy (BSE) is considered to be a prion, a misfolded isomer of PrP, a soluble protein found in cell membranes. The normal form is, by convention, referred to as PrP^{c} while the insoluble misfolded and proteinase resistant isomer is referred to as PrP^{sc}. According to the protein-only hypothesis, PrP^{sc} is the principal or sole component of the BSE agent (Bradley and Verwoerd 2004a).

18.1.2. OIE list

Listed.

18.1.3. New Zealand status

Exotic, unwanted organism.

18.1.4. Relevant epidemiology

First recognised in the UK in 1986, BSE is a transmissible, progressive and invariably fatal neurological disease of cattle which has been spread through the practice of feeding cattle on concentrated feed containing meat-and-bone meal produced from rendering offals of ruminant origin. The first point of entry and replication of the BSE agent is the lymphoid tissue of the Peyer's patches in the distal ileum.

BSE has occurred in a number of other species including several species of zoo ungulates and domestic and zoo cats. Although sheep and goats are susceptible to experimental infection with BSE, only a single field case of BSE has been reported in a goat (Hörnlimann et al 2007). Very extensive active surveillance over several years has failed to detect further cases of BSE in goats and no case has ever been detected in sheep (European Commission 2007, 2008).

18.1.5. Hazard identification conclusion

Because a single case of BSE has been reported in goats, BSE is considered a potential hazard in the commodity.

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

In the European Union, the ileum of sheep and goats is designated as 'specified risk material' (SRM) and is excluded from processing and must be destroyed (Ruffing, Windemann and Schaefer 2007; Wijnker 2009).

The likelihood that a sheep or a goat presented for slaughter would be infected with BSE is vanishingly small. Wijnker (2009) concluded that sheeps' casings have a negligible BSE risk because Peyer's patches are removed completely after cleaning. A comparative histological study of casings produced from sheeps' intestines showed that no significant differences exist between those cleaned by manual or mechanical techniques and no lymphoid tissue (Peyer's patches) remain after cleaning (Koolmees et al 2004).

The likelihood that BSE could enter New Zealand in casings prepared from sheep or goat intestines is assessed to be negligible and no specific measures are justifiable.

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