Import risk analysis:
Deer germplasm

DRAFT FOR PUBLIC CONSULTATION

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Contents

This publication is also available on the MAF website at
http://www.biosecurity.govt.nz/regs/imports/ihs/risk

Executive summary

This risk analysis qualitatively examines the risks involved with the importation of deer germplasm from any country. The germplasm to be imported must be collected and prepared to standards at least equivalent to those recommended in the OIE *Terrestrial Animal Health Code*. Semen to be imported should be frozen and embryos should be derived *in vivo* and frozen.

An extensive hazard list of organisms of potential concern that could be associated with deer germplasm has been collated in Table 1. Preliminary hazards are identified within Table 1 as those that meet specified criteria. *Mycobacterium bovis* is the only endemic organism retained as a preliminary hazard since it is the subject of an official control programme under the Biosecurity Act 1993. Organisms that do not cause diseases of deer and those that cannot be carried in germplasm are excluded. The latter group included all external and internal metazoan parasites and diseases which are transmitted only by arthropod vectors.

The preliminary hazards identified from Table 1 are subjected to individual risk assessments, following the standard process. First, in the hazard identification step, the epidemiology of the disease, including distribution, clinical signs, transmission, diagnosis and any available treatment, is considered. As a result of hazard identification, organisms are classified as either hazards in the commodity, or not.

Organisms identified as hazards in the commodity are subjected to risk assessment to provide a risk estimate by considering the likelihood of entry (the disease agent being present in an animal at the time of importation), exposure (likelihood of spread and establishment if imported) and any adverse consequences likely to follow these events.

For organisms that are classified as a risk by this process, various risk management options are presented. Various options including quarantine, serological testing, agent isolation or identification, treatment and restriction of germplasm donors to those originating from free herds, zones or countries, have been suggested.

When drafting any Import Health Standard (IHS) developed from this import risk analysis, risk management measures selected by the Animal Imports and Exports Section of the Border Standards Directorate of MAF Biosecurity New Zealand will be the least trade restrictive whilst providing a level of protection that is considered appropriate.

The following are classified as risks in the commodity and risk management options have been suggested:

Bovine viral diarrhoea virus 2

Cervine adenovirus

Cervid herpesvirus 1 and *bovine herpesvirus* 1

Foot and mouth disease virus

Lumpy skin disease virus

Peste des petits ruminants virus

Rift Valley fever virus

Vesicular stomatitis virus

Brucella spp.

Chlamydophila abortus

Exotic *Leptospira* serovars

Mycobacterium bovis

Mycoplasma bovis

Mycoplasma mycoides subsp. *mycoides* SC

Coxiella burnetii

Exotic *Salmonella* serotypes and phage types

Chronic wasting disease

1. Introduction

This risk analysis has been written in response to requests received from Mr Greg McKay of Xcell Breeding Services to develop an Import Health Standard (IHS) for elk/wapiti (*Cervus canadensis*) semen and embryos from Korea.

2. Scope and commodity definition

The cervine germplasm to be imported must be prepared to standards equivalent to those recommended for cattle in the OIE *Terrestrial Animal Health Code*[1] (hereafter referred to as the *Code*). Semen to be imported should be frozen and embryos should be derived *in vivo* and frozen. The relevant chapters in the *Code* are 4.5., 4.6. and 4.7.

Since importation of new species of deer would require approval from ERMA, the commodity definition is confined to those species of deer that are already present in New Zealand. At least seven deer species were introduced between 1864 and 1907 and have become established or are farmed. These include red deer (*Cervus elaphus*), sika (*C. nippon*), Virginian or white-tailed (*Odocoileus virginianus*), fallow (*Dama dama*), wapiti/elk (*C. canadensis*), sambar (*C. unicolor*) and the Javan rusa deer (*C. timorensis*). Axis deer and moose were introduced but failed to establish.

'Elk' and 'wapiti' are synonyms for the same species. They occur in North America and in Eastern Asia and several subspecies have been identified. Red deer, which are the most common species in New Zealand, both in the wild and farmed, can cross breed with elk and produce fertile offspring although they are considered a separate species. Some confusion may arise with common names since moose (*Alces alces*) are called elk in Europe.

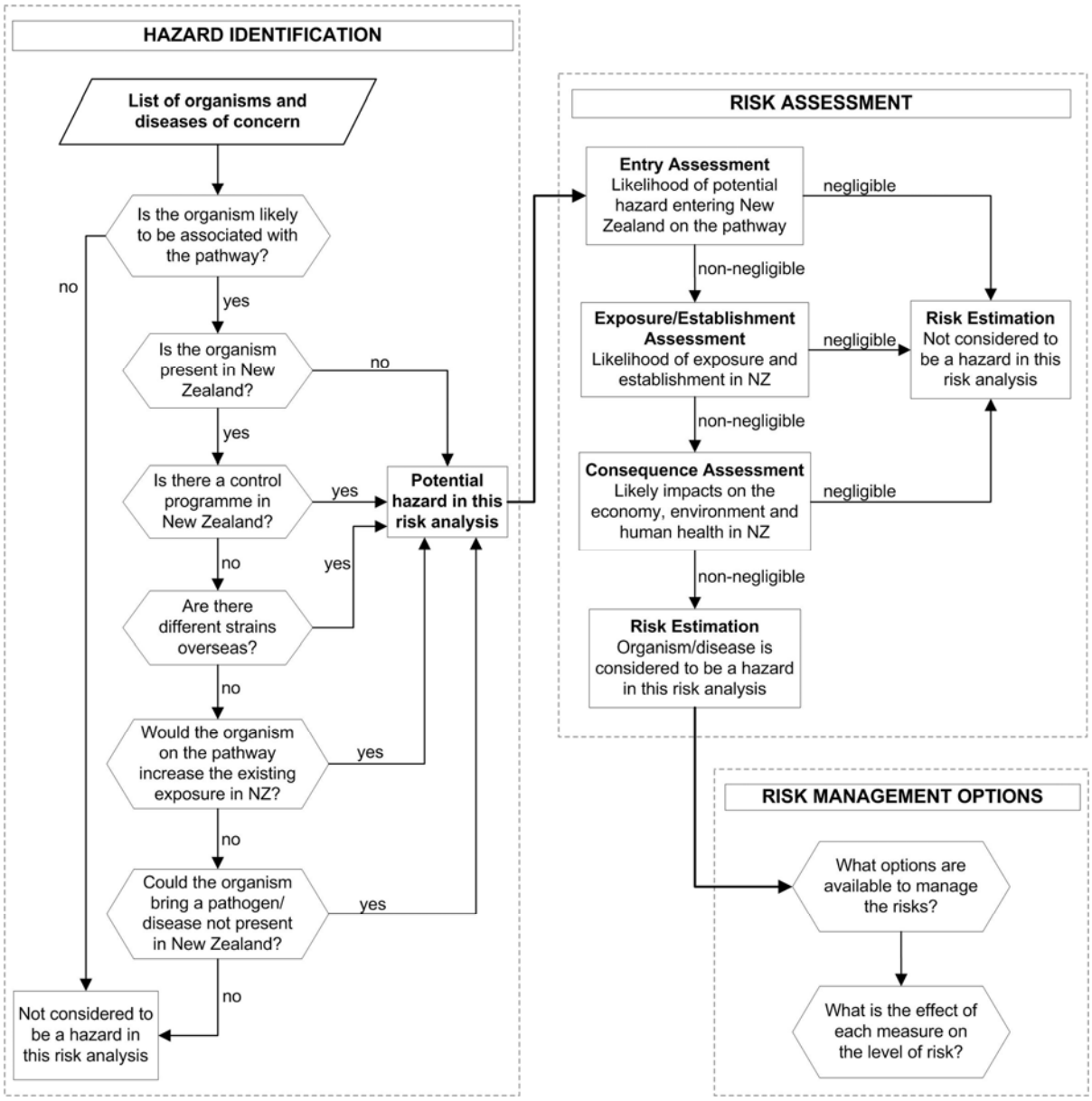
3. Risk analysis methodology

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 the Biosecurity Act 1993.

The process followed is shown in Figure 1.

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

Figure 1 The risk analysis process



3.1. PRELIMINARY HAZARD LIST

Using veterinary texts and electronic databases, an extensive list of organisms known to infect deer has been assembled (organisms of concern). Organisms of concern are identified by applying specific criteria to eliminate those that clearly are not hazards. The remaining organisms are preliminary hazards. These organisms are subjected to 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 hazard or not. All hazards are subjected to risk assessment.

3.3. RISK ASSESSMENT

Risk assessment consists of:

a) *Entry assessment*: The likelihood of a pathogenic organism being imported with the commodity.
b) *Exposure assessment*: The likelihood of animals or humans in New Zealand being exposed to the potential hazard.
c) *Consequence assessment*: The consequences of entry, establishment or spread of an imported organism.
d) *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. RISK COMMUNICATION

MAFBNZ publishes draft import risk analyses for a six-week period of public consultation to verify the scientific basis of the risk assessment and to seek stakeholder comment on the risk management options presented. Stakeholders are also invited to present alternative risk management options that they consider necessary or preferable.

Following public consultation on the draft risk analysis, MAFBNZ produces a review of submissions and determines whether any changes need to be made to the draft risk analysis as a result of public consultation, in order to make it a final risk analysis.

Following this process of consultation and review, the Imports Standards team of MAFBNZ decides on the appropriate combination of sanitary measures to ensure the effective management of identified risks. These are then presented in a draft IHS which is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to the draft IHS are reviewed before a final IHS is issued.

3.6. SPECIAL CONSIDERATIONS

The cervine germplasm to be imported must be prepared to standards equivalent to those recommended in the *Code*.

Therefore, this risk analysis rests on the following internationally-accepted principles that underline the collection and storage of embryos and semen.

• Germplasm is collected and processed at suitable collection centres and laboratories approved for the purpose by the veterinary administration of the exporting country. The place where the collection occurs, the equipment used and the laboratory in which the germplasm is processed is of a standard equivalent to that specified for other livestock species in chapters 4.5., 4.6. and 4.7. of the *Code*.

• Germplasm is collected only from clinically healthy donors.

• If any testing of germplasm is necessary to support certification of the health status of donors, it is carried out at a laboratory approved by the veterinary administration of the exporting country.

• Germplasm is processed, packaged, stored and transported according to standards recommended in the *Code* and by the International Embryo Transfer Society (IETS).

3.7. COUNTRY FREEDOM STATEMENTS

For several disease agents considered in this risk analysis it is concluded that there is no risk attached to the importation on the grounds that the exporting country is free from that disease. For such diseases, a veterinary certificate should be required confirming country freedom on the date of shipment of the commodity.

3.8. 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 organisms that might be associated with deer germplasm.

Deer are cloven hoofed ungulates related to cattle and susceptible to many cattle diseases. For these reasons, the list of agents of concern in the MAF risk analysis for the importation of bovine semen was used as the starting point for identifying the organisms of concern. Diseases specific for deer and not included in the cattle list were added. Specific deer diseases were identified following consultation with experts employed by MAF and external interested parties who were consulted.

Internal and external parasites cannot be transmitted in semen and so are not included in the list.

Organisms/diseases identified from the above sources are listed in Table 1.

The Table indicates whether the organisms are zoonotic and whether they are known to occur in New Zealand.

Organisms in Table 1 are classified as preliminary hazards (Column 5 of Table 1) 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 sub-species or strains or host associations that do not occur in New Zealand 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 for the disease in cattle and deer is in place.

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 for classification as an organism of concern.
- Disease agents of cattle for which no evidence was found that they infect deer and the risk analysis for cattle concluded that there was no risk associated with introducing germplasm from cattle. Agents in this category are marked * in Column 5 of Table 1.
- Organisms transmitted by arthropod vectors and not known to be transmitted in deer germplasm. Organisms in this category were: *Anaplasma centrale*, *A. caudatum* and *A. marginale* (see MAF risk analysis for the importation of bovine semen and reference list), *A. phagocytophilum* (Sumption and Scott 2004), *Babesia bovis*, *B. bigemina* and other *Babesia* spp. (De Vos et al 2004), *Borrelia theileri* (Bishop 2004), *B. burgdorferi* (Hodzic and Barthold 2004), *Ehrlichia chaffeensis* (Yabsley et al 2003; CDC 2009), *Bovine ephemeral fever virus* (St George 2004), *Trypanosoma congolense*, *T. vivax*, and *T. brucei* (Connor and Van den Bossche 2004), *T. evansi* (Luckins 2004), *Theileria Parva*, *T. annulata* and other mildly pathogenic *Theileria* spp. and Tick-borne encephalitis complex viruses (Coetzer and Tustin 2004). These organisms are marked ** in Column 5 of Table 1.
- Protozoan parasites that cannot be transmitted by germplasm because they have a complex life-cycle requiring an intermediate host, or intestinal parasites that are transmitted by the faecal-oral route. The organisms excluded on these grounds were *Besnoitia besnoitii* (Bigalke and Prozesky 2004), *Cryptosporidium parvum*, *Eimeria* spp. (Stewart and Penzhorn 2004) and *Sarcocystis* spp. (Marcus et al 2004). They are marked *** in Column 5 of Table 1.

Table 1 List of organisms and diseases of concern.

ORGANISM	OIE LIST	ZOONOTIC	NZ STATUS	PRELIM HAZARD
VIRUSES				
Akabane virus (Simbu viruses)	No	No	Exotic	No*
Bluetongue virus	Yes	No	Exotic	Yes
Borna disease virus	No	?	Exotic	Yes
Bovine adenovirus viruses	No	No	Endemic (Vermunt and Parkinson 2000a)	No
Bovine enteric calicivirus (tentative species ICTV)	No	No	Unknown	No*
Bovine coronavirus	No	No	Endemic (Durham et al 1979)	No
Bovine ephemeral fever virus	No	No	Exotic	No**
Bovine herpesvirus 1 (IBR/IPV) *Cervine herpesvirus* 1	Yes No	No No	BHV1.2b endemic BHV1.1 and 1.2a exotic Endemic (Tisdall and Rowe 2001)	Yes No
Bovine herpesvirus 2 *Cervid herpesvirus* 2	No No	No No	Endemic (Vermunt and Parkinson 2000b) CHV2 not reported.	No Yes
Bovine leukemia virus	Yes	No	Endemic	No
Bovine parvovirus	No	No	Unknown	No*
Bovine papular stomatitis virus	No	No	Endemic (Vermunt and Parkinson 2000b)	No
Bovine respiratory syncytial disease virus	No	No	Endemic (Motha and Hansen 1997)	No
Bovine rhinoviruses (tentative species ICTV)	No	No	Unknown	No*
Bovine viral diarrhoea virus 1 *Bovine viral diarrhoea virus* 2	Yes	No	BVDV1 endemic BVDV 2 exotic (Yeruham et al 1995)	No Yes
Cervine adenovirus (tentative species ICTV)	No	No	Exotic (not recorded)	Yes
Crimean Congo haemorrhagic fever virus	Yes	Yes	Exotic	Yes
Equine encephalitis viruses (Eastern, Western, Venezuelan)	Yes	Yes	Exotic	Yes
Epizootic haemorrhagic disease virus	Yes	No	Exotic	Yes
Foot and mouth disease virus	Yes	No	Exotic	Yes
Ibaraki virus (strain of epizootic haemorrhagic disease virus)	No	No	Exotic	No*
Jembrana disease virus	No	No	Exotic	No*
Lumpy skin disease virus	Yes	No	Exotic	Yes
Alcelaphine herpesvirus 1 (malignant catarrhal fever)	No	No	Exotic	Yes
Ovine herpesvirus 2 (malignant catarrhal fever)	No	No	Endemic	No
Miscellaneous arboviruses	No	No	All exotic	No*
Rinderpest virus	Yes	No	Exotic	Yes

ORGANISM	OIE LIST	ZOONOTIC	NZ STATUS	PRELIM HAZARD
Peste des petitis ruminants	Yes	No	Exotic	Yes
Palyam virus group	No	No	Exotic (not recorded)	No
Papilloma viruses	No	No	Various	Yes
Parainfluenza virus	No	No	Endemic	No
Pox virus of mule deer (unassigned virus ICTV)	No	No	Exotic	Yes
Pseudocowpox virus	No	Yes	Endemic (Hill 1994)	No
Rabies virus	Yes	Yes	Exotic	Yes
Rift Valley fever virus	Yes	Yes	Exotic	Yes
Ross River virus and *Barmah Forest virus*	No	Yes	Exotic	No*
Rotaviruses	No	No	Endemic (Durham et al 1979)	No
Suid herpesvirus 1 (Aujeszky's disease virus)	Yes	No	Exotic	Yes
Tick-borne encephalitis viruses	No	Yes	Exotic	No**
Vesicular stomatitis Indiana virus and *Vesicular stomatitis New Jersey virus*	Yes	Yes	Exotic	Yes
West Nile virus	Yes	Yes	Exotic	No*
BACTERIA INCLUDING SPIROCHAETES AND RICKETTSIAS				
Actinobacillus lignieresi	No	No	Endemic	No
Anaplasma marginale, A. centrale, A. caudatum	Yes	No	Exotic	No**
Anaplasma phagocytophilum	No	Yes	Exotic	No**
Arcanobacter pyogenes	No	No	Endemic	No
Bacillus anthracis	Yes	Yes	Exotic	Yes
Borrelia burgdorferi	No	Yes	Exotic	No**
Borrelia theileri	No	No	exotic	No**
Brucella abortus *B. melitenisis* *B. suis*	Yes	Yes	Exotic	Yes
Brucella ovis	Yes	No	Endemic	No
Burkholderia pseudomallei	No	Yes	Exotic	No*
Campylobacter fetus subsp. *venerealis*	Yes	No	Endemic (Loveridge and Gardner 1993)	No
Campylobacter jejuni	No	Yes	Endemic	No
Chlamydophila abortus	Yes	Yes	Exotic	Yes
Chlamydophila pecorum	No	No	Endemic (Mackereth and Stanislawek 2002)	No
Clostridium spp.	No	No	Endemic	No
Corynebacterium renale	No	No	Endemic	No
Coxiella burnetti	Yes	Yes	Exotic	Yes
Dermatophilus congolensis	No	Yes	Endemic	No
Ehrlichia ruminantium	Yes	No	Exotic	No**
Ehrlichia chaffeensis	No	Yes	Exotic	No**
Eperythrozoon spp.	No	No	Endemic	No
Escherichia coli	No	Yes	Endemic	No
Footrot associated organisms	No	No	Endemic	No
Haemobartonella bovis	No	No	Unknown	No*

ORGANISM	OIE LIST	ZOONOTIC	NZ STATUS	PRELIM HAZARD
Haemophilus somnus (Histophilus ovis?)	No	No	Endemic	No
Klebsiella spp.	No	No	Endemic	No
Leptospira spp.	Yes	Yes	6 serovars endemic (Midwinter 1999)	Yes
Listeria monocytogenes	No	Yes	Endemic	No
Mannheimia (Pasteurella) haemolytica	No	No	Endemic	No
Moraxella bovis	No	No	Endemic	No
Mycobacterium bovis	Yes	Yes	Endemic/ control programme	Yes
Mycobacterium avium subsp. *avium*	No	Yes	Endemic	No
Mycobacterium avium subsp. *paratuberculosis*	Yes	No?	Endemic	No
Mycoplasma mycoides Subsp. *mycoides* SC	Yes	No	Exotic	Yes
Mollicutes (various)	No	No	Various	Yes
Nocardia spp.	No	No	Endemic	No
Pasteurella multocida B and *E*	Yes	No	Exotic	No*
Pasteurella multocida other than *B* and *E*	No	No	Endemic	No
Salmonella spp.	No	Yes	Some serotypes exotic	Yes
Staphylococcus spp.	No	Variable	Endemic	No
Streptococcus spp.	No	Variable	Endemic	No
Yersinia spp.	No	Yes	Endemic	No
PROTOZOA				
Babesia spp.	Yes	No	Exotic	No**
Besnoitia besnoiti	No	No	Exotic	No***
Cryptosporidium parvum	No	Yes	Endemic	No***
Eimeria spp.	No	No	Endemic	No***
Neospora caninum	No	No	Endemic	No***
Sarcocystis spp.	No	No	?	No***
Theileria parva and *Theileria annulata*.	Yes	No	Exotic	No**
Theileria spp. mildly or non pathogenic	No	No	One species endemic	No**
Trichomonas foetus	Yes	No	Endemic	No
Trypanosoma evansi	Yes	No	Exotic	No**
Trypanosoma spp. tsetse fly-borne	Yes	No	Exotic	No**
PRIONS				
Bovine spongiform encephalopathy	Yes	Yes	Exotic	No*
Chronic wasting disease	No	No	Exotic	Yes

Note: Organisms classified as endemic, for which no reference is given, are commonly identified in New Zealand and reported in the quarterly reports of diagnostic laboratories published in *Surveillance*. For less commonly diagnosed

endemic organisms, a reference is given to substantiate the classification. Palyam viruses have been listed as exotic on the basis that they have not been recorded as occurring in New Zealand. All other organisms listed as exotic have been classified by MAF as unwanted or notifiable organisms (MAF 2009).

Entries marked * in Column 5 are organisms found in cattle or other species but for which no reference has been found suggesting they occur in deer. In addition, the risk analysis for cattle germplasm concluded that the likelihood of introduction in cattle germplasm is negligible.

Entries marked ** in Column 5 are organisms transmitted by arthropod vectors for which no reference has been found suggesting they are transmitted in germplasm.

Entries marked *** are organisms that have a complex life-cycle and are not known to be transmitted in germplasm.

In addition to those agents specified above, the following organisms were excluded from further consideration on the following grounds:

Borna disease virus (*BDV*). Borna disease is not regarded by the OIE as important to trade. Borna disease only occurs sporadically in horses and sheep, and exceptionally in a variety of other animals (including deer) in parts of Germany and surrounding countries (Rott et al 2004). Transmission is not fully understood. However, the virus is thought to be spread by direct contact or from fomites with entry via the nasal neuroepithelium (Gosztonyi 2008). The virus is highly neurotropic and venereal transmission has not been demonstrated (Rott & Herzog 1994; Coetzer & Tustin 2004; Gosztonyi 2008) and no reports could be found suggesting BDV is venereally transmitted. Since deer are extremely rarely reported to be affected, and no evidence could be found that BDV is venereally transmitted, the likelihood of importing BDV in germplasm that has been collected from healthy animals is considered to be negligible.

Rinderpest virus. This has been known to occur in deer (Rossiter 2001) and has been experimentally transmitted to white-tailed deer (Hamdy 1975). However, the disease has been virtually eradicated from the world, with the last case reported from Kenya in 2003. If the virus still exists it is likely that it will be present only in wildlife in East Africa. The global declaration of eradication of rinderpest is expected to occur during 2011. Therefore, the likelihood of introducing the virus in deer germplasm is considered to be negligible.

Organisms found to be preliminary hazards based on the above criteria are listed below:

Viruses

Alcelaphine herpesvirus 1 (Malignant catarrhal fever)

Bluetongue virus

Bovine herpesvirus 1 (Infectious bovine rhinotracheitis)

Cervid herpesvirus 2

Bovine viral diarrhoea virus 2

Cervine adenovirus (Adenovirus haemorrhagic disease)

Crimean Congo haemorrhagic disease virus

Epizootic haemorrhagic disease virus including *Ibaraki virus*

Equine encephalitis viruses

Foot and mouth disease virus

Lumpy skin disease virus

Papilloma viruses

Peste des petitis ruminants virus

Poxvirus of mule deer

Rabies virus

Rift Valley fever virus

Suid herpesvirus 1 (Aujeszky's disease)

Vesicular stomatitis virus

Bacteria including spirochaetes and rickettsias

Brucella spp.

Chlamydophila abortus

Coxiella burnetii

Leptospira (Exotic serovars)

Mycobacterium bovis

Mycoplasma mycoides subsp. *mycoides* SC (Contagious bovine pleuropneumonia)

Mollicutes

Salmonella (Exotic species, serovars and phage types)

Prions

Chronic wasting disease

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4. Alcelaphine herpesvirus 1 (Malignant catarrhal fever)

4.1. HAZARD IDENTIFICATION

4.1.1. Aetiological agent

Family: *Herpesvirida*e; Subfamily: *Gammaherpesvirinae*; Genus: *Rhadinovirus*; Species: *Alcelaphine herpesvirus* (Davison et al 2005).

4.1.2. OIE list

Not listed.

4.1.3. New Zealand status

Alcelaphine herpesvirus 1 is considered to be exotic.

4.1.4. Epidemiology

Malignant catarrhal fever in deer and cattle is caused by *Alcelaphine herpesvirus* 1 (AlHV1) or *Ovine herpes virus* 2. Since the latter is endemic in New Zealand it is not considered a hazard.

AlHV1 is carried subclinically by three species of wildebeest; blue wildebeest (*Connochaetes gnou*), black wildebeest (*C. taurinus taurinus*) and white bearded wildebeest (*C. albojubatus*). AlHV2 is carried by hartebeest and topi (Heuschele and Reid 2001; Reid and Van Vuuren 2004). A closely related virus is carried by roan antelope and recognised by the International Committee on the Taxonomy of Viruses as *Hippotragine herpesvirus* 1. Other viruses possibly exist, each of the species being carried subclinically by its maintenance host. When transmitted to cattle and deer AlHV1 causes an invariably fatal disease but they are dead-end hosts and are not infectious (Heuschele and Reid 2001; Reid and Van Vuuren 2004).

4.1.5. Hazard identification conclusion

AlHV1 cannot establish in the absence of suitable maintenance hosts (wildebeest species) which are not present in New Zealand. Other similar viruses could also not establish in the absence of suitable antelope maintenance hosts. Infected cattle are non contagious dead-end hosts. Therefore, these viruses are not classified as hazards in the commodity.

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5. Bluetongue virus

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family: *Reoviridae*; Genus: *Orbivirus*; Species *Bluetongue virus* (BTV). There are 24 known serotypes (Mertens et al 2005).

5.1.2. OIE list

Listed.

5.1.3. New Zealand status

Listed on the Unwanted Organisms Register as an exotic unwanted organism (MAF 2010).

5.1.4. Epidemiology

BTV can infect many ruminant species. It occurs in most tropical and sub-tropical countries. The global distribution of BTV has historically been between latitudes of approximately 53°N and 34°S but it is known to be expanding in the northern hemisphere. Recently cases occurred in several European countries including countries as far north as Norway, the Netherlands and the UK (OIE 2009). The virus causes disease mainly in sheep, occasionally in goats, and rarely in cattle. In most other species infections are subclinical. It is carried by *Culicoides* spp. (midges) and outbreaks of the disease usually occur in late summer to autumn when midges are most active. Outbreaks of disease cease with the advent of winter when *Culicoides* spp. become inactive. The disease has been experimentally transmitted to deer (Vosdingh et al 1968; Work et al 1992) and virus has been isolated from naturally infected deer (Kocan et al 1987). However, in deer in the USA, bluetongue is less important than epizootic haemorrhagic disease which is caused by a closely related, *Culicoides*-transmitted orbivirus (Maclachan and Osburn 2004). Signs of infection and pathology are similar for both diseases. Cases of bluetongue may be subclinical and serologically positive healthy animals are frequently identified (Odiawa et al 1985; Stallknecht et al 1991). Dual infections with both viruses sometimes occur (Dubay et al 2004).

No information on the presence of the virus in deer semen or embryos was found.

Diagnosis of bluetongue is by isolation of the virus, PCR identification of viral RNA or by serological tests (Daniels and Oura 2009).

5.1.5. Hazard identification conclusion

In view of the above, BTV is classified as a hazard in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry assessment

5.2.2. Semen

No information was found about BTV excretion in deer semen. Therefore, it is necessary to extrapolate from what is known about bull semen, in which excretion of the virus occurs (Parsonson et al 1981). However, the virus is excreted in semen only while the animal is viraemic (Bowen et al 1983; Bowen et al 1985b). The incubation period varies from 2-15 days following experimental infection, and is usually about 7 days in natural infections. Infected cattle remain viraemic for about 50 days (Verwoerd and Erasmus 2004). In countries where many strains of virus are endemic, a few usually dominate in any one season but, as the population becomes immune to these, the strains are replaced by others that then become dominant. In summer, and for a period up to 60 days (incubation period plus viraemic period) after *Culicoides* spp. become inactive at the onset of winter, susceptible animals may be viraemic. The likelihood of collecting infected semen during these periods is assessed to be non-negligible.

5.2.3. Embryos

There is no information on the transmission of BTV in deer embryos and extrapolation from what is known about cattle embryos is necessary. The International Embryo Transfer Society has classified bluetongue in cattle as a Category 1 disease (*Code* Article 4.7.14.). Pathogens are allocated to this category when "sufficient evidence has accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer according to the IETS Manual". For this reason, the likelihood of entry and transmission of BTV by properly prepared embryos is assessed to be negligible.

5.2.4. Exposure assessment

Imported semen will be inseminated into susceptible New Zealand recipients. Therefore, the likelihood of exposure is assessed to be high.

5.2.5. Consequence assessment

Cattle inseminated with contaminated semen became infected and developed viraemia (Bowen and Howard 1984; Bowen et al 1985a; Schlafer et al 1990) and it is probable that deer will also become infected if inseminated with contaminated semen and could be viraemic for up to 50 days. However, since bluetongue is not a contagious disease, they would not transmit the disease to other ruminants. As no reference could be found suggesting iatrogenic transmission of BTV, mechanical transmission is unlikely.

BTV is transmitted by midges of the genus *Culicoides*. A *Culicoides* surveillance programme has been operating in New Zealand since 1991. Sentinel cattle are monitored for seroconversion to viruses transmitted by *Culicoides* spp. (bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses). To date, seroconversion to arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped (Tana and Holder 2007).

The *Code* states that "A BTV free country or zone in which surveillance has found no evidence that *Culicoides* likely to be competent BTV vectors are present, will not lose its free status through the importation of vaccinated, seropositive or infective animals, or semen or embryos/ova from infected countries or infected zones".

Even if BTV were to be introduced it would not be able to establish here as there are no competent vectors.

Bluetongue is not zoonotic so it does not constitute a threat to human health.

Bluetongue is a natural disease of ruminants only and poses no threat to indigenous animals or birds. The effect it might have on thar is not known. However, since vectors for the virus are not found here the consequences of its introduction for the environment would be negligible.

Because New Zealand is free from *Culicoides* spp., the likelihood that the virus could establish in New Zealand is assessed to be negligible. The introduction of BTV in infected semen would not result in the loss of New Zealand's BTV-free status. Therefore, the consequences are assessed to be negligible.

5.2.6. Risk estimation

Since the consequence assessment for the importation of semen is negligible, the risk estimate for introduction of BTV is negligible and it is assessed not to be a risk in the commodity and risk management measures are not justified.

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6. Bovine and Cervid herpesviruses

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agents

Family: *Herpesviridae*; Subfamily: *Alphaherpesvirinae*; Genus: *Varicellovirus*; Species: *Bovine herpesvirus* 1 (BHV1), *Cervid herpesvirus* 1 (CHV1)and *Cervid herpesvirus* 2 (CHV2) (Davison et al 2005).

BHV1 (D'Arce et al 2002) is associated with infectious bovine rhinotracheitis (IBR) (Bitsch 1978) and infectious pustular vulvovaginitis/infectious pustular balanoposthitis (IPV/IPB). Subtypes BHV1.1 and BHV1.2 can be identified by restriction endonuclease analysis of DNA (Babuik et al 2004; Engels et al 1981; Wentink et al 1993). Rhinitis and respiratory signs are associated with subtype 1.1, pustular vulvovaginitis and balanoposthitis is associated with subtype 1.2. Subtype 1.2 strains can be further classified as BHV1.2a and BHV1.2b strains. Some subtype 1.1 and 1.2a strains are abortifacient, as shown by association with clinical cases of abortion and by experimental infection of pregnant heifers (Miller et al 1991). Subtype 1.2b strains are associated with respiratory and genital infections but not with abortions (Miller et al 1991; van Oirschot 1995; van Oirschot 2004). This information is summarised in Table 2.

Table 2 Bovine Herpesviruses

Type	Syndrome			
	IBR	IPV/IPB	Abortion	Encephalitis
BHV1.1	+	-	+	-
BHV1.2a	+	+	+	-
BHV1.2b	+	+	-	-

6.1.2. OIE list

Infectious bovine rhinotracheitis and infectious pustular vulvovaginitis are listed.

6.1.3. New Zealand status

Only BHV1.2b has been isolated in New Zealand (Wang et al 2006). Abortions have not been seen in New Zealand (Fairley 1996; Horner 1990). An attempt to cause abortion by experimental infection with the New Zealand strain of the virus was unsuccessful (Durham et al 1975). A more pragmatic approach is to regard all BHV1.1 and BHV1.2a as exotic organisms. Abortifacient strains are classified as unwanted notifiable organisms.

CHV1 is present in New Zealand (Tisdall & Rowe 2001) but CHV2 has not been identified here. However, this may be due to lack of detailed investigations.

6.1.4. Epidemiology

CHV2 has been found in reindeer and caribou in the artic region. It is associated with respiratory and foetal infections or, more commonly, with subclinical infections (das

Neves et al 2009b). The virus can be transmitted horizontally and become latent in the trigeminal ganglion (das Neves et al 2009a). It has not been reported in species other than reindeer and caribou. CHV are genetically and antigenetically closely related to BHV1 (Deregt et al 2005) and cross-reactions occur in serological tests, but when tests are conducted in parallel with both viruses, stronger reactions occur with homologous antigens (Castro 2001). The viruses found in European red deer and in elk in the USA are similar (Deregt et al 2005) and are possibly the same species. BHV1 and CHV1 are generally associated with inapparent infections and evidence of infection is provided by serological surveys in which there is no definite distinction between CHV1 and BHV1. However, CHV1 may in unusual circumstances cause keratoconjunctivitis (Castro 2001).

No reports were found suggesting that BHVs are pathogens in deer. However, virus isolations, PCR identification of viral DNA or serological investigations indicate that at least white-tailed deer, mule deer, caribou and red deer have been infected with BHV1 (Castro 2001; Kalman and Egyed 2005; Thiry et al 2008). These infections in deer appear to have been subclinical. Because of the paucity of information specific to deer, information in this section is derived primarily from what is known about herpesviruses in cattle.

IBR/IPV has a world-wide distribution. Australia reports that BHV-1.2b is present but BHV-1.1 and BHV-1.2a has never occurred. BHV1.2b is widely endemic in New Zealand (Neilson and Grace 1988). Both the IBR and the IPV syndrome have been described here (Fairley 1996; Horner 1990; Vermunt and Parkinson 2000). However, in the vast majority of cases clinical signs are mild or absent (Vermunt and Parkinson 2000).

The acute disease or infection is of short duration and virus is excreted in nasal secretions for up to 14 days after infection. Viraemia is hard to detect (Babuik et al 2004) but can be confirmed occasionally (van Oirschot 2004). Virus spreads to the conjunctiva and trigeminal ganglion by neuronal axonal transport (van Oirschot 2004). Many animals become persistently infected latent carriers of the virus in their trigeminal or sacral ganglia, and may excrete virus periodically when stressed (Babuik et al 2004; van Oirschot 2004). Subclinically infected bulls may excrete virus in their semen (Babuik et al 2004).

Based on what is known about cattle, it may be assumed that BHV1 occurs as a latent infection in deer too and may be reactivated during times of stress and may also be excreted in their semen.

BHV1 infection can be diagnosed by serological tests, isolation of virus or identification of viral DNA by PCR (Kramps 2009). An internationally validated real time PCR test is now available for detection of BHV1 in semen. PCR tests for BHV1 also detect CHVs, but cannot differentiate between these alphaherpesviruses. The PCR cross-reacts with several other ruminant herpesviruses, including cervine herpesviruses because the PCR detects a highly conserved sequence of herpesvirus gene (Wang et al 2007).

6.1.5. Hazard identification conclusion

Abortifacient strains of IBR/IPV virus are exotic notifiable organisms. Subclinically infected carrier animals occur. Therefore, they are classified as potential hazards. However, since there are no practical tests to identify abortifacient strains, it is necessary to regard all BHV1.1 and BHV1.2a strains and CHV1 and 2 as hazards.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

6.2.1.1. Semen

There are many reports confirming that bulls can shed BHV1 in their semen (de Gee et al 1996; Smits et al 2000; van Oirschot 1995; van Oirschot et al 1993). Therefore, it is likely that subclinically infected stags may excrete the virus in their semen and the likelihood of entry is assessed to be non-negligible.

6.2.1.2. Embryos

BHV1 adheres to the *zona pellucida* of intact embryos but is removed by trypsin treatment and washing (Stringfellow et al 1990; Wrathall et al 2005). The virus is classified by IETS as a Category 1 agent "for which sufficient evidence has accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer according to the IETS Manual" (*Code* Article 4.7.14.). The IETS classification is dependent on the requirement that the embryos are trypsin treated. Therefore, provided the embryos meet the commodity definition and are trypsin treated according to the IETS recommendations, the likelihood of introduction of BHV1 with embryos is assessed to be negligible.

6.2.2. Exposure assessment

Since semen will be inseminated into recipients the likelihood of exposure is certain. Semen contaminated with IBR/ IPV virus is infectious for susceptible recipients (Parsonson et al 1981; Schlafer et al 1990). It has been suggested that as few as 32 virus particles in semen may be required to infect a cow (van Oirschot 1995) and the likelihood that insemination will result in infection of the recipients is high. Recently infected cattle may excrete the virus in nasal secretions and aerosols for up to 14 days after infection (Babuik et al 2004). It is assumed that infected deer could act as BHV1 reservoirs and subsequently infect cattle that are in contact with them.

Therefore, the likelihood of exposure of naïve indigenous deer and cattle to potentially exotic strains of bovine and cervid herpesviruses associated with semen is assessed to be high.

6.2.3. Consequence assessment

There is no evidence to suggest that bovine or cervid herpesviruses would cause significant disease in deer. However, should abortifacient strains of BHV be introduced into New Zealand there would be economic consequences for the cattle industries.

There would be no consequences for people, since bovine and cervid herpesviruses are not zoonoses.

Wild and feral ruminants such as goats and thar could become infected. However, the virus is unlikely to cause significant disease in these animals. The consequences for the environment is therefore assessed to be negligible.

Since introducing abortifacient strains could have negative economic consequences for the cattle industries, the consequences are assessed to be non-negligible.

6.2.4. Risk estimate

Based on the assumption that transmission may occur from infected recipients to other deer and to cattle, the entry, exposure, and consequence assessments are all non-negligible. Therefore, the risk estimate for BHV1 is non-negligible. As such, BHV1 strains are classified as risks in the commodity and risk management measures may be justified.

6.3. RISK MANAGEMENT

6.3.1. Options

The *Code* does not discuss strains of BHVs but instead considers the clinical syndromes of IBR and IPV in cattle. The *Code* makes no recommendations for managing the risk of herpesviruses in deer germplasm but it is reasonable to extrapolate from its recommendations for cattle. The relevant Article for IBR/IPV states:

Article 11.11.7.

Recommendations for the importation of frozen semen

Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that:

1. the donor *animals* were kept in an IBR/IPV free *herd* at the time of collection of the semen; or
2. the donor *animals* were held in isolation during the period of collection and for the 30 days following collection and were subjected to a diagnostic test for IBR/IPV on a blood sample taken at least 21 days after collection of the semen, with negative results; or
3. if the serological status of the bull is unknown or if the bull is serologically positive, an aliquot of each semen collection was subjected to a virus isolation test or PCR, performed in accordance with the *Terrestrial Manual*, with negative results; and
4. the semen was collected, processed and stored in conformity with the provisions of Chapters 4.5. and 4.6.

The following points were considered when developing options for the management of BHV1 in cervine semen:

- The *Code* recommends several options for the importation of frozen semen to prevent introduction of IBR/IPV.
- It is unlikely that there are any deer herds anywhere that are certified as free from BHV1/CHV.
- Serological tests for detecting BHV will also detect CHV.
- A study of seronegative breeding bulls identified virus in semen samples from 50% of animals by virus isolation and from 67% of animals by PCR (Deka et al 2005). Serology, therefore, cannot be relied upon to identify animals excreting virus in their semen.
- Semen could be subjected to a virus isolation test. However, PCR has been reported to be more sensitive for virus detection than virus isolation (Smits et al 2000) and to yield quicker results (de Gee et al 1996). An internationally validated realtime PCR test is available and is a prescribed test when trading bovine semen (Kramps 2009).

One or a combination of the following measures could be considered in order to effectively manage the risks.

1. Since deer appear to be rarely affected by BHV1, and it has not been conclusively shown that they are capable of transmitting infection to other susceptible animals, it could be considered that no measures are necessary.
2. The donor animals originate from a herd where no clinical, microbiological or serological evidence of infectious bovine rhinotracheitis/cervid herpesvirus 2 has occurred for the 12 months prior to the entry of the donor animals into pre-collection isolation.

   N.B. This option reflects the current requirements in the IHS for cervine semen from Great Britain.
3. Donor animals were held in isolation during the period of collection and for the 30 days following collection and were subjected to a diagnostic test for IBR/IPV on a blood sample taken at least 21 days after collection of the semen, with negative results.

   N.B. This option is the *Code* point 2. Serological testing has poor sensitivity and the diagnostic test should be virus isolation or PCR to give equivalence to other options made available.

4. If the serological status of the bull is unknown or if the bull is serologically positive, an aliquot from each semen collection could be subjected to a virus isolation test or PCR, performed in accordance with the *Manual*, with negative results.

   N.B. This option is equivalent to the *Code* point 3. However, virus isolation is not as reliable at detecting infected semen as PCR.

5. Aliquots from each batch of imported semen could be tested for BHV1 with negative results by a virus isolation test. If positive, the virus could be isolated and strain typed before making a decision on whether to allow entry or not.

   N.B. Virus isolation is less sensitive than PCR.
6. Aliquots from each batch of imported semen could be tested for BHV1 with negative results by the internationally recommended PCR test. If positive, the virus could be isolated and strain typed before making a decision on whether to allow entry or not.

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7. Bovine viral diarrhoea virus 2

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: *Flaviviridae*; Genus: *Pestivirus*; Species *Bovine viral diarrhoea virus* 2 (BVDV2) (Thiel et al 2005). Both cytopathic and non-cytopathic biotypes occur.

7.1.2. OIE list

Listed. However, it is not covered by a chapter in the *Code*.

7.1.3. New Zealand status

Bovine viral diarrhoea virus 1 (BVDV1) is endemic but BVDV2 is exotic (MAF 2010).

7.1.4. Epidemiology

BVDV has a world-wide distribution. Only BVDV1 occurs in New Zealand and Australia (Horner 2000; Vilcek et al 1998). Most cattle in New Zealand have been exposed to BVDV1 and the prevalence of antibodies is around 60% (Littlejohns and Horner 1990). The only isolation of a BVDV2 strain in New Zealand was from a batch of foetal calf serum imported from the USA (Horner 2000).

The information below is derived from literature on BVD in cattle, except where otherwise indicated.

BVDV1 infection of non-pregnant cattle usually results in mild clinical signs typified by pyrexia and leukopenia from about 3-7 days. Viraemia and nasal excretion of the virus occur during this period (Brownlie 2005). Clinical signs are often so mild that they are not noticed, or only mild signs and, occasionally, diarrhoea are seen (Potgieter 2004). Since BDV1 is widely distributed in most cattle herds, cattle are commonly infected before they become pregnant. This results in a population of cattle that are predominantly immune and do not carry the virus. Infection of naïve pregnant animals, particularly during the first trimester, may result in death of the conceptus or full term or near full term delivery of immunotolerant persistently infected calves (Brownlie 2005; Littlejohns and Horner 1990; Potgieter 2004; Stokstad et al 2003). It was suggested that 7% of foetal deaths in Swiss dairy cattle may have been caused by infection with BVDV (Rufenacht et al 2001) and BVDV infection around the time of insemination significantly affected breeding performance (McGowan et al 1993). BVDV2 strains that cause a more severe form of the disease have been described in the USA (Pellerin et al 1994). In these cases the mortality rate was up to 10% (Potgieter 2004) and the disease was characterised by severe leucopenia and haemorrhagic signs (Brownlie 2005).

Immunotolerant persistently infected animals may be clinically normal or may fail to thrive and die within a year. They are always infected with non-cytopathic strains of the virus (Brownlie 2005). Superinfection of persistently infected animals with a cytopathic BVDV strain results in the development of mucosal disease (Brownlie 2005; Potgieter 2004). The cytopathic strain that re-infects the persistent carrier animal may result from a mutation of

the persistent non-cytopathic strain or from infection with a new extrinsic cytopathic virus (Brownlie 2005; Potgieter 2004). Mucosal disease invariably ends fatally. In acute cases, death occurs within 2-21 days while in chronic cases the animal may survive for up to 18 months (Potgieter 2004).

Although serologically positive animals are usually no longer infected with virus, exceptions occur and a minority of persistently infected animals are also serologically positive. In addition, in acute cases at the peak of viraemia, antibody may be present before the virus is cleared (Brownlie 2005).

Deer are susceptible to infection, but infections are usually subclinical. Of nine white-tailed deer that were experimentally infected after mating, eight did not produce fawns, although no signs of infection were seen. The remaining hind produced a full term fawn and a mummified foetus. The fawn was persistently infected with BVDV (Passler et al 2007). Serologically positive wild deer have been found and BVDV1a and 1b has been isolated from mule deer and white-tailed deer (Pogranichniy et al 2008; Van Campen et al 2001). A single persistently infected wild mule deer was found amongst 5,597 mule deer, white-tailed deer, elk and moose tested (Duncan et al 2008b). Twin fawns born from a hind that was infected during the first trimester of pregnancy were infected with a non-cytopathic strain at birth (Duncan et al 2008a). Contact between persistently infected cattle and pregnant deer resulted in the birth of persistently infected fawns (Passler et al 2009).

Diagnosis of BVD infection can be made by isolation of the virus from blood or tissues, demonstration of viral RNA by PCR or immunohistochemical demonstration of the viral antigen in tissues, particularly lymph node or skin biopsies. An ELISA or virus neutralisation test can be used for demonstration of antibody (Drew 2008).

7.1.5. Hazard identification conclusion

Deer are susceptible to infection with BVDV. Fawns born from hinds infected during the first trimester of pregnancy may be persistently infected. Although the cases reported were infected with BVDV1, it is likely that deer would be equally susceptible to infection with the more virulent BVDV2 virus. Therefore, BVDV2 is classified as a hazard in the commodity.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

7.2.1.1. Semen

BVDV may be excreted for several months in the semen of persistently infected bulls. Virus isolation methods have detected BVDV for 21 days after infection, but PCR tests were positive for up to 7 months, the duration of an experiment. Virus was detected in the semen of one bull 5 months after infection by inoculating his semen into a calf (Givens et al 2003; Kirkland et al 1991; Lindberg 2005b; Niskanen et al 2002; Voges et al 1998). It is apparent, therefore, that BVDV may be excreted in semen for at least 5 months after infection, despite the bull being seropositive. Therefore, the likelihood of entry of BVDV in bovine semen is assessed to be non-negligible and it should be assumed that deer semen could be similarly contaminated.

7.2.1.2. Embryos

The situation with regard to BVDV contamination of cattle embryos is uncertain. The International Embryo Transfer Society has placed BVDV in Category 3 which comprises "diseases or pathogenic agents for which preliminary evidence indicates that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer according to the IETS Manual, but for which additional *in vitro* and *in vivo* experimental data are required to substantiate the preliminary findings" (*Code* Article 4.7.14.). There is no information about deer embryos.

Some studies showed that washing procedures removed BVDV from bovine embryos (Singh et al 1986) and recipients did not become infected (Bak et al 1992; Brock et al 1997). However, other investigations showed that embryos exposed *in vitro* to BVDV could not be consistently freed from the virus by washing and trypsin treatment (Bielanski and Jordan 1996; Trachte et al 1998). It was also shown that there is a difference in the adhesion of different strains of BVDV to the *zona pellucida* and some are not easily removed by the usual washing procedures (Waldrop et al 2004a; Waldrop et al 2004b). There is also strong circumstantial evidence that a BVDV2 strain of virus was introduced into Britain with embryos imported from the USA (Smith and Grimmer 2000) and that reproductive problems and seroconversion to BVDV in a recipient herd in Sweden were caused by imported embryos contaminated with BVDV (Lindberg 2005b).

Another risk involved in the importation of embryos is that they could become contaminated with BVDV during preparation. Bovine foetal calf serum is commonly used in collection and wash fluids. Since 8-10% of foetuses in endemically infected cattle may be contaminated with BVDV (Lindberg 2005b), it is not surprising that foetal calf serum is commonly contaminated (Makoschey et al 2003). Lindberg has suggested that foetal calf serum used in the preparation of embryos could contaminate them. The concern that embryos could be responsible for the introduction of BVDV into Sweden has resulted in a change in the regulations covering the importation into that country. All recipients of embryos imported into Sweden must be tested serologically 4-12 weeks after embryo transfer to check for seroconversion (Lindberg 2005a; Lindberg 2005b).

Since there is no information relating specifically to deer embryos, it is reasonable to assume that deer embryos are likely to be similar to cattle embryos in respect to the likelihood of their being contaminated with BVDV. Therefore, the likelihood that deer embryos could be infected with BVDV is assessed to be non-negligible.

7.2.2. Exposure assessment

Imported embryos or semen would be inseminated or transferred into recipient females and therefore the likelihood of exposure is assessed to be high.

7.2.3. Consequence assessment

BVDV2 is exotic to New Zealand and, if introduced, it would be expected to spread amongst susceptible deer. Infected deer could transmit the virus to cattle. It is therefore considered that the consequences of introducing the virus would be non-negligible.

The virus does not infect people and there would be no consequences for human health.

The virus could infect feral deer and goats (Horner 2000). It is unlikely it would cause significant disease problems in these animals but they could act as a reservoir of infection

for cattle. The likelihood that there would be any other consequences for the environment is assessed to be negligible.

The consequences for the environment and human health are assessed to be negligible.

7.2.4. Risk estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate for BVDV2 is non-negligible and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

7.3. RISK MANAGEMENT

7.3.1. Options

The following points were considered when drafting options for the management of the BVDV in the commodity:

- Where information on deer is lacking it is reasonable to extrapolate from what is known in cattle. Testing is either carried out on the donors, the germplasm or both.
- There are some differences in antigenicity between BVDV1 and BVDV2 strains and both strains should be used as antigens for serological testing of donor animals when the virus neutralisation test is used (Drew 2008).
- Until recently it was assumed that bulls that are serologically positive are immune and do not excrete the virus. However, a single case of a bull that was serologically positive and had no detectable virus in its blood, but consistently excreted virus in its semen (Voges et al 1998), has led to a change in the European Union Directive on intra-Community trade in imports of bovine semen (Anonymous 2003) and the recommendations of the *Code*. It is now necessary for bulls that are antibody positive when they enter an AI station to have their semen tested for virus and for bulls that seroconvert to have every batch of semen produced since their last negative serological test tested for BVDV (Article 4.5.2.).
- Semen and embryos could be tested directly by virus isolation or RT-PCR.
- Persistent infections may be detected by virus isolation or RT-PCR on blood or by immunohistochemistry on skin (ear notch) or lymph node biopsies.
- Germplasm donors could be tested for viraemia by virus isolation or by RT-PCR.
- Bovine foetal calf serum and other serum products used in processing germplasm could be tested for freedom from BVDV.
- Isolation of BVDV virus or demonstration of viral RNA could require further testing to demonstrate whether it was a BVDV2 virus.

One or a combination of the following options could be considered in order to effectively manage the risk.

1. A straw from each batch of imported semen, and all non-fertilised, degenerated, and *zona pellucida* compromised embryos, collection fluid, and washing fluid (or an embryo from the first embryo collection for each consignment) from each donor could be tested by virus isolation or RT-PCR for BVDV. If a BVDV virus was shown to be present it could be left to the importer to determine whether the importation should be abandoned or whether further testing should be undertaken to determine whether the virus is a BVDV2 virus. If a BVDV2 virus is

demonstrated the importation of the germplasm could be prohibited. If the virus was a BVDV1 virus it could be left to the importer to decide whether to proceed with the importation.

2. Donor animals could be held in isolation from other animals for 3 weeks after collection of the germplasm for further testing as necessary.
3. Potential donors (both male and female) could be tested serologically (by ELISA) and by RT-PCR before germplasm collection immediately before being placed in quarantine.

    i. Animals that are serologically negative and viraemic (PCR positive) could be considered unsuitable for use as donors.
    ii. Those that are serologically positive and PCR negative could be considered suitable for use as donors.
    iii. Animals that are serologically negative and non-viraemic could be tested 3 weeks after germplasm collection. Animals that have remained serologically negative and PCR negative would be considered suitable for use as donors. Animals that have seroconverted while in quarantine would be retained in quarantine for a further 3 weeks before repeating the PCR. Animals that are PCR negative at the second test would be considered suitable donors.

4. Certification could be required confirming that any bovine serum products used in the preparation of the germplasm were tested and found to be free of BVDV.

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8. Cervine adenovirus (Adenovirus haemorrhagic disease)

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family: *Adenoviridae*; Genus: *Atadenovirus*; Tentative species: *Cervine adenovirus* (CAV) (*Odocoileus adenovirus* 1) (Benko et al 2005).

8.1.2. OIE list

Not listed.

8.1.3. New Zealand status

CAV is not known to occur in New Zealand and it is not listed as an unwanted or notifiable organism.

8.1.4. Epidemiology

CAV was described as a cause of adenovirus haemorrhagic disease in California (Boyce et al 2000; Woods 2001; Woods et al 1996). It was reported that in 1993 more than 1,000 deer were estimated to have died in 17 counties of California. An estimated 400 deer died in a three month period in 2002 in the Crooked River Ranch area of Oregon (Oregon Department of Fish and Wildlife 2008) and mortalities occurred in captive moose in Canada (Shilton et al 2002).

The disease has been seen in captive and wild deer. No reports were located of the disease occurring outside of North America. In the systemic form of the disease, erosions, ulceration and abscessation occurred in the upper alimentary tract and haemorrhagic enteritis occurred in some cases. Pulmonary oedema occurred in all cases. Death may occur within a few days. In the localised form of the disease swollen muzzles and oral abscessation is followed by anorexia, emaciation and death. Inapparent infections occur and antibody titres have been found in animals that were not known to have been infected (Boyce et al 2000). Experimental infection of black-tailed deer produced disease with typical signs and lesions (Woods et al 1997; Woods et al 1999). Calves were resistant to experimental infection (Woods et al 2008).

The virus cross-reacts with fluorescein labelled antibody raised against bovine adenovirus 5 and this antibody can be used for immunohistological demonstration of the virus in tissues. Virus was demonstrated in archive tissue of suspected bluetongue cases from 1987. Widespread systemic vasculitis and endothelial intranuclear inclusion bodies were seen. The histological picture was broadly similar to that which occurs in bluetongue. The virus has been identified from black-tailed deer, white-tailed deer, mule deer and moose.

In experimental infections, the incubation period is from 4-16 days. Transmission occurs by direct contact between experimentally infected deer and those in contact with them. Young fawns appear to be more susceptible than yearlings (Woods et al 1997; Woods et al 1999). Transmission is believed to be by contact with body excretions and faeces and, possibly, the airborne route (Oregon Department of Fish and Wildlife 2008; Woods et al

1997; Woods et al 1999). No evidence was found that animals remain chronic carriers or that the virus is transmitted by insect vectors. The period for which animals remain viraemic has not been investigated but evidence suggests that virus can only be demonstrated during the period in which the animals are clinically affected.

Diagnosis is by detection of typical gross lesions and histopathology, with demonstration of the virus in tissues by immunohistochemistry and electron microscopy, and virus isolation in tissue culture (Boyce et al 2000; Sorden et al 2000; Woods 2001; Woods et al 1999; Woods et al 1996). The serum neutralisation test has been used to detect antibody in animals with prolonged infections or recovered animals or those that have had subclinical infections (Boyce et al 2000).

No information was found about transmission of the virus through semen or embryos but it is unlikely that such germplasm would be contaminated with CAV unless collected from animals that were clinically affected and therefore viraemic.

8.1.5. Hazard identification conclusion

CAV is confined to North America. Therefore, it is not a hazard in germplasm from deer from other countries. It is classified as a hazard in deer germplasm imported from North America only.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

There is no evidence that CAV is transmitted by semen or embryos. However, although unlikely, it is possible that semen or embryos from viraemic animals could be contaminated. Therefore, the likelihood that germplasm from deer resident in North America could be contaminated with adenovirus is assessed to be low but non-negligible.

8.2.2. Exposure assessment

It is assumed that insemination or implantation of infected germplasm could result in transmission to the recipients. Since imported germplasm will be used in New Zealand deer, exposure is certain.

8.2.3. Consequence assessment

Deer infected by imported germplasm could develop the disease and transmit infection to other deer. The virus could then be spread and become established in New Zealand, with consequent mortalities and losses in production in farmed deer and mortalities in wild deer.

The virus is not known to infect humans or animals other than deer. Therefore, there would be no consequences for human health or farmed animals, other than deer. The only effect on the environment would be mortalities in wild deer. Since there could be mortalities in deer the consequences are assessed to be non-negligible.

8.2.4. Risk estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate is non-negligible and CAV is classified as a risk in the commodity. Therefore, risk management measures may be justified.

8.3. RISK MANAGEMENT

When considering options for the management of CAV in the commodity the following points were considered:

- The disease occurs only in North America.
- Evidence suggests that the virus can only be demonstrated during the period when the animal is showing clinical signs and it is likely that long-term carriers do not occur.
- Germplasm is likely to be contaminated only during the viraemic period.
- There is no information about the length of the viraemic period but it is likely to be short.
- The virus is unlikely to occur in farmed deer with a long history of freedom from the disease.
- Serological tests are available for the detection of antibodies.

One or a combination of the following options could be considered in order to manage the risk of introducing CAV.

1. Donors of germplasm could be certified as clinically healthy on the day of collection and for 16 days post-collection.
2. Donors of germplasm could be restricted to farmed deer originating from farms on which the disease has not occurred for at least 2 years.
   N.B. The period of 2 years is arbitrary.
3. Donors could be tested with negative results by a serological test; before donation and again 3 weeks after germplasm collection.
4. Germplasm (including male donors used for embryo production) could be restricted to donors originating from countries other than the USA or Canada.

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9. Crimean Congo haemorrhagic fever virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family: *Bunyaviridae*; Genus: *Nairovirus*; Species: *Crimean Congo haemorrhagic fever virus* (CCHFV) (Nichol et al 2005).

9.1.2. OIE list

Listed.

9.1.3. New Zealand status

Classified as an exotic, unwanted organism.

9.1.4. Epidemiology

CCHF occurs in Africa, Asia, the Middle East, and Eastern Europe. The virus can infect humans and a wide variety of ruminants and other smaller animals such as hares. It can also infect ostriches (Swanepoel and Burt 2004). No direct evidence could be found that it infects deer. Serological methods, including ELISA, can be used to detect antibody against CCHFV (Burt et al 1993; Qing et al 2003; Swanepoel and Burt 2004) and PCR methods and viral isolation can be used to detect virus (Burt et al 1998; Schwarz et al 1996). Cattle have often been found to be positive in serological surveys (Burt et al 1996; Mariner et al 1995; Swanepoel and Burt 2004; Swanepoel et al 1987). In humans, the virus causes a serious disease but in most animals it causes a transient, inapparent infection (Swanepoel and Burt 2004).

The principal methods of spread are by tick bite and by contact with infected blood and meat. People involved in slaughtering animals are at risk (Swanepoel et al 1985) and nosocomial infections have occurred in a South African hospital (Shepherd et al 1985). The virus has been isolated from at least 30 species of *Ixodid* ticks, but transmission studies have not been carried out for most and there is no evidence that they are capable of serving as vectors (Swanepoel and Burt 2004). Transovarial transmission of the virus in ticks has been described in a few species of the genera *Rhipicephalus*, *Hyalomma* and *Dermacentor* but it has been suggested that this does not occur regularly and that transstadial infection following amplification in a mammalian host is the usual method of transmission (Swanepoel and Burt 2004). *Hyalomma* spp. are the principal vectors of the disease and the distribution of the disease mirrors the distribution of these ticks (Swanepoel et al 1987). A literature review on the vector competence of the New Zealand cattle tick *Haemaphysalis longicornis* did not suggest this tick to be a competent vector of CCHFV (Heath 2002).

No reference could be found to the incubation period of CCHF in cattle or deer. In humans it is 1-3 days after tick bite infection and can be up to a week in people exposed to infected blood, but incubation periods of up to 9 days have also been reported (Sundberg et al 1985; Swanepoel et al 1989; Swanepoel and Burt 2004). In sheep, the incubation period also appears to be around 3 days (Gonzalez et al 1998). It is assumed that the incubation period

in cattle and deer will be up to 10 days. The viraemic period lasts for up to 7 days (Swanepoel and Burt 2004). There are no reports of long-term carriers in cattle or deer.

9.1.5. Hazard identification conclusion

CCHFV is not known to infect deer. However, many livestock species and other mammals are susceptible and it is assumed that deer are too. CCHFV causes a serious disease in humans. As the virus is not present in New Zealand, but may be introduced with deer germplasm, it is considered to be a hazard in the commodity.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

CCHFV is primarily a tick-borne disease and no evidence suggesting transmission by semen or embryos could be found. There are no long-term carriers and, since viraemia occurs for a period of about 7 days in cattle, it is assumed that germplasm collected from deer collected during a similar viraemic period could lead to contaminated commodity.

The likelihood of collecting germplasm during a viraemic episode is low, but assessed to be non-negligible.

9.2.2. Exposure assessment

The CCHFV is maintained predominantly in a tick-animal cycle and the ticks predominantly responsible for its maintenance (*Hyalomma* spp.) do not occur in New Zealand. The New Zealand cattle tick is not known to be a vector and the virus is found only where *Hyalomma* spp. occur. However, any imported germplasm would be inseminated or implanted directly into susceptible deer and, therefore, exposure is certain. Further, humans exposed to fresh blood and other tissues of infected livestock or, indeed, human patients are at risk of becoming infected.

CCHF is unlikely to establish in New Zealand since a competent tick vector is not present. However, humans exposed to infected animals could become infected with possibly severe consequences, including death. Therefore, exposure is assessed to be non-negligible.

9.2.3. Consequence assessment

The virus causes subclinical infections in livestock, so there are likely to be negligible consequences for the deer industry or for feral or wild animals. However, human health consequences of infection are non-negligible.

9.2.4. Risk estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate is non-negligible and CCHFV is classified as a risk in the commodity. Therefore, risk management measures may be justified.

9.3. RISK MANAGEMENT

When considering options for the management of CCHFV in the commodity the following points were considered:

- Although CCHF is an OIE listed disease, there is no *Code* chapter. Therefore, there are no international standards for managing CCHFV in the commodity.
- CCHF is predominantly tick-borne. Venereal transmission by natural mating or via artificial means has not been reported.
- Viraemic cattle, and probably deer, have short incubation and viraemic periods and long-term carriers of infection do not occur.
- A quarantine period of 21 days, with the donors being kept tick-free, would be adequate to cover the infectious period.
- Serological testing is available and donor animals could be tested before or after collection has been carried out.

One or a combination of the following options could be considered in order to effectively manage the risks.

1. Certification that no case of CCHF has been diagnosed during the previous 21 days where the donors have been resident.
2. Donors could be isolated and treated for ticks, being certified as being maintained free from ticks for 21 days prior to collection.
3. Donors could be tested serologically within 7 days of donating with negative results shown. If serologically positive, the animal could be retested after a further 21 days, with a stable or declining titre being shown before germplasm is eligible for export.
4. Testing as outlined in point 3 except testing could be carried out post-collection.
5. Donors could be certified to have been resident continuously for at least 21 days in a free country or zone before becoming eligible to export germplasm.

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10. Epizootic haemorrhagic disease virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Orbivirus*: Species: *Epizootic haemorrhagic disease virus* (EHDV). There are 10 serotypes/strains of the virus, one of which is Ibaraki virus (Benko et al 2005).

10.1.2. OIE list

Not listed.

10.1.3. New Zealand status

EHDV is listed as an exotic unwanted organism.

10.1.4. Epidemiology

EHDV is an *Orbivirus* transmitted by midges of the genus *Culicoides*. The disease it causes is similar to bluetongue in regard to epidemiology, transmission, clinical disease and pathological changes. It occurs commonly in the United States where it causes mortality in wild deer. Strains of the virus are widely distributed in the world but the disease occurs in wild deer only in the USA and, sporadically, in Canada (Howerth et al 2001).

10.1.5. Hazard identification conclusion

Since EHDV epidemiology is similar to *Bluetongue virus*, being non-contagious and transmitted by *Culicoides* spp. that are not present in New Zealand, it could not establish here. Therefore, it is not classified as a hazard in the commodity.

N.B. For further discussion on *Orbiviruses*, refer to the Bluetongue virus chapter.

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11. Equine encephalitis viruses

11.1.1. Aetiological agent

Family: *Togaviridae*; Genus: *Alphavirus*; Species: *Eastern equine encephalitis virus* (EEV), *Western equine encephalitis virus* (WEE), *Venezuelan equine encephalitis virus* (VEE) (Weaver et al 2005).

11.1.2. OIE list

Listed.

11.1.3. New Zealand status

The equine encephalitis viruses are exotic unwanted organisms.

11.1.4. Epidemiology

EEE and WEE are mosquito-borne diseases that are maintained in bird-mosquito cycles. There is occasional spill-over into horses, humans and other mammals but infected mammals are dead-end hosts in which the virus titre in their blood is too low to infect mosquitoes (Gibbs 2004). The disease has been described in white-tailed deer (Schmitt et al 2007; Tate et al 2005). Deer have not been described as maintenance hosts. The equine encephalitides have never spread beyond northern South America and the USA and it is improbable that competent vectors are present in New Zealand. In the *Code* EEE and WEE are regarded as equine diseases and there are no recommendations relating to germplasm thereby indicating that these viruses are not of concern for trade in these commodities.

VEE is also a mosquito-borne disease. Deer experimentally infected with VEEV developed a transient fever and had a low viraemia for 3 days (Hoff and Trainer 1972). Deer were shown to have antibodies to VEEV and it was suggested they may play a role in the maintenance of the disease (Smart and Trainer 1975). However, no more recent reports supporting this suggestion were located. Rodents are probably the most important vertebrate host but virus, or antibody to virus, has been demonstrated in at least 100 species of birds (Gibbs 2004). The virus has been demonstrated in at least 41 species of mosquito from 11 genera. Horses develop high titres of viraemia and can infect mosquitoes. A wide range of mammals can also be infected. At least two extensive outbreaks of disease have occurred (1969 and 1995) in equids and humans (Gibbs 2004). There are at least eight antigenic variants of the virus. Both endemic and epidemic types of virus have been described and the extensive outbreaks that have occurred are due to epidemic types of virus while endemic types are considered to be avirulent. Viruses of antigenic subtype 1 are considered to have the greatest potential to mutate to become epidemic variants.

For trade purposes, the *Code* considers the incubation period for VEEV to be 5 days and the infective period to be 14 days. No evidence was found that VEEV is transmitted in germplasm. However, the *Code* recommends that:

> "*Veterinary Authorities of VEE free countries may prohibit importation or transit through their territory, from countries considered infected with VEE, of domestic and wild equines, and may prohibit the importation into their territory, from*

countries considered infected with VEE, of semen and embryos/ova of domestic and wild equines".

The *Code* make no reference to animals other than equids, thus indicating that deer and other species are not of concern for international trade.

11.1.5. Hazard identification conclusion

EEV and WEE are diseases of equids that do not occur outside the Americas. Horses are considered to be dead-end hosts and the viruses are not known to be transmitted in semen or embryos. Therefore, EEEV and WEEV are not classified as hazards in the commodity.

VEE is also confined to the Americas and the virus is not known to be transmitted in germplasm. Horses are recognised as important amplifiers of virus. Deer however, have not been described as being involved in the maintenance of the virus. Therefore, VEEV is not classified as a hazard in the commodity.

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12. Foot and mouth disease virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Picornaviridae*; Genus: *Aphthovirus*, Species*: Foot and mouth disease virus* (FMDV) (Stanway et al 2005). There are seven serotypes of the virus: O, A, C, SAT 1, SAT 2, SAT 3, and Asia 1.

12.1.2. OIE list

Listed.

12.1.3. New Zealand status

Listed as an exotic notifiable disease.

12.1.4. Epidemiology

Extensive reviews on FMDV are available (Sanson 1994; Thomson and Bastos 2004; Thomson et al 2001) and much of the information given below is taken from these.

FMD is the most contagious and economically devastating animal disease known. It can infect all cloven hoofed animals. The disease has been reported in at least ten species of deer (Thomson et al 2001).

The outbreaks of the disease in Britain in 2001 (Thompson et al 2002) and in Taiwan in 1997 (Yang et al 1999) cost those countries billions of dollars.

Animals infected with FMD excrete the virus in saliva, faeces, urine, milk, semen and in ocular and nasal discharges (Sanson 1994; Thomson and Bastos 2004; Thomson et al 2001; Yang et al 1999). FMDV is also discharged in aerosol form in expired air. The incubation period is usually 2-14 days (Sanson 1994). Virus can be excreted in semen from 4 days before until 7 days after the onset of clinical signs (Sanson 1994). Viraemia usually continues from 1 day before until 11 days after signs of disease first appear. Transmission can be from direct contact, contact with infected fomites, ingestion of infected animal products, or from inhaling aerosolised virus (Sanson 1994; Thomson and Bastos 2004). Long term carriers excrete small amounts of virus from the pharynx for long periods. Cattle may excrete virus in this way for up to 3 years although the amount of virus excreted is low and the ability of persistently infected cattle to spread the disease is controversial (Thomson and Bastos 2004). Information reviewed by Thomson et al (2001) indicated that fallow and sika deer, and occasionally red deer, may develop persistent infections.

Vaccines are available for the control of FMD but may mask the occurrence of clinical disease in countries where the virus is still circulating. Furthermore, vaccinated animals can be infected and retain the virus in the pharynx for months or years without showing signs of infection (Thomson and Bastos 2004).

When clinical signs are present, FMD can be confirmed by virus isolation, PCR tests and serological tests (Paton et al 2009).

12.1.5. Hazard identification conclusion

FMD is a devastating highly contagious disease and the virus is an exotic, notifiable organism. Therefore, the virus is classified as a hazard in the commodity.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

12.2.1.1. Semen

FMDV is excreted in the semen of bulls during the viraemic period (Callis 1996; Hare 1985; Sellers 1983; Sellers et al 1968). Transmission of the virus to susceptible females can result from insemination with contaminated semen (Callis 1996). It must be assumed that deer also excrete virus in their semen while they are viraemic. The likelihood of entry of virus in semen is assessed to be non-negligible.

12.2.1.2. Embryos

FMD of cattle is classified by IETS as Category 1, that is, a disease "for which sufficient evidence has accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer according to the IETS Manual" (*Code* Article 4.7.14.). The IETS classification is based on several reports that show that FMD virus can be removed from *in vivo* derived embryos by washing and that embryos from viraemic cattle did not infect recipients or calves derived from the embryos (McVicar et al 1986; Mebus and Singh 1991; OIE 2009; Singh et al 1986). The likelihood that *in vivo* derived embryos from infected cattle would be contaminated with FMDV is negligible so long as embryos are processed properly.

However, FMDV cannot be removed from *in vitro* derived embryos by the normally used washing processes (Marguant-Le Guienne et al 1998) and, if trypsin washing is not carried out correctly, virus could still, possibly, contaminate *in vivo* derived embryos.

More significantly, the possibility of FMDV being carried on deer embryos has not been investigated. It cannot be presumed with the same confidence as cattle embryos that deer embryos are unable to transmit infection, even when processed according to IETS recommendations. Therefore, the likelihood of entry is assessed to be non-negligible.

12.2.2. Exposure assessment

Imported semen and embryos would be inseminated or transplanted into susceptible New Zealand animals. Therefore, the likelihood of exposure is assessed to be high.

12.2.3. Consequence assessment

Insemination and transplantation using FMDV-contaminated semen or embryos would be likely to result in infection of recipients (Callis 1996). The infected animals would develop disease and become highly contagious and likely to infect any cloven hoofed animals they came in contact. The further movement of infected animals, airborne and fomite transmission, could rapidly cause a widespread outbreak.

An outbreak of FMD would cause serious disruption to the livestock industries, economic losses to individual farmers, very large expenses for an eradication campaign, and serious disruption to export markets for both animals and animal products. The overall effects

could be catastrophic as demonstrated by the losses resulting from the 2001 outbreak in the United Kingdom, where the costs to government were estimated at 3.1 billion pounds (Thompson et al 2002).

FMD infection of humans is extremely rare and of negligible importance (Sanson 1994). Therefore, there would be no consequences for human health.

The virus infects cloven hoofed animals and could infect feral pigs, goats, and deer thereby establishing the disease in feral populations which would constitute an ongoing source of infection for domestic stock.

Introduction of the disease would have extremely severe effects for individual farmers and the economy of the country. The consequences are assessed to be non-negligible.

12.2.4. Risk estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate for FMDV is non-negligible, and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

12.3. RISK MANAGEMENT

The following points were considered when drafting options for the management of the FMDV risk:

- It could be possible to have a policy of introducing semen and embryos from infected countries if both the donors and germplasm collection centres are free from the virus. Despite the apparent risks, cattle semen has been safely imported from infected countries into the USA over a 10 year period from 1964. Semen was collected from disease-free bulls in semen collection facilities that were maintained free from the disease. In this way 1.7 million doses of semen were safely imported into the USA (Callis 1996). The *Code* gives conditions under which semen can be imported from infected countries into FMD-free countries. These conditions include the stipulation that animals are kept on FMD free premises in an area where no FMD has occurred within a radius of 10 kilometres for the 30 days before collection.
- Serological test are available for diagnosis and could be used on germplasm donors some time after semen collection to ensure that the donors were not incubating the disease at the time of germplasm collection.
- Germplasm from vaccinated animals should not be imported since these animals may carry the virus without showing signs of infection.
- The *Code* recommends that, irrespective of the FMD status of the exporting country, Veterinary Administrations should authorise without restriction the importation of *in vivo* derived cattle embryos provided they were collected and processed according to OIE recommended methods. However, the safety of deer embryos has not been validated. Further embryo transfer procedures depends on the technical and ethical excellence of the individual in charge of the process (Sutmoller and Wrathall 1997; Thibier 2006).

The relevant articles in the *Code* are reproduced below:

Article 8.5.14

Recommendations for importation from FMD free countries or zones where vaccination is not practised

for frozen semen of domestic ruminants and pigs

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that:

1. the donor animals:
   a. showed no clinical sign of FMD on the day of collection of the semen and for the following 30 days;
   b. were kept at least 3 months prior to collection in an FMD free country or *zone* where vaccination is not practised;
2. the semen was collected, processed and stored in conformity with the provisions of Chapters 4.5. and 4.6.

Article 8.5.15.

Recommendations for importation from FMD free countries or zones where vaccination is practised

for semen of domestic ruminants and pigs

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that:

1. the donor animals:
   a. showed no clinical sign of FMD on the day of collection of the semen and for the following 30 days;
   b. were kept for at least 3 months prior to collection in a country or *zone* free from FMD;
   c. if destined to an FMD free country or *zone* where vaccination is not practised:
      i. have not been vaccinated and were subjected, not less than 21 days after collection of the semen, to tests for antibodies against FMD virus, with negative results; or
      ii. had been vaccinated at least twice, with the last vaccination not more than 12 and not less than one month prior to collection;
2. no other animal present in the *artificial insemination centre* has been vaccinated within the month prior to collection;
3. the semen:
   a. was collected, processed and stored in conformity with the provisions of Chapters 4.5. and 4.6.;
   b. was stored in the country of origin for a period of at least one month following collection, and during this period no animal on the *establishment* where the donor animals were kept showed any sign of FMD.

Article 8.5.16.

Recommendations for importation from FMD infected countries or zones

for semen of domestic ruminants and pigs

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that:

1. the donor animals:
   a. showed no clinical sign of FMD on the day of collection of the semen;
   b. were kept in an *establishment* where no animal had been added in the 30 days before collection, and that FMD has not occurred within 10 kilometres for the 30 days before and after collection;
   c. have not been vaccinated and were subjected, not less than 21 days after collection of the semen, to tests for antibodies against FMD virus, with negative results; or
   d. had been vaccinated at least twice, with the last vaccination not more than 12 and not less than one month prior to collection;
2. no other animal present in the *artificial insemination centre* has been vaccinated within the month prior to collection;
3. the semen:

a. was collected, processed and stored in conformity with the provisions of Chapters 4.5. and 4.6.;
b. was subjected, with negative results, to a test for FMDV *infection* if the donor animal has been vaccinated within the 12 months prior to collection;
c. was stored in the country of origin for a period of at least one month following collection, and during this period no animal on the *establishment* where the donor animals were kept showed any sign of FMD

Article 8.5.17.

Recommendations for the importation of *in vivo* derived embryos of cattle
Irrespective of the FMD status of the *exporting country* or *zone*, *Veterinary Authorities* should authorise without restriction on account of FMD the import or transit through their territory of *in vivo* derived embryos of cattle subject to the presentation of an *international veterinary certificate* attesting that the embryos were collected, processed and stored in conformity with the provisions of Chapters 4.7. and 4.9., as relevant.

One or a combination of the following options could be considered in order to effectively manage the risks.

1. Prohibition of importation of germplasm from any country or zone in which FMD is present or any country practising vaccination.

   N.B. This option would be in excess of the requirements of the *Code*.

2. Semen could be imported from countries that are free from FMD and where vaccination is not practised provided the provisions of Article 8.5.14 of the *Code* have been adhered to.

3. Embryos could be imported from any country provided it was prepared in a manner specified in Chapters 4.7. and 4.9. of the *Code* and the donors (both male and female) were tested by a serological test with negative results 4 weeks after the collection of the embryos.

   N.B. Testing of donors after collection of embryos is not required in the *Code* but may be justified as an additional safeguard because of the extreme seriousness of the disease.

4. Testing of samples (non-viable embryos, collection and washing fluids) could be requested to confirm the absence of FMDV (Article 4.7.7.).

5. Semen could be imported from infected countries only if all the conditions of Article 8.5.16. of the *Code* have been followed.

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13. Lumpy skin disease virus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Family: *Poxviridae*; Genus: *Capripox*, lumpy skin disease virus (LSDV).

13.1.2. OIE list

Listed.

13.1.3. New Zealand status

Listed as an exotic, notifiable organism.

13.1.4. Epidemiology

Lumpy skin disease (LSD) is a pox disease of cattle. The disease has generally remained confined to Africa. The most recent outbreaks outside Africa occurred in the Middle East in 2006 and 2007 and in Mauritius in 2008. There are no reports of infection of sheep, goats, or deer. Although very closely related it is probable that sheep and goat pox virus does not occur naturally in cattle and similarly LSDV does not transmit naturally to sheep and goats (Tuppurainen 2010).

Reports of naturally occurring cases of the disease or experimental infection of antelope, oryx, giraffe, and Asian buffalo have been reviewed (Coetzer 2004). However, these cases are rare and there is no evidence that these animals play a significant role in the epidemiology of the disease (Hedger and Hamblin 1983). There are no reports of infection of deer since they are generally not present in the endemic regions of Africa. Since there are no populations of farmed deer, germplasm donors from endemic regions would be confined to zoos or similar collections.

The epidemiology of the disease indicates that it is carried by biting insects but attempts to isolate the virus from *Culicoides*, mosquitoes, ticks, and various other biting flies have generally been unsuccessful. However, Chihota et al (2001) transmitted the disease mechanically with *Aedes aegypti* but failed to transmit it with other potential vectors (Chihota et al 2003). Isolation from *Stomoxys calcitrans* and *Musca confiscata* has been successful but attempts to transmit the disease with these vectors was not (Coetzer 2004). Yeruham et al (1995) describes circumstantial evidence of transmission amongst dairy herds attributed to *Stomoxys calcitrans*. Transmission of the disease by intradermal infection is inefficient but intravenous infection caused typical disease. Spread by contact did not occur (Tuppurainen 2010).

The disease occurs sporadically and only in some years. In the severe South African epidemics of 1989/90 and 2000/2001 the morbidity rates varied 1% up to 20% but mortality was less than 10% (Coetzer 2004). Cattle usually develop a biphasic febrile response 2-4 weeks after experimental exposure to the virus and remain febrile for 4-14

days (Tuppurainen 2010). For the purposes of the *Code* the incubation period is 28 days. Generally signs of disease are typical with eruptions of pox lesions (lumpy lesions) on the skin occurring on or before the second febrile phase. Swollen limbs and lymph nodes also occur and complications can occur when lesions develop in internal organs. Lesions are common in the nasal cavity and muzzle area and may extend into the respiratory tract.

Severe economic losses due to reduced milk production, mastitis and loss of condition have been described. Abortions occurred in 1-7% of cows (Coetzer 2004).

After the development of fever in experimentally infected cattle, virus was demonstrated in semen for 22 days (Weiss 1968). In experimental infections viraemia persisted for 4 days (Coetzer 2004). However, in more recent studies, viral DNA has been demonstrated by PCR in semen for up to 5 months and virus has been isolated from semen up to 42 days after experimental infection (Irons et al 2005). Despite the presence of the virus in semen, there is no definitive evidence on whether semen infected with LSDV can cause clinical disease in the recipient.

Interpretation of serological tests is problematic because some animals only develop low titres after infection. Immunity in recovered cattle is lifelong and long term carriers have not been described (Coetzer 2004; Tuppurainen 2010).

There is good cross-protection between sheep and goat pox virus and lumpy skin disease virus, and sheep pox vaccine can be used to immunise cattle against lumpy skin disease (Capstick et al 1959; Kitching 2003).

13.1.5. Hazard identification conclusion

Lumpy skin disease is an OIE listed disease that is exotic and causes severe economic loss particularly in dairy herds. In general, there are currently no deer populations where the disease is endemic. However, these regions could develop deer industries in the future.

It is not known if deer are susceptible to infection, but it is assumed that they are. Therefore, LSDV is classified as a hazard in the commodity from countries where the virus occurs.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

13.2.1.1. Semen

Lumpy skin disease virus was isolated from bulls semen for 42 days and viral DNA demonstrated in semen for 5 months after experimental infection (Irons et al 2005). However, the disease only occurs in some years in Africa and infected semen would only be collected from recently infected animals.

Although there is no direct evidence of LSDV infecting deer, it is assumed that, similar to cattle, deer could also persistently shed LSDV in semen if infected. Therefore, the

likelihood that the virus will be present in imported deer semen is considered to be low but non-negligible.

13.2.1.2. Embryos

The IETS has not categorised LSD in regards risk of dissemination of the virus by properly processed and handled *in vivo* embryos. However, the *Code* makes recommendations for the safe trade of bovine embryos. Lumpy skin disease is an economically important disease and a conservative stance is justified. It is considered that the likelihood of transmission by embryo transfer is non-negligible.

13.2.2. Exposure assessment

Lumpy skin disease is known to be present in bovine semen and may be transmitted by insemination of infected semen (Hare 1985). There is no information concerning the transmission of the virus by embryo transfer, but the *Code* recommends measures for bovine semen and embryos.

Since germplasm is inseminated or transferred into susceptible recipients the likelihood of exposure is certain.

Since the disease has never spread beyond Africa and the Middle East the likelihood of the disease establishing in New Zealand is probably very low. However, it is assumed that overt disease of the recipient would occur and that LSDV could be subsequently spread by *Stomoxys calcitrans* amongst deer and to cattle.

Therefore, exposure is assessed to be non-negligible.

13.2.3. Consequence assessment

The disease is of economic importance because it causes reduced production particularly in dairy herds (reduced milk yield, mastitis, abortion and infertility in bulls).

Further, a significant number of our trading partners require that, as a condition of continuing trade in germplasm and animals, New Zealand must remain free from the virus. The occurrence of overt cases of the disease in recipients of germplasm would mean that until such time as the position had been clarified and new agreements negotiated, continued trade in germplasm and animals would not be possible with some countries.

The disease is not zoonotic. Therefore, there would be no consequences for human health.

There may be negative impacts on animal health and welfare with lost production particularly for dairy cows. Trade in animals and germplasm would be affected. Therefore, the consequences of introduction of the virus are assessed to be non-negligible.

13.2.4. Risk estimation

Deer are cloven hoofed ungulates related to cattle and susceptible to many cattle diseases.

Under the assumption that LSDV may be present in deer germplasm where the disease occurs and cause clinical disease that is transmissible in the recipients; entry, exposure, and consequence assessments are all non-negligible.

The risk estimate for lumpy skin disease virus is therefore non-negligible. LSDV is classified as a risk in the commodity and risk management measures may be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

The following points were considered when drafting options for the effective management of LSDV in the commodity:

- Very little is known about lumpy skin disease in deer. The recommendations made in the *Code* are for the safe trade in bovines and their semen and embryos, rather than deer or deer germplasm. Although there are no international standards that are directly applicable when trading deer germplasm, it is reasonable to apply recommendations in the *Code*.

- The *Code* recommendations for the importation of bovine semen from infected countries are that donors should be kept in an establishment or artificial insemination centre where no case of lumpy skin disease has been reported for the 28 days prior to collection and that the donors remained free from lumpy skin disease for 28 days after semen collection.

- However, it has been shown that lumpy skin disease virus may be present in bovine semen for at least 42 days after infection and that viral DNA may be present for 5 months (Irons et al 2005).

- In addition to isolation on a semen collection centre for 28 days, semen could be tested directly by virus isolation or by PCR (Irons et al 2005). Alternatively, the donors could be isolated for 6 months prior to semen collection since virus may be present for at least 5 months (Irons et al 2005).

- The *Code* recommendations for the importation of bovine embryos from infected countries are that donors should be kept in an establishment where no case of lumpy skin disease has been reported for the 28 days prior to collection and that the donors showed no clinical signs of LSD on the day of collection and either were vaccinated between 30 and 90 days before collection or were tested negative, or showed a stable titre on paired samples taken 14-60 days apart with one of the samples taken on the day of collection.

- In the case of countries that are free from LSD, the *Code* recommends that the animals should have shown no signs of LSD on the day of collection of semen or for the following 28 days and that the animals should be kept in a LSD free country.

- For embryos from free countries, the donors must have shown no clinical signs of LSD on the day of collection with embryos being collected processed and stored as per *Code* recommendations. These conditions could be applied to germplasm for importation into New Zealand from free countries.

One or a combination of the following measures could be considered in order to effectively manage the risk.

1. Donors could be required to be resident for the 6 months prior to germplasm collection in a country or zone that is free from LSD according to the *Code* definition of freedom.

2. In countries where LSD occurs semen donors could be required to be resident in an establishment or germplasm collection centre that has been free from lumpy skin disease for at least 6 months. All animals on the centre including the donor deer could be required to be free from any sign of lumpy skin disease for at least 28 days after completion of semen collection.

3. For embryo donors in countries where lumpy skin disease occurs, deer could be required to be resident in an establishment where no case of LSD has been reported during the 28 days prior to collection and showed no clinical signs of LSD on the day of collection and either have been vaccinated 30-90 days before collection or tested negative according to the *Manual* or showed a stable titre on paired samples with one of the sample taken on the day of collection.

4. Aliquots of semen and embryo wash fluid and substandard embryos, or an aliquot of embryos, from each batch of imported germplasm could be tested by a PCR method for LSDV DNA.

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14. Peste des petits ruminants virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Paramyxoviridae*; Genus: *Morbillivirus*; Species: *Peste des petits ruminants virus* (PPRV) (Lamb et al 2005).

14.1.2. OIE list

Listed.

14.1.3. New Zealand status

Exotic notifiable disease.

14.1.4. Epidemiology

PPR is a disease mainly of sheep and goats. Other species of the family Bovidae are also susceptible, examples being nilgai, gazelles, ibex and gemsbok (Furley et al 1987). There is little information available on the susceptibility of the family Cervidae. White-tailed deer have been shown to be susceptible to experimental infection (Hamdy and Dardiri 1976) and a report on a website states that PPR is "thought to have caused" an outbreak that affected deer (Spickler 2008). Unfortunately, no primary source for this claim is cited.

The OIE does not mention deer as being naturally infected (OIE 2002). Therefore, although they are experimentally susceptible to infection with PPR virus, it is doubtful that the disease occurs naturally in deer. Information about the disease in deer is limited and information below is extrapolated from what is known about the disease in sheep and goats and from the closely-related rinderpest virus in cattle.

PPR occurs in countries in Central, West and North Africa, the Middle East, Turkey, India, Bangladesh and China (Tibet) (OIE 2002; OIE 2008).

Mortality from PPR in sheep and goats varies from 4-5% in endemic populations to 20-90% in naïve populations (Rossiter 2004). Less virulent strains occur in endemically infected areas and cause mild disease, but it is likely that susceptible New Zealand animals would contract the acute form of the disease.

Infection with PPRV occurs most commonly in the oropharynx and upper respiratory system through inhalation of aerosol particles. The incubation period is from 2-6 days (Rossiter 2004). Primary infection establishes in the pharangeal lymph nodes and tonsils and, following a period of viraemia, in all lymphoid tissues (Rossiter 2004). The viraemic period usually precedes the onset of acute clinical signs and high fever. During the acute phase of the disease infected animals excrete virus in ocular and nasal excretions, urine and faeces (Mushi and Wafula 1984; Rossiter 2004; Wafula et al 1989). This stage may last for about 10 days. Viraemia begins 1-2 days before the onset of illness and declines when circulating antibody first appears (Scott 1990).

Pregnant animals that recover from rinderpest may abort some weeks after recovery and the foetus and vaginal discharges are infected with virus (Rossiter 2004; Wafula et al 1989). However, animals that recover from PPR do not become carriers (Scott 1990).

Vaccination with attenuated rinderpest vaccine provides long-term immunity against both PPR and rinderpest and attenuated and recombinant PPR vaccines are also available (Rossiter 2004).

14.1.5. Hazard identification conclusion

PPR is a highly contagious OIE listed disease that is notifiable in New Zealand. Deer have been shown to be susceptible to experimental infection. Therefore, PPR is considered to be a hazard in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry Assessment

14.2.1.1. Semen

PPR has been listed as a disease in which the virus has been found in small ruminant semen and is likely to be transmitted by semen (Hare 1985). It has also been reported that animals shed the virus in all their excretions and secretions (Scott 1990). Therefore, deer semen should be assumed to be contaminated with PPRV during the acute stage of the disease when infected animals are viraemic. Because viraemia may occur before the onset of clinical signs (Scott 1990), semen from healthy donors could be infected with PPRV.

The entry assessment is therefore assessed to be low but non-negligible.

14.2.1.2. Embryos

It is assumed that the likelihood of transmission PPRV is similar to that of rinderpest. The IETS has classified rinderpest virus into Category 3 which is described as a “disease or disease agent for which preliminary evidence indicates that the risk of transmission is negligible, provided that the embryos are properly handled between collection and transfer, but for which additional *in vitro* and *in vivo* experimental data are required to substantiate the preliminary findings” (*Code* Article 4.7.14.). Extrapolating from this, the likelihood of transmission of PPRV viruses by embryos is assessed as low but non-negligible.

14.2.2. Exposure Assessment

Imported semen or embryos would be transferred into susceptible recipients. Therefore, the likelihood of exposure is high.

14.2.3. Consequence Assessment

Insemination or transplantation of contaminated germplasm into susceptible recipients would be likely to transmit infection (Hare 1985). During the course of the resulting illness animals would be contagious and could infect sheep and goats they are in contact with. The disease could spread rapidly throughout New Zealand causing high morbidity and mortality in sheep and goats. PPR would have serious effects on the economy of individual farms and the country and the productivity of the livestock industries concerned. The ability to export sheep meat would be seriously compromised since the *Code* recommends

that countries importing from infected countries should require certification that "meat products have been processed to ensure the destruction of the PPRV".

PPRV is not zoonotic and there would be no consequences for human health.

Feral sheep, goats, thar and deer could, possibly, become infected and suffer mortalities and become a source of infection. Other non-ruminant animals and birds would not be affected.

Since the introduction of infected germplasm could have serious effects on sheep and goat health and productivity the consequences of introducing the virus are assessed to be high.

14.2.4. Risk Estimation

Since entry, exposure, and consequence assessments are all non-negligible, the risk estimate for PPRV is non-negligible, and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

14.3. RISK MANAGEMENT

The following points were considered when drafting options for the management of the risk of importing PPRV in deer germplasm.

- Deer are susceptible to PPRV infection but cases of natural infection have not been verified.
- Because the incubation period and viraemic period are short, quarantine of germplasm donors would be effective.
- Serological diagnostic test are available but not recommended in the *Code*.
- Vaccines are available and their use is recommended as an option in the *Code*.

The relevant sections for sheep and goats from the *Code* are given below:

Article 14.8.9.

Recommendations for importation from PPR free countries

for semen of domestic small ruminants

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the donor animals:

1. showed no clinical sign of PPR on the day of collection of the semen and during the following 21 days;
2. were kept in a PPR free country for not less than 21 days prior to collection.

Article 14.8.10.

Recommendations for importation from countries considered infected with PPR

for semen of domestic small ruminants

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the donor animals:

1. showed no clinical sign of PPR on the day of collection of the semen and during the following 21 days;
2. were kept in the *exporting country* for the 21 days prior to collection, in an *establishment* or *artificial insemination centre* where no *case* of PPR was officially reported during that period, and that the *establishment* or *artificial insemination centre* was not situated in a PPR *infected zone*;
3. have not been vaccinated against PPR; or
4. were vaccinated against PPR.

Article 14.8.11.

Recommendations for importation from PPR free countries

for embryos of domestic small ruminants and cervids

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that:

1. the donor females were kept in an *establishment* located in a PPR free country at the time of collection of the embryos;
2. the embryos were collected, processed and stored in conformity with the provisions of Chapters 4.7., 4.8. and 4.9., as relevant.

Article 14.8.12.

Recommendations for importation from countries considered infected with PPR

for embryos of domestic small ruminants and cervids

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that:

1. the donor females:
   a. were kept in an *establishment* to which no animals had been added for the 21 days prior to collection;
   b. and all other animals in the *establishment* showed no clinical sign of PPR at the time of collection of the embryos and during the following 21 days;
   c. have been vaccinated against PPR not less than 21 days and not more than 4 months prior to collection; or
   d. have not been vaccinated against PPR and were subjected to a diagnostic test for PPR with negative results at least 21 days after collection;
2. the embryos were collected, processed and stored in conformity with the provisions of Chapters 4.7., 4.8. and 4.9., as relevant.

One or a combination of the following options could be considered in order to effectively manage the risks.

1. Germplasm could be imported without restriction.
   N.B. this option assumes that the likelihood that deer germplasm would be infected with PPRV is negligible.
2. Germplasm could be imported from PPR free countries provided the donors have resided in the country for at least 21 days prior to collection.
3. Germplasm could be imported from countries considered infected with PPRV under the conditions specified in the *Code* Articles 14.8.10 and 14.8.12.

N.B. The *Code* Article 14.8.10. gives an option to be vaccinated against PPR only. However, this must be expanded to stipulate timing of the vaccine since protecting against animals being vaccinated whilst incubating is necessary. Therefore, the vaccine should be administered at least 21 days prior to collection to cover the incubation period and not more than 4 months prior to collection (consistent with Article 14.8.12. for embryo donors).

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15. Papilloma viruses

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agents

Family: *Papillomaviridae*; Genus: *Deltapapillomavirus*; Species: Deer papillomavirus, European elk papillomavirus (de Villiers et al 2005).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Not listed.

15.1.4. Epidemiology

There are a great number of papilloma viruses. Many are insufficiently characterised and their taxonomy is poorly defined and confusing (de Villiers et al 2005). Most cause benign warts in various species of animals. The viruses that are relatively well defined in deer are *Deer papillomavirus* that occur in white-tailed and mule deer (Sundberg et al 1985a; Sundberg and Nielsen 1981; Sundberg and Nielsen 1982; Sundberg et al 1985b) and *European elk papillomavirus* of elk and reindeer (Groff and Lancaster 1985; Moreno-Lopez et al 1987; Stenlund et al 1983). These deer viruses are closely related to bovine papillomavirus 1 (Groff and Lancaster 1985; Moreno-Lopez et al 1987; Stenlund et al 1983). A papillomavirus that occurs in roe deer in Central Europe is closely related to, and is possibly the same species as *European elk papillomavirus* (Erdelyi et al 2009). These viruses cause fibropapillomas on the skin of infected deer, but one case of pulmonary fibroblastomas has been described (Koller and Olson 1971). The lesions usually resolve spontaneously. Identification of fibromas is usually made on the basis of clinical or histological examination of lesions. Virus can be identified in tumour tissue by immunohistochemistry using antibody against bovine papilloma virus 1 and PCR. Laboratory tests are not available for identification of virus in animals without lesions. The disease appears to be confined to certain species of deer only, but detailed knowledge is not available on which species are susceptible. Papilloma virus infection has not been described in red deer. No reference could be found suggesting that the disease occurs in New Zealand.

There is nothing to suggest that infected animals are viraemic or that virus is transmitted in semen or by embryos.

15.1.5. Hazard identification conclusion

The virus causes a mild disease of little economic consequence. There is no indication that viraemia occurs or that the virus can be transmitted by semen or embryos. Therefore, *papillomavirus* is not considered to be a hazard in the commodity.

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16. Poxvirus of mule deer

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Family: *Poxviridae*: Mule deer poxvirus is an unassigned virus within the family (Buller et al 2005).

16.1.2. OIE list

Not listed.

16.1.3. New Zealand status

Poxvirus of mule deer has not been described in New Zealand. *Orf virus* and *Parapoxvirus of red deer* are endemic (Homer et al 1987; Smith et al 1988).

16.1.4. Epidemiology

This poxvirus has been described in two fatal cases in mule deer (Williams et al 1985). An orthopox virus with similar features was isolated from a black-tailed deer that died of adenovirus infection, without showing lesions typical of a poxvirus infection (Patton et al 1996). According to Robinson and Kerr (2001), Barker et al isolated an orthopox virus from a herd of reindeer in the Toronto zoo. No other references to the occurrence of orthopox viruses in deer were located. Clearly the disease is rare and of minor significance. In addition there is no suggestion in the scientific literature that poxviruses are transmitted in semen or embryos.

16.1.5. Hazard identification conclusion

The disease is rare and of limited significance and there is no evidence of transmission in germplasm. Therefore, poxvirus is not classified as a hazard in the commodity.

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17. Rabies virus

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological Agent

Family: *Rhabdoviridae*; Genus: *Lyssavirus*; Species: *Rabies virus* (Tordo et al 2005). In addition to the true *Rabies virus* there are six closely related lyssaviruses which cause similar disease.

17.1.2. OIE list

Listed.

17.1.3. New Zealand status

Rabies virus is an exotic notifiable organism.

17.1.4. Epidemiology

Rabies is a disease of all mammals including humans. It is characterised by severe nervous signs and is invariably fatal. It occurs rarely in deer (South Eastern Cooperative Wildlife Disease Study 1993) and much of what follows has been extrapolated from what is known about rabies in other animals, particularly cattle.

Rabies occurs widely around the world but there are a number of countries, mainly island and peninsular countries, that are disease-free. In some countries, such as Denmark and Australia, that are free from true rabies, bats are endemically infected with closely related lyssaviruses (Swanepoel 2004).

In all endemically infected countries the virus is maintained in a population of domestic or wild carnivores or bats. True rabies in bats is confined to the Americas (Swanepoel 2004) but infections of bats with related lyssaviruses occur in Europe (Fooks et al 2003), Africa (Swanepoel 2004) and Australia (Thompson 1999).

Rabies virus is carried mainly by carnivores. In the final stages of the disease they excrete the virus in their saliva and transmit it to other animals when they bite them. Other forms of transmission, such as aerosol transmission in bat colonies (Swanepoel 2004) and *per os* infection of kudu (Hubschle 1988), are rare exceptions. Following deposition in a bite wound the virus enters peripheral nerves and is transported through the nerves to the central nervous system. Once it enters the peripheral nerves, the virus is not found in any other body tissues or in the blood The passage of virus through the nervous system is slow and, depending on the site of infection, the dose of virus and the animal concerned, the incubation period may vary from weeks to years. In cattle, 2-12 weeks has been reported, but an incubation period of 87 weeks has been reported in a case of experimental infection (Swanepoel 2004). In the *Code* the incubation period is defined as 6 months. Viraemia is an exceptional event except in experimental infections of young mice with large doses of virus (Swanepoel 2004).

The virus spreads to the salivary glands at about the stage that there is generalised dissemination of infection in the brain. It then multiplies in the salivary glands and is excreted

in the saliva. Typically, animals become uncoordinated and aggressive and salivate excessively or develop a paralytic form of the disease (Swanepoel 2004). Ruminants are generally dead-end hosts since they are unlikely to bite other animals.

17.1.5. Hazard identification conclusion

Rabies virus can infect all mammals including humans. It is an important zoonosis and is classified as a hazard in the commodity.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

17.2.1.1. Semen

Contamination of semen with rabies virus has not been described. However, viraemia does not occur except in experimental infections of mice, and the infection of organs other than the nervous system does not occur except in the terminal stages of the disease when the salivary glands and some other organs may be infected (Swanepoel 2004). It is inconceivable that anyone would collect semen from a rabid animal in the final stages of the disease and, therefore, the likelihood of collecting semen contaminated with rabies virus is assessed to be negligible.

17.2.1.2. Embryos

In pregnant females transplacental infection may occur in rare cases due to the immunosuppressive effects of pregnancy (Howard 1981; Martell et al 1973; Sipahioglu and Alpaut 1985). This has been demonstrated experimentally (Swanepoel 2004). However, viraemia and infection of organs other than the central nervous system do not occur except in the terminal stages of the disease when collection of embryos would not be undertaken. The likelihood of embryos being contaminated with rabies virus is assessed to be negligible.

17.2.2. Risk estimation

Since the likelihood of entry of virus in semen or embryos collected from clinically healthy deer is negligible, the risk estimate for rabies virus is negligible and it is not classified as a risk in the commodity. Therefore, risk management measures are not justified.

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18. Rift Valley fever virus

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

Family: B*unyaviridae*; Genus: *Phlebovirus*, species: Rift Valley fever virus (RVFV).

18.1.2. OIE list

Listed as a disease of multiple species.

18.1.3. New Zealand status

Listed as an exotic notifiable organism.

18.1.4. Epidemiology

Rift Valley fever (RVF) is an acute disease of sheep, goats, cattle and humans. The causative agent is an arbovirus carried by mosquitoes. No evidence was found that RVF infects deer. This may be because RVF is endemic in tropical regions of eastern and southern Africa where there are no populations of farmed deer and deer from the region would be confined to zoos or similar collections.

The first recorded outbreak outside the African continent was in 2000 in Saudi Arabia and Yemen. However, the virus has not established in the Arabian peninsula (OIE 2009).

RVF causes massive abortion storms in sheep and deaths in neonatal lambs. In typical outbreaks in southern Africa mortality rates of 5-30% and abortion rates of 40-90% have been reported. In the 1977 outbreak in Egypt up to 60% of sheep died and 80-100% of ewes aborted (Swanepoel and Coetzer 2004). In cattle, the disease is less severe and infection is often subclinical. Mortality varies from 10% for all ages of cattle to 20% for calves but the death rate in an outbreak in Egypt was reported to be 30% (Swanepoel and Coetzer 2004).

Typically the disease is not seen in the years between epidemics. This may be 25-35 year cycles or 5-15 year cycles dependent on exceptionally heavy rain events and sustained flooding of particular regions. The epidemics occur in association with cycles of abnormally heavy rainfall that expand the breeding sites of vector mosquitoes. In Africa, low depression accumulations of water where mosquitoes flourish are known as ‘dambos’ which have specific geographical features. Enormous numbers of aedines emerge from flooded dambos.

It is obligatory that eggs of aedines be subjected to a period of drying as the water recedes before they will hatch on being wetted again when next the dambo floods (Swanepoel and Coetzer 2004).

The exact mechanism by which the virus survives inter-epidemic periods is not known. However, although rarely, transovarial infection occurs in aedine mosquitoes. It has been suggested that because aedine eggs survive for long periods in dried mud and since transovarial transmission occurs this may allow survival of RVFV between epidemics. Further, to prevent the entire mosquito population from being lost when rain has not been sufficient to sustain breeding, only a proportion of eggs hatch at each successive dambo flooding (Swanepoel and Coetzer 2004).

Apart from exceptional rain flooding the dambos, tropical humid weather also favours mosquito breeding and is thought to aid development of infectivity in the mosquito by shortening the extrinsic period. The virus has been isolated from at least 12 species of mosquitoes including members of the genera *Aedes*, *Culex*, *Anopheles,* and *Eremapodites* (Swanepoel and Coetzer 2004).

The incubation period varies from 1-3 days (Geering et al 1995; Mebus 1998; Swanepoel and Coetzer 2004). However, for trade in animals and animal products the *Code* specifies that the incubation period shall be 30 days. Very high titres of virus are found in the blood and viraemia persists for up to 7 days (Swanepoel and Coetzer 2004) whilst virus persists in visceral organs of sheep up to 21 days. Long term carriers of the virus have not been described.

The virus affects humans, infection being from mosquito bites or contact with infected foetuses, raw meat or other infected animal material. In humans there is generally influenza like illness with fever, photophobia, and muscular weakness. In a recent outbreak in Mauritania there were 17 deaths reported, but no denominator data had been included (ProMED 2010).

The total case fatality rate in humans has varied widely between different epidemics but, overall, has been less than one percent in those documented. Thus, in less than 1% of human cases, a haemorrhagic or encephalitic form of the disease develops that causes death.

Both modified live virus vaccines and inactivated vaccines are available. In cattle the use of inactivated vaccine has been recommended (Swanepoel and Coetzer 2004).

Reports of transmission by germplasm were not found.

18.1.5. Hazard identification conclusion

RVF is zoonotic, and an OIE listed disease of multiple species that causes severe disease and economic loss. The virus is exotic and notifiable.

In general, there are currently no deer populations where the disease is endemic. However, these regions could develop deer industries in the future.

It is not known if deer are susceptible to infection, but it is assumed that they are since a range of ruminants and humans are susceptible. Therefore, RVFV is classified as a hazard in the commodity from countries where the virus occurs.

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

18.2.1.1. Semen

The *Code* makes no recommendations for safe trade in ruminant semen and there is no information available about the excretion of virus in semen. However, Hare (1985) lists RVFV as likely to be present in semen and therefore could be transmitted by semen.

It is assumed that the virus would be excreted in deer semen during the viraemic period which lasts for up to 7 days in other ruminant species (Swanepoel and Coetzer 2004). There is a more remote possibility that virus could be excreted in semen during the period of up to 21 days when, although blood is no longer infected, visceral organs may still harbour the virus (Swanepoel and Coetzer 2004). Therefore, the likelihood of virus being introduced with semen is assessed to be non-negligible.

18.2.1.2. Embryos

There is no information about the presence of the virus in embryos. The likelihood that embryos could transmit the virus has not been estimated by IETS. However, the *Code* makes recommendations for trading *in vivo* derived embryos of ruminants. Therefore, it appears that embryos could be infected and the likelihood of entry of virus with embryos is assessed to be non-negligible.

18.2.2. Exposure assessment

Imported semen or embryos would be inseminated or implanted into susceptible recipients. Therefore, the likelihood of exposure is certain.

The epidemiology of this tropical disease is complex and it is unlikely conditions here would allow long-term establishment. At least 12 species of mosquitoes have been found to be infected with the virus (Swanepoel 1994). The endemic mosquito *Aedes notoscriptus* is a laboratory vector of Rift Valley fever virus (Turrell and Kay 1998), but it is not known whether the disease could establish in *Aedes notoscriptus* in New Zealand.

In Africa where the disease is endemic, it is transmitted during epidemics by flood water mosquitoes during seasons of massive build-ups of mosquito numbers. Pharo (1999) reviewed the literature and considered that it was unlikely that the disease could establish in New Zealand.

The disease has never established beyond Africa, therefore, the likelihood of establishment is assessed to be very low.

However, it is assumed that infection of the recipient would occur and that infected animals and their products would pose a human health threat.

Therefore, exposure is assessed to be non-negligible.

18.2.3. Consequence assessment

It is assumed that germplasm from viraemic deer donors could contain virus and if inseminated or implanted into susceptible recipients could lead to infection of the recipients. If this occurred infected recipients could carry the virus in their organs for up to 21 days (Swanepoel and Coetzer 2004). However, during this period they would not be contagious and would not infect in contact animals. While they are viraemic, recipients could infect competent vector mosquitoes. It is not known whether the disease could establish in *Aedes notoscriptus* in New Zealand.

A significant number of our trading partners require that, as a condition of continuing trade in germplasm and animals, New Zealand must remain free from the virus. The occurrence of overt cases of the disease in recipients of germplasm would mean that until such time as the position had been clarified and new agreements negotiated, continued trade in germplasm and animals would not be possible with some countries. Additionally, *Code* certification requirements would be applied to milk and meat, and to milk and meat products for export.

The virus is zoonotic and it could be expected that people would be occupationally exposed. Humans are most likely to become infected by mosquito inoculation or by contact with infected animals, carcasses/meat, abortion material, or laboratory samples. The majority of these infections would result in a flu-like illness but a small percentage of cases could progress to serious disease and death. Therefore, infection would have consequences for human health.

The disease is known to infect domestic ruminants but has not been described in any animals found in New Zealand except sheep, goats, and cattle. Therefore there would be no consequences for the environment other than possibly for feral goats and thar.

In conclusion, RVF is zoonotic and there may be negative impacts on animal health and welfare with lost production particularly for sheep, goats and to a lesser extent the cattle industries. Trade in animals, germplasm and animal products would be affected.

Therefore, the consequences of introduction of RVFV are assessed to be non-negligible.

18.2.4. Risk estimation

Deer are cloven hoofed ungulates related to cattle and susceptible to many cattle diseases.

Under the assumption that RVFV may be present in deer germplasm where the disease occurs and causes clinical disease in recipients; entry, exposure, and consequence assessments are all non-negligible.

The risk estimate for RVFV is therefore non-negligible. It is classified as a risk in the commodity and risk management measures may be justified.

18.3. RISK MANAGEMENT

18.3.1. Options

The following points were considered when drafting options for the effective management of RVFV in the commodity:

- The *Code* makes recommendations when trading *in vivo* derived ruminant embryos, but not for semen.

- Rift Valley fever has a short incubation period (12-36 hours) and the period of viraemia is of short duration (up to 7 days). For the purposes of the *Code* the incubation period shall be 30 days. Long-term carriers of virus do not occur. Therefore, quarantine of donors protected from mosquitoes is an effective means of preventing the importation of infected germplasm.

- Infected countries remain free from disease for periods of several years during periods when mosquito activity is low. The *Code* refers to infected, disease free countries and recommends that animals can be safely traded from such countries if they have been in such a country for 6 months during which time there have been no climate changes predisposing to outbreaks of RVF; or were vaccinated 21 days prior to shipment; or were held in mosquito-proof premises for 30 days prior to shipment. These recommendations could be applied directly to donors of germplasm.

- The *Code* recommendations for *in vivo* derived ruminant embryos are that donors are to be certified as having no evidence of RVF within the period from 28 days prior to 28 days following collection of the embryos, and were vaccinated at least 21 days prior to collection; or were serologically tested on the day of collection and at least 14 days following collection with no significant rise in titre shown.

One or a combination of the following measures could be considered in order to effectively manage the risk:

1. Germplasm donors could be required to have resided for the 30 days prior to the collection of germplasm and during germplasm collection in a Rift Valley fever-free country or zone.

2. Germplasm donors could be required to have resided for the 6 months prior to and during the collection of germplasm in a RVF infected country in which climatic changes predisposing to outbreaks of RVF have not occurred in the previous 6 months.

3. Germplasm donors could be required to have been held in mosquito-free premises for at least the 30 days prior to the collection of germplasm and during germplasm collection.

4. Donors are to be certified as having no evidence of RVF within the period from 28 days prior to 28 days following collection of germplasm; and be vaccinated at least 21 days prior to collection; or be serologically tested on the day of collection and at least 14 days following collection with no significant rise in titre shown.

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19. Suid herpesvirus 1 (Aujeszky's disease)

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Family: *Herpesviridae*; Subfamily: *Alphaherpesvirinae*; Genus: *Varicellovirus*; Species: Suid herpesvirus 1 (Aujeszky's disease virus, pseudorabies virus) (Davison et al 2005).

19.1.2. OIE list

Listed.

19.1.3. New Zealand status

Listed as an exotic, notifiable organism.

19.1.4. Epidemiology

Aujeszky's disease (pseudorabies) is a disease of pigs that was eradicated from New Zealand in 1995. It occurs world-wide, except in Australia, Canada, Finland, Sweden, Denmark and the UK. Several countries are attempting eradication (van Oirschot 2004). The virus can be transmitted to cattle and other animals by close contact with infected pigs. Stallknecht and Howerth's (2001) review of the literature included reports of a natural case in a roe deer and experimental infection to white-tailed deer. In animals other than pigs, the disease is characterised by pruritis and acute neurological signs and is invariably rapidly fatal (Baker et al 1982; Henderson et al 1995; Herweijer and de Jonge 1977; Navetat et al 1994; Sweda et al 1993; van Oirschot 2004).

19.1.5. Hazard identification conclusion

Since Aujeszky's disease virus can infect deer it is classified as a hazard in the commodity.

19.2. RISK ASSESSMENT

19.2.1. Entry assessment

Aujeszky's disease is rare in deer and could only occur if they were kept in close contact with infected pigs. In animals other than pigs the clinical signs are dramatic (Baker et al 1982; Henderson et al 1995; Herweijer and de Jonge 1977; Navetat et al 1994; Sweda et al 1993; van Oirschot 2004) and the outcome is invariably fatal. It has been shown that the risk of disease transmission via *in vivo* derived swine embryos is negligible provided embryos are trypsin treated and properly handled between collection and transfer according to the IETS Manual.

The likelihood that deer germplasm would be collected from infected donors and that virus would be associated with the commodity is considered negligible and the likelihood of entry is therefore assessed to be negligible.

19.2.2. Risk estimation

Because the entry assessment is negligible, the risk estimate for Aujeszky's disease is negligible and it is not classified as a risk in the commodity. Therefore, risk management measures are not justified.

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20. Vesicular stomatitis virus

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Family: Rhabdoviridae; Genus: *Vesiculovirus*: Species: *Vesicular stomatitis virus, Alagoas virus, Vesicular stomatitis virus Indiana virus, Vesicular stomatitis virus New Jersey virus* (Tordo et al 2005).

20.1.2. OIE list

Listed.

20.1.3. New Zealand status

Listed as an exotic and notifiable disease.

20.1.4. Epidemiology

Vesicular stomatitis (VS) is considered to be a disease of horses, cattle, pigs and, more rarely, of sheep and goats (Schmidt 2004). In addition to being a virus of vertebrates, it has also been shown to multiply in insects such as blackflies (*Simulium* spp.), sandflies (*Lutzomyia* spp.), mosquitoes (*Aedes aegypti*) and leafhoppers (*Peregrinus maidis*) (Mare and Mead 2004).

VS is important mainly because it is clinically indistinguishable from foot and mouth disease (Rodriguez 2002; Schmidt 2002; Sellers and Daggupaty 1990). Therefore, initial diagnosis of the disease, before laboratory confirmation, may trigger the massive initial response usually reserved for foot and mouth disease. Alternatively, if an outbreak of foot and mouth disease is incorrectly assumed to be VS, as occurred in Saskatchewan in 1951, the response to the foot and mouth disease outbreak can be delayed (Sellers and Daggupaty 1990).

The disease is endemic in Central and South America and thousands of outbreaks occur each year from southern Mexico to northern South America (Rodriguez 2002). In the USA, the disease occurs sporadically in some southern states but is endemic in at least one location, Ossabaw Island in Georgia (Stallknecht 2000). In some seasons the disease spreads northward along riverbeds into locations in the northern USA (Schmidtmann et al 1999) and even as far as Canada (Wilks 1994). VS does not occur outside of the Americas.

The most commonly held view is that the virus is transmitted by an insect vector. Virus has been isolated from sandflies (*Lutzomyia shannoni*), which are the most likely vectors (Braverman 1994; Comer et al 1994; Rodriguez et al 1996; Schmidtmann et al 2002; Stallknecht 2000), but *Culicoides* spp. are also possible vectors and have been infected experimentally (Nunamaker et al 2000). According to one source, the virus can be biologically transmitted by blackflies (*Simulium vittatum*) and mechanically by *Culicoides*. The virus can also be transmitted by teat cups during milking of cows with teat lesions or by infection of wounds and abrasions (Wilks 1994).

Although the maintenance hosts of VS have not been conclusively established, deer, raccoons (Stallknecht 2000) and the cotton rat *Sigmodon hispidus* (Jimenez et al 1996) have been found with antibodies against the virus. Many other species of animals can be infected or develop antibodies against the virus (Blood et al 1989; Hanson and McMillan 1990).

VS is zoonotic and people are infected by direct contact or as a result of laboratory accidents (Letchworth et al 1999; Wilks 1994).

The incubation period of VS is 1-3 days (Wilks 1994), but for regulatory purposes a period of 21 days is given in the *Code*.

There is some controversy about the pathogenesis of VS. Lesions on teats and feet are primary lesions caused by entry of the virus directly at these sites (Wilks 1994). Similarly, in experimental infection of pigs, lesions occurred at the injection sites but there was no viraemia (Howerth et al 1997). In a description of the pathogenesis, it is stated that virus replicates in the lower layers of the epidermis and there is no mention of viraemia (Mare and Mead 2004). It has been stated that viraemia does not occur in mammalian hosts but transmission of the virus to non-infected blackfly when infected and non-infected blackfly fed on the same host has been demonstrated (Mead et al 2000). In contrast, one secondary source has claimed that there is a primary viraemia with subsequent localisation of virus in mucous membranes of the mouth and the skin around the coronets (Blood et al 1989). However, a later edition of the same text book does not repeat this claim and, indeed, states "viremia [sic] has not been detected in any domestic animal species naturally or experimentally infected with the New Jersey serotype of the virus" (Radostits et al 2007). It appears that viraemia is unlikely in cases of VS.

Serotype specific antibody develops within 5-8 days of infection and can be detected by a blocking or a competitive ELISA or virus neutralization. Both New Jersey and Indiana types are used as antigen (Schmidt 2004).

Antibody has been demonstrated in 43% of white-tailed deer on Ossabow Island and in about 1% of deer in other locations in the USA (Fletcher et al 1991). Antibody has also been demonstrated in elk and mule deer (Webb et al 1987). Virus was not isolated from blood, nasal or throat swabs of infected white-tailed deer, nor from sandflies fed on them (Comer et al 1995). Clinical disease is rare in deer but may occur in white-tailed deer (Hanson and McMillan 1990).

The complete host range of VS is not known but infection or antibody production has been described in pigs, white-tailed deer, raccoon, skunk, bobtail, kinkajou, two- and three- toed sloths, night monkeys, marmosets, agoutis, and rabbits (Hanson and McMillan 1990). In view of the wide host range it is possible that wild and feral animals, including deer could be infected but indigenous birds are unlikely to be susceptible.

There is no evidence that deer are important in the epidemiology of the disease and transmission in germplasm has not been described in any animal species.

20.1.5. Hazard identification conclusion

VS virus is an exotic pathogen of horses and cattle and deer may also be infected. There are no reports of transmission in germplasm. Since there are specific recommendations in the *Code* relating to the disease it is considered that a conservative approach is appropriate. Therefore, VS virus is classified as a hazard in the commodity.

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

20.2.1.1. Semen

There is no evidence that VS virus is transmitted in semen. Large ruminants were listed in one publication as likely to excrete the virus in semen and possibly able to transmit infection (Hare 1985), but no evidence was cited to support this view. Similarly, VS has been listed as a disease "with evidence that risk of transmission (by artificial insemination) is moderate or high", but again, no evidence was given to support this contention. The same authors listed VS as a disease for which isolation of the agent from semen has not been reported (Eaglesome and Garcia 1997). As viraemia probably does not occur in natural infections (Radostits et al 2007), excretion of VS virus in semen is unlikely. The likelihood of entry of VS virus in germplasm is therefore unlikely but is assessed to be non-negligible.

20.2.1.2. Embryos

VS virus adhered to the *zona pellucida* when cattle embryos were exposed *in vitro* and could not be removed by washing (Lauerman et al 1986). However, as viraemia probably does not occur in this disease, contamination of embryos with virus is unlikely. The IETS has classified VS as Category 4 in cattle and swine, that is "a disease on which preliminary work has been conducted or is in progress" (*Code* Article 4.7.14.). Despite the lack of evidence that VS might be associated with the commodity, the *Code* makes recommendations relating to embryos. In conclusion, the likelihood of embryos being contaminated with the virus is assessed to be non-negligible.

20.2.2. Exposure assessment

Imported semen and embryos would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is assessed to be high.

20.2.3. Consequence Assessment

No reports of the use of infected semen or embryos in susceptible recipients are available. Therefore it should be assumed that insemination or transplantation of infected germplasm into susceptible recipients could result in infection. Infected animals would be expected to show signs of VS but would not be contagious and would not infect animals in contact with them. They could, however, infect competent vectors, but the likely vector is not known to occur in New Zealand (*Lutzomyia shannoni*). The establishment of the disease would create difficulties in distinguishing the disease from foot and mouth disease. However, it seems unlikely that a suitable combination of competent vectors and maintenance host exists outside the endemic areas of the Americas as the disease has never established anywhere else. Despite this, the *Code* recommends that trade in embryos should be restricted to disease free countries or zones. Although extremely unlikely, establishment of VS in New Zealand could, therefore, have implications for trade in embryos and live animals.

VS virus can cause disease in people, as a result of direct contact or laboratory accidents. Many cases of the disease probably go undiagnosed as the disease symptoms are similar to influenza. Many people in endemic areas have antibody against the virus. In laboratories

the route of infection is probably by inhalation of aerosols and in the field by transfer by hand to nose and eyes in farmers and livestock handlers (Hanson and McMillan 1990; Wilks 1994). In the unlikely event that VS should establish in New Zealand, sporadic infections in humans could be expected during outbreaks of disease in livestock.

Infections in feral and wild species are likely to be subclinical. Therefore the effects on the environment would be negligible.

In view of the above, the consequences of introduction are assessed to be non-negligible.

20.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for VS is non-negligible, and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

20.3. RISK MANAGEMENT

20.3.1. Options

The following points were considered when drafting options for the management of the risk of introducing VS virus in deer germplasm.

- The *Code* gives recommendations for trade in live animals and embryos.
- The *Code* gives no recommendations regarding trade in cattle semen, thereby implying that it will not be contaminated with VS virus.
- Serological tests are available for VS antibodies. Serological testing could be carried out after, rather than before, germplasm collection (as recommended in the *Code*). This would ensure that animals were not incubating the disease at the time of embryo collection.
- Conditional on a suitable quarantine period with protection from insect vectors, seropositive animals demonstrating a stable or declining titre could be safely utilised as donors. This is because viraemia has not been demonstrated in infected animals and there is no evidence for a carrier state in recovered animals (Pharo 1999).

The *Code* recommendations relating to embryos are:

Article 8.15.9.

Recommendations for importation from countries or zones considered infected with VS

for in vivo derived embryos of ruminants, swine and horses

Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the donor females:
   a. were kept for the 21 days prior to, and during, collection in an *establishment* where no *case* of VS was reported during that period;

b. were subjected to a diagnostic test for VS, with negative results, within the 21 days prior to embryo collection;

2. the embryos were collected, processed and stored in conformity with the provisions of Chapters 4.7. and 4.9., as relevant.

One or a combination of the following measures could be considered in order to effectively manage the risk.

1. Germplasm could be imported without restriction since the likelihood of importing VSV in germplasm is extremely low.
2. Donors could be required to be resident for at least the 21 days prior to germplasm collection and during germplasm collection, in an establishment where no case of VS was reported during that period.
3. Donors could be required to be kept on a quarantine station where no case of VS has occurred during the 21 days before germplasm collection and be subjected to an OIE-recommended serological test with a negative result between 3-6 weeks after germplasm collection.
4. Donors could be subjected to an OIE-recommended serological test diagnostic test and in the case of any positive result, donors could be re-tested not less than 14 days subsequently. The results of testing could indicate that the donor has a negative, stable or declining titre.

N.B. This testing option allows serologically positive animals to be eligible for donation.

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21. *Bacillus anthracis* (Anthrax)

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

Bacillus anthracis.

21.1.2. OIE list

Listed.

21.1.3. New Zealand status

Exotic, notifiable disease last diagnosed in 1954.

21.1.4. Epidemiology

Anthrax can infect a wide range of hosts including deer (Gates et al 2001). However, despite searching three electronic data bases and three detailed reviews on anthrax no reference could be found to the transmission of the disease by germplasm. No recommendations are made in the *Code* in relation to trade in semen or embryos, thereby implying that no measures are warranted. In addition the *Code* states that "there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs". Further, it is inconceivable that germplasm would be collected from animals clinically ill with anthrax.

21.1.5. Hazard identification conclusion

Anthrax is not transmitted by germplasm and therefore *Bacillus anthracis* is not considered to be a hazard in the commodity.

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22. *Brucella* spp.

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Brucella abortus, *B. melitensis* and *B. suis*.

22.1.2. OIE list

Listed.

22.1.3. New Zealand status

Exotic notifiable organisms.

22.1.4. Epidemiology

Brucellosis has been documented in naturally and experimentally infected fallow deer, red deer, sika deer, mule deer, white-tailed deer, moose, roe deer, reindeer and caribou (Thorne 2001). Therefore, it can be assumed that all species are susceptible. *Brucella abortus* is usually a pathogen of cattle but can occasionally infect other species. *Brucella suis* infects pigs but may occasionally infect cattle while *B. melitensis* is primarily a pathogen of sheep and goats.

Brucella suis biovar 4 is common in reindeer and caribou in the Arctic region and has been described in moose (Godfroid 2004; Godfroid et al 2004b). It has been experimentally transmitted to white-tailed deer (Qureshi et al 1999). However, species in which natural infection has been described are not listed in the commodity definition.

Brucella melitensis is rarely described in wildlife (Godfroid 2004) and no reference was found to it infecting deer.

Brucella abortus is the species most commonly infecting deer. However, infections are rare in wildlife where the disease has been eradicated in cattle and generally it is self-limiting. An exception is the infection of elk in the Yellowstone National Park area of the United States where the infection is probably associated with the concentration of deer associated with winter feeding and appears to be self-sustaining in the population (Godfroid 2004). No evidence was found that brucellosis has been described as a problem in farmed deer.

Brucella infections are primarily associated with infections of the reproductive tract and abortions in females and orchitis and accessory sex gland infection in males. Infections of joints are common in pigs. In humans, brucellosis causes fever and a variety of other symptoms including arthritis and, more rarely, orchitis. In all species, the incubation period may be protracted and has a chronic course. In cattle, infections are often subclinical in non-pregnant animals but pregnant animals develop a placentitis causing abortion. Subsequent pregnancies are usually carried to term, but uterine and mammary infections recur, with excretion of the organism in milk and vaginal discharges at subsequent calvings. Bulls may develop orchitis, which causes infertility, and may also develop hygromas of the leg joints.

Diagnosis can be made by isolation of the organism or a variety of serological tests. (Neilsen and Ewatt 2009). The three species covered in this section are smooth *Brucella* with similar surface antigens and, for this reason, serological tests are not species specific. Antigens prepared from *B. abortus* are therefore suitable for the diagnosis of all three species. The same diagnostic methods used in cattle can be used for deer.

22.1.5. Hazard identification conclusions

It is unlikely that infected deer would be used as germplasm donors. However, in this risk analysis a conservative stance is adopted and *Brucella* spp. are classified as hazards in the commodity.

22.2. RISK ASSESSMENT

22.2.1. Entry assessment

22.2.1.1. Semen

Orchitis, epididymitis and infection of the accessory glands occur in bulls (Godfroid et al 2004a). Semen from infected bulls may be contaminated with *B. abortus* (Plant et al 1976; Riddel et al 1989). Antibiotics used in semen diluting fluids may not effectively penetrate cells in which the intracellular organisms are located. Therefore, the likelihood that deer semen could contain *B. abortus* is assessed to be non-negligible.

22.2.1.2. Embryos

There is a considerable body of evidence to show that *B. abortus* is not carried by properly prepared and washed bovine embryos (Stringfellow et al 1982; Stringfellow and Wright 1989; Voelkel et al 1983). The organism does not attach to intact *zona pellucida* or is efficiently removed by washing (Stringfellow et al 1984). However, it is recommended that wash media should contain antibiotics (Riddel et al 1989; Riddell et al 1989). *Brucella abortus* has been shown to be sensitive to the antibiotics used in preparation of embryos (Stringfellow et al 1986). The IETS classifies *B. abortus* as a Category 1 disease, that is one "for which sufficient evidence has accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer" (*Code* Article 4.7.14.). Therefore, provided bovine embryos are properly processed and treated with appropriate antibiotics, the likelihood that they would contain *B. abortus* is assessed to be negligible.

There is no evidence relating to deer embryos. However, in view of the findings regarding bovine embryos, and because antibiotics will be used in their preparation, the likelihood that deer embryos would be contaminated with viable *Brucella* spp. is assessed to be negligible.

22.2.2. Exposure assessment

Since semen will be inseminated into susceptible recipients, the likelihood of exposure is high.

22.2.3. Consequence assessment

Contaminated semen inseminated into susceptible females is likely to cause infection (Manthei et al 1950). Since the disease is self-sustaining in deer in the Yellowstone

National Park area deer, are clearly capable of spreading brucellosis and could transmit it to cattle, with subsequent economic losses.

Brucellosis is a zoonosis and re-establishment of the disease in New Zealand could lead to some cases of brucellosis in people. Therefore, the consequences for human health are considered to be non-negligible.

Feral deer could become infected through contact with farmed deer. However, this is unlikely since there were no reports of infection in New Zealand deer when the disease was endemic in cattle. The infection in wildlife has been described as "a marginal problem that poses little risk to the species concerned or to livestock" (Godfroid 2004). The consequences for the New Zealand environment are therefore assessed to be negligible.

In conclusion, the consequences of introducing infected deer semen are considered to be non-negligible since this could result in the establishment of a serious infectious disease in cattle and also could have deleterious consequences for human health.

22.2.4. Risk estimation

Since entry, exposure, and consequence assessments for *B. abortus* in deer semen are non-negligible, the risk estimate for semen is non-negligible and it is classified as a risk in the commodity and risk management measures may be justified.

Since the entry assessment for *B. abortus* in deer embryos is negligible, the risk estimate is negligible. Therefore, risk management measures are not justified for deer embryos.

22.3. RISK MANAGEMENT

22.3.1. Options

The following points were considered when drafting options for the management of *Brucella* spp. in the commodity:

- Deer are most commonly infected with *B. abortus* but deer species likely to be imported are probably also susceptible to *B. melitensis* and *B. suis*.
- *Brucella abortus* was eradicated from New Zealand at great expense and a conservative approach should be taken with regard to possible re-introduction of the organism.
- The same diagnostic tests used in cattle can be applied to deer but tests used should be validated for deer.
- Treatments and vaccination are not suitable measures to be used for management of the organism in imported deer semen.

There are no recommendations in the *Code* relating to deer semen but the Article for bovine semen is reproduced below:

Article 11.3.7.

Recommendations for the importation of bovine semen

Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that:

1. when the semen is from an *artificial insemination centre*, the testing programme includes the buffered *Brucella* antigen and complement fixation tests;
2. when the semen is not from an *artificial insemination centre*, the donor animals:

a. were kept in a country or zone free from bovine brucellosis; or
b. were kept in a *herd* officially free from bovine brucellosis, showed no clinical sign of bovine brucellosis on the day of collection of the semen and were subjected to a buffered *Brucella* antigen test with negative results during the 30 days prior to collection; or
c. were kept in a *herd* free from bovine brucellosis, showed no clinical sign of bovine brucellosis on the day of collection and were subjected to the buffered *Brucella* antigen and complement fixation tests with negative results during the 30 days prior to collection; or
3. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.

One or a combination of the following options could be considered in order to manage the risk.

1. Deer semen could be imported only from donors that have been resident for their entire lives in a country or zone that is free from *B. abortus*, *B. melitensis* and *B. suis*.
2. Semen for export is from an artificial insemination centre, and the testing programme includes the buffered *Brucella* antigen and complement fixation tests.
3. Donor stags could be required to be kept in a herd in which no case of brucellosis has been known to occur, show no clinical sign of bovine brucellosis on the day of collection and were subjected to buffered *Brucella* antigen and complement fixation tests with negative results at least 30 days after collection. Note that testing carried out after, rather than before, semen collection would ensure that animals were not incubating the disease at the time of collection.
4. An aliquot of semen from each collection batch could be cultured for *Brucella* organisms with negative results.
5. Semen could be imported without restriction since it will be prepared according to the methods recommended in the *Code* (as specified in the commodity definition) and will contain antibiotics that are effective against *Brucella* spp.

   N.B. This option does not provide assurance that the antibiotics will be effective against intracellular *Brucella* organisms in semen.

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23. *Chlamydophila abortus*

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Chlamydophila abortus

23.1.2. OIE list

Ovine chlamydiosis is listed.

23.1.3. New Zealand status

Chlamydophila abortus is listed as an unwanted notifiable organism.

23.1.4. Epidemiology

Enzootic abortion caused by *C. abortus* is primarily a disease of sheep and goats (Aitken 1983) but the pathogen also infects cattle, causing epizootic bovine abortion. Less commonly it may also infect deer (Longbottom 2008).

Transmission probably occurs by the faecal-oral and venereal routes. Persistent infections are common. Anderson (2004) described persistent infection of male accessory glands and presence of *C. abortus* in semen. Ewes that have aborted remain long-term intestinal carriers (Aitken 1983) and may also be chronically infected in their reproductive tract (Longbottom 2009; Papp et al 1994; Papp et al 1998). Bulls may remain carriers for at least 18 months (Domeika et al 1994). There is little information about deer but it is assumed that they may excrete the organism in semen and remain long-term carriers.

The incubation period of *C. abortus* infections in sheep is variable. Some animals become infected in one season, remain infected and abort in the subsequent season, while in other cases abortion may occur in the same season as infection (Aitken 1983).

The disease is diagnosed by demonstration or isolation of the organism in placental material. Diagnostic techniques include examination of suitably stained smears, antigen detection ELISA, PCR, demonstration of organisms in tissue section by direct staining or immunostaining, or by isolation of the organism in tissue culture or embryonated eggs (Andersen 2004; Dagnall and Wilsmore 1990; Longbottom 2008; Szeredi and Bacsadi 2002; Thomas et al 1990). *Chlamydophila pecorum* (present in New Zealand) antibodies cross-react with *C. abortus* so that serological responses to *C. abortus* and *C. pecorum* need to be differentiated by sequence analysis of the 16S rRNA (Mackereth and Stanislawek 2002). Serological tests include the complement fixation test and ELISA, but specificity is not high and cross-reactions occur between *C. abortus* and some gram negative organisms (Longbottom 2008). However, from a biosecurity perspective when screening imported animals, sensitivity is more important than specificity.

23.1.5. Hazard identification conclusion

Chlamydophila abortus is an exotic, notifiable organism and ruminants may be long-term carriers. The organism commonly infects the reproductive tract. Therefore it is classified as a hazard in the commodity.

23.2. RISK ASSESSMENT

23.2.1. Entry assessment

23.2.1.1. Semen

Bulls and rams may excrete *C. abortus* in their semen and venereal transmission has been demonstrated (Amin 2003; Andersen 2004; Appleyard et al 1985; Domeika et al 1994; Storz et al 1976; Suri et al 1986). Therefore the likelihood that deer semen imported into New Zealand could contain *Chlamydophila* spp. is assessed to be non-negligible.

23.2.1.2. Embryos

It has been shown that embryos collected from ewes that were excreting the organism in their uterine discharges did not infect recipients of the embryos or the progeny derived from them (Williams et al 1998). However, the number of animals involved in the study was small and the finding cannot be considered conclusive. The IETS has classified *C. abortus* as a Category 4 organism for which "preliminary information has been conducted or is in progress" (*Code* Article 4.7.14.). The safety of embryo transfer in sheep remains to be conclusively proven and no information is available for deer. Therefore, the likelihood that deer embryos could be contaminated with *C. abortus* is assessed to be non-negligible.

23.2.2. Exposure assessment

Imported germplasm would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is assessed to be high.

23.2.3. Consequence assessment

Heifers inseminated with semen spiked with what was described as *Chlamydia psittaci*, failed to conceive and became infected with the organism (Bowen et al 1978). However, since the classification of the chlamydial organisms was uncertain at that time it was likely to have been *C. abortus*. In addition, insemination with infected semen resulted in seroconversion and a recovery of the organism from three out of ten ewes (Appleyard et al 1985).

Introduction of the organism would be likely to result in the establishment of infection in deer and the organism could be transmitted to cattle and sheep, thus introducing a significant production limiting disease.

Chlamydophila abortus is zoonotic and may cause abortion in women who have been in contact with infected ewes during the lambing season (Longbottom 2008). Although no descriptions of transmission from deer to women were found it is assumed that women could similarly be infected directly from deer. Therefore, introduction of the disease could have consequences for human health.

Feral goats, deer, and thar could become infected. However, the consequences for the environment are likely to be minor since enzootic abortion is a disease associated with intensive farming unlikely to become a problem in free ranging wildlife.

In conclusion, since the organism could establish in New Zealand and cause economically significant effects on sheep farming and sporadic cases of human disease, the consequences are assessed to be non-negligible.

23.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for *C. abortus* is non-negligible and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

23.3. RISK MANAGEMENT

23.3.1. Options

The following facts were considered when drafting options for the management of the organism in the commodity:

- Semen and embryos are both considered to be risk commodities.
- Long-term carriers of infection may occur and, therefore, quarantine is not a useful method to prevent its entry.
- Serological tests are available for diagnosis in sheep. The complement fixation test is still the most widely used test but lacks specificity (Longbottom 2008).
- The organism can be isolated in tissue culture or demonstrated by PCR, antigen capture ELISA or immunostaining methods (Longbottom 2008).
- The disease is rarely recognised in deer and there are no criteria for herd freedom and probably no herds could be certified as officially free from the disease.
- The most widely used antibiotics for treatment and prophylaxis are tetracyclines (Andersen 2004).

The *Code* section relating to *C. abortus* provides guidelines for safe trade of sheep semen but not for trade in embryos or for trade in deer germplasm. Therefore, similar precautions need to be taken for both semen and embryo donors. The sections for sheep semen from the *Code* are reproduced below:

Article 14.5.4.

Recommendations for the importation of semen of sheep
Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:
1. the donor animals:
a. have been kept in *establishments* or *artificial insemination centres* free from EAE during the past 2 years, and have not been in contact with animals of a lower health status;
b. were subjected to a diagnostic test for EAE with negative results 2 to 3 weeks after collection of the semen;
2. an aliquot of the semen to be exported was shown to be free of Chlamydia psittaci, by culture techniques.

One or a combination of the following measures could be considered in order to effectively manage the risk.

1. Donors are not to be from any premises or have been in contact with any species of animal from a premise where *C. abortus* has been diagnosed.
2. Individual donors could be tested serologically using an OIE-recommended test for *C. abortus*, 2-3 weeks after germplasm collection.
3. Aliquots of semen and embryos could be tested for *C. abortus* by PCR or antigen detection ELISA, with negative results. In the case of embryos, wash fluid and embryos that are not suitable for export, could be used for testing.
4. Tetracycline or macrolide antibiotics could be added to imported germplasm.

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24. *Leptospira* serovars

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

There are over 200 *Leptospira* serovars classified into 23 serogroups (Bolin 2009). A newer alternative scheme based on genomic characteristics classifies the pathogenic organisms into several species. However, for the purposes of this risk analysis, serovars are written as if they were single species e.g. *Leptospira hardjo, L. pomona* etc.

24.1.2. OIE list

Leptospirosis is a listed disease of multiple species but the *Code* does not have a chapter on the disease. In 2007 the Terrestrial Animal Health Standards Commission stated that "development of a chapter at this time is not a priority because the disease is virtually ubiquitous and international trade is not considered to increase risks to human or animal health. Rather than leave the title and no chapter in the *Code*, the commission has decided to delete the title" (OIE 2007). At the OIE General Session in May 2009, the International Committee accepted the recommendation of the Terrestrial Animal Health Standards Commission that the empty *Code* chapter on leptospirosis should be deleted.

24.1.3. New Zealand status

Leptospira hardjo, L. pomona, L. balcanica, L. copehageni, L. ballum, and *L. tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). A single isolation of *L. australis* has been reported from a human (Thompson 1980). Serological examinations have shown that five of the species endemic in farm animals infect humans but *L. balcanica*, which is associated with possums, has not been diagnosed in people (ESR 2007). Other *Leptospira* spp. are classified by MAF as "other exotic organisms".

24.1.4. Epidemiology

Leptospirosis is a complex of diseases caused by at least 200 different serovars of *Leptospira*. Many *Leptospira* are adapted to a particular host species (Farina et al 1996). Species other than the maintenance host may be more resistant to infection but, if they become infected, are more susceptible to disease. *Leptospira hardjo*, for example, infects most cattle in an endemic situation but causes only occasional cases of disease in that species. However, it may cause sporadic disease in other species, such as humans. In maintenance hosts, *Leptospira* may localise in the kidneys and the animal may continue to excrete the organism in urine for years. Cattle may remain carriers of *L. hardjo* for at least 450 days (Hunter 2004). In New Zealand, the prevalence of the disease in humans is relatively high for a country with a temperate climate and *L. hardjo* accounts for nearly half the cases (Thornley et al 2002).

Leptospira spread in water and mud contaminated with infected urine. Infection can occur by mouth or through the skin, particularly through abrasions and wounds. Animals showing clinical signs shed more organisms and are more important sources of infection than chronic carriers (Horsch 1989).

In accidental hosts, the incubation period may be from 2-16 days and is followed by a period of bacteraemia. A variety of signs may be shown by diseased animals including abortion, haemolytic anaemia, icterus and nephritis. Leptospirosis can be diagnosed by the isolation of the organism, but because this is difficult it is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low titre indicating resolution or chronic infection. The microscopic agglutination test is still the most commonly used, but a number of variations of ELISA are also available. ELISAs generally lack serovar specificity (Bolin 2009). Leptospirosis is seldom the cause of economically serious disease in animals and is mainly of concern because it is zoonotic, occasionally causing serious disease in humans (Thornley et al 2002).

Deer are commonly serologically positive and are susceptible to experimental infection, but naturally occurring disease is not known to occur (Leighton and Kiuiken 2001). In New Zealand serological evidence of infection in farmed deer is common in apparently healthy animals (Reichel et al 1999; Wilson et al 1998). There is, however, a report of infection associated with haemolytic disease in red deer calves (Fairley et al 1986).

Leptospira spp. are sensitive to several antibiotics, particularly streptomycin and penicillin.

24.1.5. Hazard identification conclusion

Leptospira spp. other than the six endemic serovars, are exotic, zoonotic organisms and are classified as hazards in the commodity.

24.2. RISK ASSESSMENT

24.2.1. Entry assessment

24.2.1.1. Semen

Leptospira spp. are commonly excreted in the semen of bulls (Heinemann et al 2000; Heinemann et al 1999; Kiktenko et al 1976; Masri et al 1997) and it is highly likely that they are excreted in deer semen also. *Leptospira* spp. are sensitive to the antibiotics normally used in the preparation of diluted semen, and properly prepared semen is unlikely to infect recipients. For this reason, for the purposes of international trade, treatment of animals or animal germplasm with suitable antibiotics provides an effective means of controlling the spread of exotic serovars. The *Code* recommendation for germplasm is that it should include suitable bactericidal antibiotics (Articles 4.5.7. and 4.7.6.). For many years, New Zealand has required the antibiotic treatment of imported semen or embryos to successfully exclude serovars of *Leptospira*. The likelihood of entry is dependent upon the efficacy of the antibiotics used in semen preparations or the donor animal, rather than the absence of the organism in the semen. The likelihood of entry is assessed to be low.

24.2.1.2. Embryos

No information was found relating to *Leptospira* in deer embryos and so extrapolation from what is known about cattle embryos is necessary. *Leptospira* were found in the genital tract of heifers experimentally infected with *L. hardjo*, but could not be cultivated from *in vitro* fertilised embryos (Bielanski et al 1998). *Leptospira hardjo* were found to adhere to, and penetrate into the pores of, the *zona pellucida* of embryos exposed *in vitro* (Bielanski and Surujballi 1996). However, when cultured in antibiotic-containing medium, *L. hardjo* could not be isolated from the embryos although they could be isolated from

controls cultured in medium without antibiotics. When embryos were transplanted into recipient heifers, *L. hardjo* was not transmitted to the recipients or their progeny (Bielanski and Surujballi 1996). The risk of entry is dependent upon the efficacy of the antibiotics used in embryo preparation rather than the freedom of the embryos from leptospires. The likelihood of entry is therefore assessed to be low.

24.2.2. Exposure assessment

Imported germplasm would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is assessed to be high.

24.2.3. Consequence assessment

According to Horsch "the genital excretions of animals can function as primary infection sources" for leptospirosis (Horsch 1989). Therefore, insemination or transplantation of infected, imported germplasm that has not been treated with antibiotics would be likely to lead to infection of the recipients. Infection of a recipient would be dependent on the particular *Leptospira* serovar being one to which deer are susceptible. If an infected recipient were able transmit the organism to suitable maintenance hosts during the period it was excreting the organisms in urine, the organism could become established.

The establishment of a new *Leptospira* serovar to which humans are susceptible could lead to sporadic occurrence of leptospirosis in humans. The number and seriousness of the cases would depend on the serovars involved and the possibility of contact with infected animals. Some serovars are not important as human pathogens. For example, in New Zealand *L. balcanica* is common in its maintenance host the brush tailed possum, but infections of humans have not occurred despite the close contact between possums and possum hunters.

There are not likely to be noticeable consequences for wild animals but some species, such as *L. gippotyphosa, L. canicola, L. sejroe,* and *L. saxkoebing,* could become established in mice and rats (Horsch 1989).

The establishment of new *Leptospira* serovars could cause sporadic cases of disease in humans. Therefore, the consequences of establishment are assessed to be non-negligible.

24.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for exotic *Leptospira* serovars is non-negligible and they are classified as a risk in the commodity. Therefore, risk management measures may be justified.

24.3. RISK MANAGEMENT

24.3.1. Options

The following points were considered when drafting options for the management of *Leptospira* in the commodity:

- Quarantine would not be a suitable measure because subclinical long-term carriers of infection occur.
- The disease is not considered by the OIE as being a disease that has important implications for international trade.

- *Leptospira* are sensitive to several antibiotics and germplasm can be sanitized by using suitable antibiotics in its preparation.

The following options could be considered in order to effectively manage the risk.

1. Donors could be tested serologically to demonstrate freedom from exotic *Leptospira* serovars although this is complex to perform and the results are difficult to interpret because of the many serovars and the difficulty in interpretation of the meaning of cross-reactions and low titre reactions.
2. Aliquots of semen or embryos could be tested by culture or PCR although this is problematic because isolation of organisms is difficult and selection of primers for PCR that will recognise all serovars has not yet been achieved.
3. Germplasm could be prepared according to the *Code* recommendations including the use of suitable antibiotics in semen diluents and embryo washing media.
4. Donor animals could be treated for leptospirosis using an injection of dihydrostreptomycin or another antibiotic. However, no antibiotics have been documented as effective at eliminating the carrier state in deer.

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25. *Mycobacterium bovis* (Bovine tuberculosis)

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Mycobacterium bovis.

25.1.2. OIE list

Listed.

25.1.3. New Zealand status

Endemic and the subject of an eradication campaign in the form of a Pest Management Strategy under the Biosecurity Act of 1993.

25.1.4. Epidemiology

Bovine tuberculosis is primarily a disease of cattle but it affects many other species of animals, including humans. In New Zealand, it occurs in cattle and deer, with rare cases in sheep and goats. It also occurs in possums, feral pigs and goats, and ferrets. It has been reported in several species of deer in Europe and white-tailed deer in the USA (Wilson et al 2008).

The lesions of the primary complex of infection are localised to the organ of entry and/or the associated lymph node. In many cases the infection remains localised to the primary complex. Sometimes it spreads to infect other organs or becomes generalised or occasionally causes miliary tuberculosis (Cousins et al 2004). The clinical signs and pathology vary according to which organs are infected but lesions are essentially granulomas with abscessation and sometimes calcification.

Transmission is by contact with other infected animals and is usually by the respiratory route but can also be by ingestion. Infection of the uterus and female genital tract is rare but endometritis, salpingitis and oophoritis have been described (Biolatti et al 1989; Cousins et al 2004; Muscarella et al 1974) and a typical lesion has been described in the prepuce of a bull (Thoen et al 1977). Tuberculosis involving the testes and epididymis is rare and cases attributed to *M. tuberculosis* (Adeniran et al 1992) are probably incorrect diagnosed cases.

Bovine tuberculosis has been eradicated from many developed countries or is the subject of eradication campaigns. The eradication campaign in New Zealand has failed to eradicate the disease from cattle and deer since the disease is established in possums which continually re-infect cattle and deer.

The immune response to infection is mainly a cellular response and serological tests are insensitive and of little value. The most commonly used test for the diagnosis of tuberculosis in cattle and deer is still the intradermal tuberculin test (Cousins et al 2004). A more recently developed test that has been adopted for use in international trade is the gamma-interferon test (Cousins 2008). A lymphocyte transformation test has been used particularly in deer, and a combination of laboratory tests which measure lymphocyte

transformation, antibody and inflammation has been recommended (Griffin and Buchan 1994) but not been validated for international trade.

Mycobacterium bovis can be cultured by standard methods or bacterial DNA can be identified by PCR analysis (Cousins 2008).

25.1.5. Hazard identification conclusion

Mycobacterium bovis is an endemic organism that is the subject of a Pest Management Strategy administered by the Animal Health Board. It causes disease in a number of animal species including deer and may affect humans. Infections of both male and female genital tracts have been described. Therefore, *M. bovis* is considered to be a hazard in the commodity.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

25.2.1.1. Semen

Mycobacterium bovis is known to be excreted in bull semen (Hare 1985; Niyaz Ahmed et al 1999). It has also been isolated from a typical granulomatous lesion in the prepuce of a bull (Thoen et al 1977). The prevalence of animals excreting *M. bovis* in their semen is assumed to be low as reports in the literature are rare. No reports of excretion in the semen of deer were found but it is assumed that this could occur. The likelihood of the entry of the organism in semen is assessed to be low.

25.2.1.2. Embryos

The infection of embryos with *M. bovis* has not been described. However, the uterus and genital tract of cattle can be infected by *M. bovis* (Biolatti et al 1989; Cousins 2008; Muscarella et al 1974). The IETS has categorised *M. bovis* as a Category 4 pathogen whereby no conclusions are yet possible with regard to the likelihood of transmission by *in vivo*-derived embryos or the risk might not be negligible even if handled in accordance with IETS recommendations.

Mycobacterium paratuberculosis is known to adhere strongly to the *zona pellucida* of embryos and to be resistant to removal by washing (Rhode et al 1990). Therefore, infection of the genital tract could occur in deer and the organisms could adhere to the *zona pellucida*. However, infections of the genital tract are rare in cattle and no records were found of such cases in deer. The likelihood of entry of the organism in embryos is therefore assessed to be low.

25.2.2. Exposure assessment

Since semen and embryos would be inseminated or transferred into susceptible New Zealand recipients the likelihood of exposure is assessed to be high.

25.2.3. Consequence assessment

Insemination of cattle with infected semen led to the infection of recipients (Roumy 1966). It is assumed that implantation of infected embryos could also lead to infection of deer. Infected deer could develop the disease and become infectious to in-contact cattle, deer,

possums, and other susceptible animals. Establishment of infection in animal populations that were previously free from infection would cause additional expenses in the eradication campaign. Individual farms that became infected would be subject to movement restrictions and would suffer losses as a result of condemnation of individual animals and restricted ability to sell animals.

M. bovis is a zoonotic organism and any increase in the prevalence of the disease in livestock increases the risk to humans. However, the disease is already endemic in cattle, possums, and deer and *M. bovis* infections in humans are rare and the increase in the number of cases in humans caused by introducing infected germplasm is likely to be small and the overall effect negligible.

Introduction of the organism could lead to infections in feral animals such as possums, pigs, ferrets, deer, and other animals (Coleman and Cooke 2001). New Zealand native birds and animals would not be susceptible.

Since the introduction of infected germplasm could lead to new outbreaks of bovine tuberculosis the consequences are assessed to be non-negligible.

25.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for *M. bovis* in germplasm is non-negligible and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

25.3. RISK MANAGEMENT

25.3.1. Options

The following relevant points were considered when drafting options for the effective management of the hazard in the commodity:

- Infection of the genital tract of both male and female deer is rare.
- Identification of deer herds that are free from bovine tuberculosis in exporting countries may be difficult.
- Vaccines and treatments are not available.
- Since the disease is chronic, quarantine would not be a useful measure.
- Diagnostic tests available for consideration include tuberculin testing (including the avian/bovine comparative test- the OIE-prescribed test for international trade), the gamma-interferon test (listed in the *Manual* as an alternative test) and the lymphocyte transformation test.
- The organism can be isolated in culture but this is a lengthy procedure. PCR tests are also available.
- The *Code* makes recommendations for the safe importation of farmed deer semen and embryos.

The *Code* recommendations for farmed deer germplasm are given below:

Article 11.8.7.

Recommendations for the importation of semen of farmed cervidae

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the donor animals showed no signs of bovine tuberculosis in any species, on the day of collection of the semen; and either:
   a. were kept in a herd free from bovine tuberculosis in a country, zone or compartment free from bovine tuberculosis of farmed cervidae, and which only accepts animals from free herds in a free country, zone or compartment; or
   b. showed negative results to tuberculin tests carried out annually and were kept in a herd free from bovine tuberculosis;
2. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.

Article 11.8.8.

Recommendations for the importation of embryos/ova of farmed cervidae

Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:

1. the donor females and all other susceptible animals in the herd of origin showed no signs of bovine tuberculosis during the 24 hours prior to embryo collection; and either
   a. originated from a herd free from bovine tuberculosis of farmed cervidae in a country, zone or compartment free from bovine tuberculosis; or
   b. were kept in a herd free from bovine tuberculosis of farmed cervidae and were subjected to a tuberculin test for bovine tuberculosis with negative results during an isolation period of 30 days in the establishment of origin prior to collection;
2. the embryos/ova were collected, processed and stored in conformity with the provisions of Chapters 4.7., 4.8. and 4.9., as relevant.

One or a combination of the following measures could be considered in order to effectively manage the risk.

1. Germplasm donors could be required to show no clinical sign of bovine tuberculosis on the day of collection of the semen.
2. Donors could be required to originate from a bovine tuberculosis free herd in a country, zone or compartment free from bovine tuberculosis.
3. Donors could be kept in a herd free from bovine tuberculosis and be tested at least annually with a tuberculin test with negative results shown.
4. Donors could be required to be kept in a herd free from bovine tuberculosis and subjected to a tuberculin test with negative results during an isolation period of 30 days in the establishment of origin prior to collection.
5. Germplasm donors could be subjected to a tuberculin test and additional diagnostic tests (e.g. gamma-interferon test or lymphocyte transformation test) with negative results 6 weeks after collection of the semen.

6. Donors could show negative results to tuberculin tests carried out annually and be kept in a herd free from bovine tuberculosis.

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26. *Mycoplasma mycoides* subsp. *mycoides* SC

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

Mycoplasma mycoides subsp. *mycoides* SC (*Mmm* SC) is a bacteria that causes the disease contagious bovine pleuropneumonia (CBPP).

N.B. SC= small colonies since when cultured, colonies are 1 mm in diameter, with a classical 'fried-egg' appearance (Thiaucourt 2008).

26.1.2. OIE list

Listed as a disease of bovidae.

26.1.3. New Zealand status

CBPP was introduced with Australian cattle in 1863 and although disease established, it disappeared by 1873 (Fisher 2006). *Mmm* SC is listed as an exotic notifiable organism.

26.1.4. Epidemiology

Contagious bovine pleuropneumonia is a disease of cattle that causes significant economic losses. Occasionally, water buffalo are affected (Brown 2008; Thiaucourt et al 2004). Asian buffalo and goats may also be infected but their role as a reservoir of infection is considered to be negligible (Thiaucourt et al 2004).

Mmm SC has been transmitted experimentally to white-tailed deer (Yedloutschnig 1976) but natural cases have not been described in deer. This may be because all infected countries have no deer populations. Deer are not known to be involved in maintenance or transmission of the disease. However, deer are cloven hoofed ungulates related to cattle and susceptible to many cattle diseases.

The disease has been reported from African countries between 2000-2010, with the exception of 2002, where disease was reported outside Africa in Yemen and Afghanistan. The last case in Europe was reported from Portugal in 1999. Currently, the disease is confined to Africa (WAHID 2010).

In cattle, the incubation period of the disease is between 3 weeks and 4 months (Brown 1998; Thiaucourt et al 2004; OIE 2009) and for the purposes of the *Code*, is 6 months. Disease spreads through direct contact of an infected animal with a naive one by droplet infection. Distances of up to 200 metres under favourable conditions are reported (OIE 2009). Thiaucourt et al (2004) reports aerosol spread over distances of 20 metres or more. CBPP is a debilitating respiratory disease and typical lesions of pleuropneumonia are seen at post-mortem. Many animals are resistant to infection and, in an infected herd, as few as 8% may develop clinical signs (Thiaucourt et al 2004). In another report, the morbidity is said to be variable and the mortality 10-70% (Brown 1998). Infected young calves may develop arthritis without respiratory disease possibly due to colostrally derived immunity (Brown 1998; Thiaucourt et al 2004).

Recovered animals may have sequestered lesions in their lungs for at least a year. These so-called 'lungers' are potential carriers of infection (Brown 1998; Thiaucourt et al 2004).

The disease can be diagnosed by the demonstration of typical macroscopic and microscopic lesions at post-mortem, by culture and identification of the organism, demonstration of the organism by PCR, or by serological tests. The complement fixation test and ELISA are prescribed tests for international trade. A high specificity and sensitivity is claimed for serological tests and PCR. However, it is noteworthy that the validity of the serological tests is based at the herd level and not individual animals. Tests on single animals can be misleading. For example, the complement fixation test can detect nearly all sick animals with acute lesions, but a rather smaller proportion of animals in the early stages of infection or animals in the chronic stage of the disease when very few animals are seropositive (Thiaucourt 2008).

European and African strains of the virus are recognised. Sporadic cases of CBPP have emerged in Europe almost 15 years after the last endemic case occurred in 1967. The new cases were clearly of the European type indicating that the organism may persist in the absence of cases of CBPP (Cheng et al 1995). In this respect it is noteworthy that the organism has been isolated from semen of clinically healthy bulls and bulls with seminal vesiculitis (Goncalves 1994; Stradaioli et al 1999). Isolation of the organism from semen suggests venereal transmission may be possible, but this route of infection requires further investigation (OIE 2009).

26.1.5. Hazard identification conclusion

Mycoplasma mycoides mycoides SC is an exotic notifiable organism that causes a severe disease in cattle. In general, there are currently no deer populations where the disease is endemic. However, these regions could develop deer industries in the future.

Deer have been experimentally infected but it is not known if deer are susceptible to natural infection. It is assumed that they are since deer are cloven hoofed ungulates related to cattle and susceptible to many cattle diseases.

Therefore, *Mmm* SC is classified as a hazard in the commodity.

26.2. RISK ASSESSMENT

26.2.1. Entry assessment

26.2.1.1. Semen

There are no reports of venereal transmission of CBPP. However, *Mycoplasma mycoides mycoides* SC has been isolated from semen and sheath washings from a clinically normal bull (Goncalves 1994) and from semen from bulls that were suffering from seminal vesiculitis (Stradaioli et al 1999). In these cases the bulls showed no signs of CBPP and were seronegative. The clinical significance of these findings is unknown and it is also not known whether the strains isolated were virulent strains able to cause CBPP.

For the purposes of international trade, isolation and identification of the organism from semen constitutes the occurrence of *Mmm* SC infection. The *Code* makes recommendations for the safe trade in bovine semen from free and infected countries or zones.

The likelihood that semen could be contaminated with *Mmm* SC is assessed to be non-negligible.

26.2.1.2. Embryos

For the purposes of international trade, isolation and identification of the organism from embryos constitutes the occurrence of *Mmm* SC infection. The *Code* makes recommendations for the safe trade of bovine embryos. Therefore, embryos may be contaminated and the organism can be isolated from embryos.

The IETS has categorised *Mycoplasma* spp. of cattle into category 4. In regards risk of dissemination of the bacteria by properly processed and handled *in vivo* embryos, category 4 means that no conclusions are yet possible to make; or the risk via embryo transfer might not be negligible.

Therefore, the likelihood of entry of *Mmm* SC with embryos is assessed to be non-negligible.

26.2.2. Exposure assessment

The insemination or transfer of germplasm infected with *Mmm* SC into susceptible recipients has not been described. However, it is known that *Mycoplasma mycoides mycoides* LC and other *Mycoplasma* spp. have frequently been isolated from stillborn and aborted calves and from the genital tract of cows (Kapoor et al 1993; Stradaioli et al 1999). Furthermore, *Mycoplasma mycoides mycoides* LC adheres to and infiltrates the *zona pellucida* (Sylla et al 2005). The organism has been listed as one that could be transmitted by semen (Hare 1985).

Mycoplasma mycoides mycoides SC may be present in bovine semen and embryos and may be transmitted by insemination of infected germplasm. The *Code* recommends measures when trading bovine semen and embryos.

Deer are presumed susceptible to infection and that routes of transmission would be similar to that of cattle. The likelihood of exposure is certain since deer germplasm would be inseminated or transferred into susceptible recipients.

The disease has previously been introduced into New Zealand with Australian cattle imports in 1863. The disease successfully established at that time but was eradicated through slaughtering sick animals, movement controls and ‘tail inoculation’. The disease prevalence was reduced to a point that led to the eventual disappearance of the disease 10 years later (Fisher 2006).

It is assumed that overt disease of the recipient would occur from receiving imported contaminated germplasm. The disease would thus spread amongst in-contact deer and to

cattle. Since the disease has a long and variable incubation period combined with the existence of a carrier status, infection would become wide-spread from animal movements.

Therefore, exposure is assessed to be non-negligible.

26.2.3. Consequence assessment

Infection of recipients could result in cases of CBPP developing after an incubation period that could be prolonged for several months (OIE 2009; Thiaucourt et al 2004). The disease could spread by contact between deer and to cattle leading to the establishment of an economically important disease in New Zealand. Deer and cattle industries and trade in animals and germplasm would be affected. It would also be likely to result in an expensive campaign to eradicate the disease.

The organism is not transmissible to humans so there would be no consequences for human health.

Since the introduction of infected germplasm could result in the establishment of an economically significant disease, the consequences are assessed to be non-negligible.

26.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and *Mmm* SC is classified as a risk in the commodity. Therefore, risk management measures may be justified.

26.3. RISK MANAGEMENT

26.3.1. Options

The following points were considered when drafting options for the effective management of *Mmm* SC in the commodity:

- CBPP is one of the diseases for which the OIE has official recognition status. The *Code* Chapter specifies the steps a country must follow in order to be officially recognised by the OIE as free of CBPP. However, the OIE list of CBPP officially free countries is limited to only 6 Members as at 03/06/10 when the list was last up-dated.

- The *Code* gives recommendations for the importation of bovine embryos and semen from infected and non-infected countries. These recommendations could be used as a basis for formulating the risk management measures that should be used for the importation of deer germplasm.

- It could be specified that donors of germplasm be kept from birth or for the 6 months before germplasm donation in an establishment that is not in a CBPP infected zone and where no case of CBPP was reported for at least 6 months.

- It could also be specified that the donors are not vaccinated and be subjected to a serological test for CBPP on two occasions with a 21-30 day interval, the last test being within 14 days of germplasm collection. The donors could be kept isolated from other animals from the day of the first serological test, until germplasm collection was complete.

- Although internationally prescribed tests for trade are recommended, it should be kept in consideration that these tests have been validated on herds, and not the individual animal. The recommended tests are very good at detecting sick animals. Tests on single animals can be misleading and it is noteworthy that early and chronically infected animals may give false negative results (Thiaucourt 2008).

One or a combination of the following measures could be considered in order to effectively manage the risk:

1. Donors of germplasm should be certified as showing no clinical sign of CBPP on the day of collection.

2. Donors of germplasm could be required to originate from a country or zone that is free from CBPP and have been kept in that free country since birth or for at least the past 6 months.

3. Donors could be required to not be vaccinated against CBPP and be kept since birth or for at least 6 months in an establishment where no case of CBPP has been reported and the establishment is not situated in a CBPP infected zone.

4. Donors could be subjected to an OIE recommended serological test with negative results on two occasions 21-30 days apart with the last test done within 14 days prior to germplasm collection.

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27. *Mollicutes*

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agents

Class: Mollicutes; Order: Mycoplasmatales: Family: Mycoplasmataceae;

Genus: *Mycoplasma*

Genus: *Ureaplasma*

Genus: *Acholeplasma*

27.1.2. OIE list

Not listed.

27.1.3. New Zealand status

The following Mollicutes have been identified in New Zealand and will not be considered further:

Mycoplasma mycoides subsp. *mycoides* LC (Jackson and King 2002), *M. alkalescens* (Brookbanks et al 1969), *M. arginini* (Belton 1990; Belton 1996), *M. dispar* (Hodges et al 1983), *Acholeplasma laidlawi* (Belton 1990; Belton 1996) and *Ureaplasma* spp. (Hodges and Holland 1980; Thornton and Wake 1997).

Mycoplasma hyorhinis and *M. hyopneumoniae* have been isolated from pigs (MacPherson and Hodges 1985).

Mycoplasma mycoides subsp. *mycoides* SC is listed on the Unwanted Organisms Register as an unwanted notifiable organism.

The following Mollicutes have not been identified in New Zealand and may be exotic:

Mycoplasma bovigenitalium, M. bovis, *Mycoplasma verecundum*, *M californicum*, *M. canadense*, *Mycoplasma* group 7, *Acholeplasma axanthum*, *A modicum* and *Ureaplasma diversum*.

There are probably other unidentified species that occur both in New Zealand and overseas.

27.1.4. Epidemiology

There are at least 124 species in the *Mycoplasma* genus, eight in the *Ureaplasma* genus and 18 in the *Acholeplasma* genus (Anonymous 2004). These organisms are widely distributed in nature and often occur as saprophytes or commensals associated with specific species of animals. In several cases they have been associated with various disease syndromes but, in many cases, the role they play as pathogens is uncertain since they have also been isolated from healthy animals. In diseased animals, they sometimes occur as mixed infections and in only a few cases can they be considered to be pathogens for which Koch's postulates can be fulfilled. Examples of such are *M. mycoides mycoides* SC in cattle and *M. capricolum capripneumoniae* in goats. Many species are best thought of as opportunistic pathogens. Furthermore, they are sometimes difficult to culture and classify

and there have been some confusing changes in taxonomy in recent years. The number of organisms in the group is gradually increasing but it is not clear whether this is because they are truly "new" organisms or because they were wrongly typed in the past. For these reasons, older literature cannot always be considered reliable. Basic information, such as incubation periods and how long animals remain carriers, is often not available. Finally since the amount of work done to diagnose these infections in New Zealand may be inadequate and a statement that "the organism has not been described in New Zealand" does not necessarily imply that it is absent or exotic.

The hazard identification concludes that only the following should be considered to be potential hazards:

Mycoplasma bovigenitalium

Mycoplasma bovis

Mycoplasma verecundum

Mycoplasma californicum

Mycoplasma canadense

Mycoplasma group 7

Ureaplasma diversum

No information was found to suggest that any of these organisms, other than *M. bovis*, causes disease in deer. A single case of pulmonary mycoplasmosis has been documented in white-tailed deer (Dyer et al 2004). This discussion is therefore limited to *M. bovis* and, in view of the paucity of information about *Mycoplasma* in deer, much is extrapolated from what is known about the disease in cattle.

Mycoplasma bovis was first isolated in the USA in 1961 and spread to many countries between 1970 and 2000 (Nicholas et al 2008a). It was the *Mycoplasma* species most commonly isolated in Britain between 1990 and 2000 (Ayling et al 2004). Most isolates were from the lung or upper respiratory tract. It also occurs commonly in Europe (Nicholas et al 2008a). The organism has been described as a major cause of respiratory disease, mastitis and arthritis, and as being responsible for a quarter to a third of the cases of calf pneumonia in Europe (Nicholas and Ayling 2003). Infected animals may remain carriers shedding the organism via the respiratory tract for months or years. It has been associated with mastitis (Gonzalez et al 1992; Kirk et al 1997; Pfutzner and Sachse 1996) and with polyarthritis (Henderson and Ball 1999). It has also been isolated from semen (Eder-Rohm 1996; Ozdemir and Turkarslan 1998) and the female genital tract (Irons et al 2004). The organism is commonly transmitted by artificial insemination (Pfutzner 1990). It has been found in milk samples from cattle in Australia (Ghadersohi 2003; Ghadersohi et al 2005; Ghadersohi et al 1999). However, *M. bovis* has not been found in surveys of milk samples from New Zealand cattle (McDonald et al 2009; Reichel et al 1999). Diagnosis can be made by culture of the organism (Nicholas et al 2008a), PCR methods or detection by an antigen detection ELISA (Nielsen et al 1987).

The range of antibiotics to which various Mollicutes are sensitive varies and no comprehensive information is available. Most testing has been conducted on *M. bovis*. Combinations of penicillin, streptomycin, lincomycin, spectinomycin, gentamicin, and tylosin failed to inactivate *M. bovis* on contaminated bovine embryos (Bielanski et al 2000)

and gentamicin, tylosin, lincomycin and spectinomycin failed to eliminate it from semen (Visser et al 1999). In another study, the sensitivity of *M. bovirhinis*, *M. alkalescens* and *M. bovis* to 12 antibiotics was examined. Tiamulin was most effective and erythromycin had no effect (Hirose et al 2003). In another study, many strains of *M. bovis* were found to be resistant to tylosin, spectinomycin, lincomycin, tetracycline and oxytetracycline (Thomas et al 2003). Twenty one field isolates of *M. hyopneumoniae* were tested and one strain was resistant to tylosin, tilmicosin and lincomycin and five were resistant to flumequine and enrofloxacin (Vicca et al 2004). In a study on 58 isolates of *M. bovis*, enrofloxacin was found to be effective but acquired resistance to tetracycline, spectinomycin, azthromycin and clindamycin was demonstrated (Francoz et al 2005). An extensive review of the minimum inhibitory concentrations of a range of antibiotics for small ruminant mycoplasmas has been compiled (Nicholas et al 2008c). Evidence has been found that *M. bovis* strains in Europe and North America are becoming resistant to several antibiotics and treatment with them is often ineffective (Nicholas et al 2008a).

27.1.5. Hazard identification conclusion

Mycoplasma bovis may infect the genital tracts of male and female cattle and may be transmitted by artificial insemination. It occurs in most countries and has been described in respiratory infections of white-tailed deer. Therefore, it is classified as a hazard in the commodity.

27.2. RISK ASSESSMENT

27.2.1. Entry assessment

27.2.1.1. Semen

Semen may be contaminated with *M. bovis* and it has been shown that infection can be transmitted by artificial insemination (Pfutzner 1990). Therefore, the likelihood that *M. bovis* could enter in deer semen is assessed to be non-negligible.

27.2.1.2. Embryos

A number of studies have demonstrated that *Mycoplasma* species readily attach to the *zona pellucida* and are not removed effectively by washing (Bielanski et al 2000; Bielanski et al 1989; Riddell et al 1989). Furthermore, the antibiotics usually used in semen extenders and in the preparation of embryos may not be effective against *Mycoplasma* or *Ureaplasm*a spp. (Bielanski et al 2000; Bielanski and Jordan 1996; Visser et al 1999). Therefore, the likelihood that embryos will be contaminated with *M. bovis* is assessed to be non-negligible.

27.2.2. Exposure assessment

Since imported germplasm will be inseminated or transplanted into susceptible females the likelihood of exposure is assessed to be high.

27.2.3. Consequence assessment

Mycoplasma bovis is a pathogen of economic importance that causes widespread respiratory disease in calves in Europe. The organism could be transmitted from deer to cattle and have significant effects on the New Zealand cattle industries.

There is no evidence to suggest that the introduction of *M. bovis* would adversely affect the environment. However, feral deer and, possibly, other ruminants could be infected although *M. bovis* has not been reported to cause significant disease in wild ruminants.

Mycoplasma bovis has been isolated from humans in at least two cases but, in one, it is doubtful that it was the cause of disease and the patient concerned did not seroconvert. In the other case, the patient responded to tetracycline treatment (Nicholas et al 2008b). Since *M. bovis* is a rare and unusual isolate from humans and is of doubtful significance, it is not considered to be a pathogen of humans.

Although the introduction of new species of Mollicutes would not have deleterious effects on human health or the environment, the likely consequences of introduction for the cattle industries are assessed to be non-negligible.

27.2.4. Risk estimation

Since the entry, exposure and consequence assessments are non-negligible, the risk estimate for *M. bovis* is non-negligible and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

27.3. RISK MANAGEMENT

27.3.1. Options

The following points were considered when drafting options for the management of *M. bovis* in the commodity:

- The organism may be excreted in semen and could contaminate embryos.
- Long-term carriers of infection have been described and therefore quarantine would not be useful for preventing entry of the organism.
- Diagnosis can be made by culturing the organism, PCR to detect DNA or an antigen detection ELISA.
- Antibiotics are available for treatment but development of resistance to several antibiotics has been described and they may not be effective when used in semen extender or in embryo washing fluids.
- Effective vaccines that could be confidently recommended to prevent introduction of the organism are not available.
- There is no *Code* chapter relating to *M. bovis*.

One or a combination of the following options could be considered in order to effectively manage the risk.

1. The current IHSs allow the importation of bovine semen from Europe, North America and Australia and cervine semen from Great Britain with no safeguards other than reliance on antibiotics in the germplasm. This practice could be continued. However, Mollicutes are susceptible to a limited number of antibiotics only. The inherent resistance of *Mycoplasma* spp. to many antibiotics, the increasing emergence of resistant strains (Loria et al 2003) and the undesirability of replacing traditional antibiotic cocktails with ones that are specific for *Mycoplasma* spp. but may not be as effective against other organisms, suggest that the use of antibiotics in extender and wash solutions is not a completely reliable method for sanitizing germplasm. Since information regarding the Mollicutes is constantly

changing, MAF should remain flexible and revise the requirements for the antibiotics to be used in semen extender and embryo wash solutions as necessary.

2. Culture, PCR or ELISA of germplasm with negative results. Germplasm could be cultured from an aliquot taken before the addition of antibiotics, PCR or ELISA could be done on germplasm containing antibiotics.

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28. Coxiella burnetii

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agent

Coxiella burnetii.

28.1.2. OIE list

Listed.

28.1.3. New Zealand status

Listed as an unwanted notifiable organism.

28.1.4. Epidemiology

Q fever occurs worldwide with the exception of New Zealand (Worthington 2001), Iceland (WAHID 2009) and possibly Norway (Jensenius et al 1997).

Coxiella burnetii probably infects all mammalian species, birds and many arthropods (Marin and Raoult 1999; Marrie 1990). In animals, infections are of minimal economic importance and rarely cause disease, but *C. burnetii* is zoonotic and sometimes causes serious disease in humans. Some cases of infection are associated with abortions, especially in goats (Arricau-Bouvery and Rodolakis 2005). Most human infections are asymptomatic or present as a mild influenza-like disease, but acute or chronic infections sometimes occur and some of these result in serious complications such as myocarditis, endocarditis, hepatitis and renal failure (Marin and Raoult 1999; Woldehiwet 2004). *Coxiella burnetii* causes sporadic abortions in both humans and animals (Hatchette et al 2003; Marin and Raoult 1999; Raoult et al 2002; Woldehiwet 2004).

Transmission frequently occurs through contact with infected uterine discharges and placenta and, probably, by inhalation of dust contaminated by animals and their birth products (Arricau-Bouvery and Rodolakis 2005; Behymer and Riemann 1989; Hawker et al 1998; Marin and Raoult 1999; Marrie 1990; Selvaggi et al 1996; Tissot-Dupont et al 1999) Infected ticks may also play a role in spreading the disease. At least 40 species of tick from 11 genera can be infected (Kelly 2004) and their dried faeces form dust that can contaminate animals' coats. Infected cattle shed the organism intermittently in their milk for many years (Kelly 2004).

Infected animals generally show no clinical signs, thus making the determination of the incubation period and the interval to the development of antibodies difficult to determine. In humans the incubation period is given as 1-3 weeks and the development of detectable antibody titres takes 2-3 weeks after the onset of symptoms (Marin and Raoult 1999). It is assumed that infected deer will develop antibody within a similar time interval after infection.

The infection is diagnosed by serological tests, especially ELISA, or by PCR or isolation of the organism by traditional methods (Arricau-Bouvery and Rodolakis 2005; Rousset et al 2004).

28.1.5. Hazard identification conclusion

Coxiella burnetii is an exotic, notifiable, zoonotic organism. It is classified as a hazard in the commodity.

28.2. RISK ASSESSMENT

28.2.1. Entry assessment

28.2.1.1. Semen

Coxiella burnetii is excreted in semen of bulls and mice (Kruszewska and Tylewska-Wierzbanowska 1997; Kruszewska and Tylewska-Wierzbanowska 1993). Therefore the likelihood that it could enter in the semen of deer is assessed to be non-negligible.

28.2.1.2. Embryos

No reports were found to suggest transmission of *C. burnetii* by embryo transfer. Since *C. burnetii* is frequently isolated from placentas and foetuses (Hatchette et al 2003; Marin and Raoult 1999; Marrie 1990), it is possible that the genital tract of females could be infected and that embryos could be contaminated. The likelihood that embryos could be contaminated with *C. burnetii* is assessed to be low.

28.2.2. Exposure assessment

Imported germplasm would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is assessed to be high.

28.2.3. Consequence assessment

Since other species can remain infected with *C. burnetii* for long periods it is possible that infected recipient deer too could remain carriers and shed the organism at parturition and in milk (Marrie 1990).

Establishment of *C. burnetii* in New Zealand would be likely to have a negligible effect on animal industries as infection of livestock is usually subclinical. It might have more important effects on the goat industry as up to 30% abortions have been recorded in some flocks (Arricau-Bouvery and Rodolakis 2005). The New Zealand cattle tick could also become infected (Heath 2002) and play an important role in the organism becoming endemic.

Establishment of the disease would result in sporadic cases of serious disease in people. Virtually all animal species, including birds and fish, could be infected although these infections are likely to be subclinical. The effects on the environment would not be noticeable.

Since the disease could establish in New Zealand and result in sporadic human infections, the consequences are assessed to be non-negligible.

28.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for *C. burnetii* is non-negligible and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

28.3. RISK MANAGEMENT

28.3.1. Options

The following points were considered when drafting options for the management of *C. burnetii* in the commodity:

- Q fever is not an economically important disease of livestock but sporadically causes serious disease in humans.
- Infected animals may remain carriers for long periods and quarantine would not be an effective measure to prevent entry.
- Contamination of deer germplasm has not been described but venereal transmission has been reported in cattle, humans and mice.
- The organism can infect many species of ticks and may be transmitted in tick faeces.
- Treatment and vaccination are not validated methods for eliminating the risk of importing the organisms in deer germplasm.

There are no recommendations in the *Code* relevant to *C. burnetii*.

One or a combination of the following options could be considered.

1. Quarantine in tick-free premises could ensure that animals do not become infected with the disease shortly before or during the collection of germplasm. Donors could be treated with a suitable acaricide and inspected to ensure that they are free from ticks and maintained tick-free while in quarantine for 30 days.

2. Donor animals could be quarantined for at least 30 days before the date of collection and subjected to either an ELISA or complement fixation test with negative results.

3. Donors could be tested by an ELISA, with negative results, 21-60 days after the final collection of the germplasm. A positive test could result in prohibition of importation of the germplasm. Given the tendency for infected animals to be long-term carriers, any donors which are known to have previously tested positive for *C. burnetii* should be excluded.

N.B. Work is presently being undertaken to develop a PCR for testing semen. If this test is validated and becomes available, testing of individual batches of semen and embryos may become a possibility and could replace serological testing of donors.

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29. *Salmonella* (Exotic spp.)

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agent

There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2008). 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 (Hogue et al 1997; Jones et al 2002). It is now widely distributed in the world.

29.1.2. OIE list

Salmonellosis (*S.* Abortusovis) is listed within the category of sheep and goat diseases, however, there is no *Code* chapter. In the OIE *Manual of Diagnostic Tests and Vaccines*, salmonellosis is included in the section "Diseases not covered by List A and List B" (Davies 2008).

29.1.3. 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.

29.1.4. Epidemiology

Salmonella spp. isolated in New Zealand from humans and animals, by all major laboratories, are identified to serovar and phage type by the ESR's Enteric Reference Laboratory and recorded in a database (ESR 2009). In this database *Salmonella* spp. isolated from deer are not listed separately. However, 55 of 2,350 isolates made in 2007 and 2008 were from unidentified sources. Therefore it is concluded that isolates from deer are uncommon.

Information in this section relates mainly to *S.* Typhimurium and *S.* Dublin which are the most common *Salmonella* spp. isolated from cattle overseas. *S.* Dublin has not been isolated in New Zealand. In other countries, it occurs most commonly in cattle but also occurs in sheep and, may therefore, be a pathogen of deer also.

Salmonella Typhimurium is endemic in New Zealand in both animals and humans, but DT104 has only been isolated rarely from humans and not from livestock. It has been

isolated from three dogs in a household where the owners suffered from diarrhoea after returning from overseas (Julian 2002). The sporadic occurrence of *S. typhimurium* DT104 in a few humans and once in dogs indicates that it has not become established in the New Zealand animal population.

Salmonella have been isolated from sick and healthy farmed and wild deer (McAllum et al 1978; Renter et al 2006; Sato et al 2000) including an isolate of *S.* Typhimurium DT104 from elk (Foreyt et al 2001). Deer are susceptible to salmonellosis and there is no reason to believe that the epidemiolology, pathogenesis, signs of infection and pathology would differ from that in cattle.

Salmonella infection occurs by the oral route and factors such as infecting dose, the particular strain and species, and various stress factors influence the outcome of infection (Fenwick and Collett 2004). The incubation period is variable but the organisms may be found in the bloodstream of newborn calves within 15 minutes of ingestion (Radostits et al 2007). The intestine is initially infected and acute enteritis is the primary lesion. Initial infection may be followed by penetration of the intestinal and mesenteric lymph node barrier followed by bacteraemia and dissemination to several organs. In the case of pregnant animals, abortion is common, particularly with *S.* Dublin. Animals that recover from *S.* Dublin infections frequently become carriers for life, shedding organisms sporadically in their faeces. Animals infected with *S.* Typhimurium may be carriers for 3-4 months.

Excreted organisms contaminate the environment and become a source of infection (Radostits et al 2007). Young animals are more often affected by the disease than adults and very young animals may die after a short period of bacteraemia.

In cattle, carriers of infections can be detected by culturing faecal samples but, because excretion is intermittent, repeated sampling and culture is necessary (Davies 2008). Serology may be useful but is best applied on a herd basis (Davies 2008).

29.1.5. Hazard identification conclusion

Deer could be carriers of exotic *Salmonella* serovars and so these pathogens are classified as a hazard in the commodity.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

29.2.1.1. Semen

There is no information about the occurrence of *Salmonella* serovars in deer semen and therefore information must be extrapolated from what is known about cattle. Infection of bulls with *Corynebacterium pyogenes* resulted in secondary infection of the reproductive tract with *S.* Morbificans which had been present in the alimentary tract (Boryczko and Furowicz 1971). Since septicaemia occurs during *Salmonella* infections, the organism may contaminate semen. Semen could also become contaminated by faeces particularly in animals with diarrhoea and have soiled skin and hair.

Because of the common occurrence of antibiotic resistance in *Salmonella* spp. (Jones et al 2002; Wray et al 1991), the use of antibiotics in semen diluents is not a reliable method of

eliminating them from semen. The likelihood of entry of *Salmonella* spp. in semen is therefore assessed to be non-negligible.

29.2.1.2. Embryos

Salmonella spp. are excreted in vaginal discharges following abortions and, therefore, could contaminate embryos. The IETS does not list *Salmonella* spp. in any risk category, thereby indicating that work on transmission by embryo transfer has not been undertaken (*Code* Article 4.7.14.). Because of the common occurrence of antibiotic resistance in *Salmonella* spp. (Jones et al 2002; Wray et al 1991), the use of antibiotics in embryo preparation cannot be regarded as a reliable method of eliminating *Salmonella* spp. from embryos. The likelihood of entry of *Salmonella* spp. in embryos is therefore assessed to be non-negligible.

29.2.2. Exposure assessment

Imported germplasm would be inseminated or implanted into susceptible cattle and the likelihood of exposure is assessed to be high.

29.2.3. Consequence assessment

Whether the intrauterine deposition of *Salmonella* would lead to infection is not known but seems unlikely. However, in the absence of information, a risk-averse position should, perhaps, be taken and it be assumed that infected germplasm could result in infection of recipients, which could then excrete the organism and infect other livestock and people. The introduction and establishment of any new *Salmonella* spp. could result in its spread in New Zealand. An example of how an emerging *Salmonella* sp. can affect New Zealand animal industries can be seen in the case of *S.* Brandenburg which first emerged in 1997 and subsequently spread to many farms causing abortions in hundreds of sheep flocks (Kerslake and Perkins 2006). Introduction of new *Salmonella* spp. could therefore have significant economic consequences for the animal industries.

Because of its resistance to antibiotics and zoonotic nature, establishment of *S.* Typhimurium DT104 in animal populations (Davies 2008; Hogue et al 1997) would be of particular concern for human health.

Salmonella Typhimurium DT160 was reported to be associated with several hundred deaths in sparrows (Alley et al 2002). The outbreak was self-limiting and did not cause lasting damage to the sparrow population. However, in subsequent years that phage type became the one most commonly isolated from humans (ESR 2009). Newly introduced serovars could potentially have similar deleterious effects on human health.

There would be no particular consequences for the environment other than possibly sporadic cases of salmonellosis in wild animals such as deer and goats. Infected wild animals could be a source of infection for domestic animals

In conclusion, the introduction of contaminated germplasm could lead to the establishment of new *Salmonella* spp. that have the potential to cause disease in humans and animals. Therefore the consequences are assessed to be non-negligible.

29.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for exotic *Salmonella* serovars is non-negligible, and they are classified as a risk in the commodity. Therefore, risk management measures may be justified.

29.3. RISK MANAGEMENT

The following points were considered when drafting options for the management of *Salmonella* serovars in the commodity:

- Many strains of *Salmonella* are resistant to a wide range of commonly used antibiotics (Jones et al 2002; Wray et al 1991) and their use in semen diluents or embryo wash fluids cannot be relied upon to eliminate *Salmonella*. Extenders used to dilute turkey semen failed to eliminate *Salmonella* (Donoghue et al 2004).
- Repeated culture of faeces to ensure that donors are not carriers may not be a reliable procedure because shedding of the organism is intermittent.
- Since culture of *Salmonella* spp. from a variety of sample types is well documented (Davies 2008), culturing aliquots of semen and embryos from all collection batches could be used to demonstrate freedom from *Salmonella* spp. As germplasm for export has generally had antibiotics added to it, it must be assumed that failure to culture organisms indicates that they are either not present or have been inactivated by the antibiotics. This measure is not ideal because antibiotics that are bacteriostatic may suppress growth of organisms in culture without eliminating them. However, use of pre-enrichment medium would assist the isolation of damaged organisms by dilution of any antibiotics present and resuscitation of damaged organisms (Davies 2008).
- As long-term carriers occur commonly, quarantine is not a useful option.
- Suitable vaccines are not available for a wide range of serotypes.
- It should be kept in perspective that the likelihood of introducing exotic *Salmonella* in deer germplasm is extremely unlikely when compared to the risk of introduction by millions of human travellers that arrive annually in New Zealand.

One or a combination of the following options could be used in order to manage the risk.

1. Importation could be permitted without restriction based on the fact that transmission in germplasm has not been described in deer, the semen will contain antibiotics and introduction of new *Salmonella* through deer germplasm represents a minor pathway. There are currently no specific measures against *Salmonella* required when importing cervine semen from Great Britain.

2. The veterinary administration of the exporting country could be required to certify that the donors originate from farms on which outbreaks of salmonellosis have not occurred during the previous 3 years.

3. Aliquots of semen and substandard embryos or an aliquot of embryos could be cultured according to OIE recommended culture methods (Davies 2008). All *Salmonella* spp. isolated could be serotyped (and, where appropriate, phage typed) and the results reported to MAF. A pre-enrichment medium could be used before culturing on selective and non-selective media. Where pathogenic

Salmonella spp., exotic to New Zealand are isolated, importation of germplasm could be prohibited.

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30. Chronic wasting disease

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

Chronic wasting disease (CWD) is generally considered to caused by a protein-only agent called a prion.

30.1.2. OIE list

Not listed.

30.1.3. New Zealand status

Exotic.

30.1.4. Epidemiology

CWD is a naturally occurring disease of elk, white-tailed deer, mule deer (Salman 2003; Williams 2005) and moose (Singeltary 2008). The disease is confined to North America (Scientific Steering Committee of the European Commission 2003). However, a single case in an elk imported into Korea from Canada led to the slaughter of 101 of 144 imported deer. Nine of these were diagnosed as having CWD. During the investigation the Korean authorities were unable to trace 43 of the 144 imported deer. Vertical and horizontal transmission of CWD was also examined by slaughtering and inspecting all deer that had been kept with the imported deer and no further cases were identified in the indigenous deer. However, a second outbreak, involving four cases in elk occurred in 2004. It is not known whether these cases were in imported deer missed in the 2001 investigation or were in indigenous animals (Kim et al 2005).

CWD is an invariably fatal chronic disease of wild and farmed/captive deer characterised by typical prion disease brain pathology. It was described in 2005 as increasing in prevalence (Williams 2005). At least 34 communications relating to CWD have been posted in ProMed in the period 11/2007 to 11/2009 (ProMed 2009).

In cases of CWD, infectivity is present in many tissues and infected animals are contagious. It has been stated that prevalence of infection can reach 100% in farmed deer (Martin et al 2009). The disease has a long incubation period with a minimum of 16 months and a probable average of 2-4 years. The disease has been diagnosed in an elk aged more than 15 years old and a white-tailed deer older than 12 years (Williams 2005).

There is no evidence that CWD is transmitted in semen or by embryos. It has been stated that the “epidemiology of chronic wasting disease (CWD) of deer, and on transmission risks in other species, provides optimism that transmission of CWD via semen and embryos of deer is unlikely” (Wrathall et al 2005). However, there is no evidence to support this statement and veterinary authorities have generally adopted a risk-averse stance for the importation of germplasm (AQIS 2009; USDA 2009) or for surveillance and certification programmes (Oklahoma Department of Agriculture 2009). The fact that there

is an early and widespread appearance of infectivity in tissues underlines the necessity for a conservative approach to regulation.

The disease is diagnosed post-mortem by histopathology or immunohistochemistry (Martin et al 2009). In live animals diagnosis can be made by immunohistochemical examination of biopsy samples from retropharyngeal lymph nodes, palatine tonsil or recto-anal mucosa-associated lymphoid tissue (RAMALT) (Spraker et al 2009). Five rapid diagnostic tests (ELISA) are licenced in the USA. The BioRad ELISA was found to have 100% sensitivity in elk and 98.3% sensitivity in mule deer when compared to immunohistochemistry (Williams 2005). In elk 10-15% of infected animals had detectable prions in the brain but not in retropharyngeal lymph nodes.

30.1.5. Hazard identification conclusion

Germplasm donors from North America and Korea could be infected with CWD. Therefore, germplasm from North America and Korea represents a hazard. Germplasm from other countries does not constitute a hazard for introduction of the CWD agent.

30.2. RISK ASSESSMENT

30.2.1. Entry assessment

30.2.1.1. Semen

CWD has a long incubation period during which infected animals do not show any clinical signs and could be used as donors during this time.

The transmission of CWD in semen has not been studied. However, one can extrapolate from what is known about related diseases. The related disease of cattle, bovine spongiform encephalopathy (BSE), is non-contagious and the *Code* (Article 11.6.1.) lists semen as a commodity for which there is no BSE risk. However, CWD is contagious and analogy with BSE is inappropriate. The sheep disease scrapie could be considered more analogous than BSE.

Evidence from epidemiological studies and experimental matings of scrapie-affected rams suggests that scrapie is unlikely to be transmitted by semen (Wrathall 2000, Wang et al 2001, Wrathall et al 2008). Palmer (1959) failed to transmit scrapie by subcutaneous injection into lambs of semen from a clinically affected ram. The lambs were, however, only observed for 30 months post-inoculation and this observation period would now be considered less than ideal.

Bioassays in mice have failed to detect scrapie infectivity in testis, seminal vesicles and semen of affected rams (Hourrigan et al 1979, Hourrigan 1990, Hadlow 1991, Hourrigan and Klingsporn 1996). Hourrigan reported the failure to detect infectivity in semen samples from 21 cases of scrapie (Hourrigan 1990).

Gatti and colleagues were unable to detect PrP^{sc} in seminal plasma of rams with scrapie, suggesting that infectivity was absent (Gatti et al 2002). The conclusions of this study have recently been confirmed by an experiment in which semen from infected rams was inoculated into scrapie-susceptible transgenic mice expressing the VRQ allele of the sheep prion gene (Sarradin et al 2008).

The transgenic mouse model used by Sarradin and colleagues (2008) has been shown to be capable of detecting very low levels of infectivity. The study reported by Sarradin and others (2008) demonstrated that scrapie was not transmitted by semen at any time during the incubation period of scrapie in four rams, even from one of the highly susceptible VRQ/VRQ genotype during the clinical stages of the disease.

Extrapolating from what is known about the presence of scrapie infectivity in sheep semen, it can reasonably be assumed that there is very little likelihood that CWD infectivity would be present in semen of deer.

However, although not studied, it has been suggested that there could be potentially abundant infectious agent in the lymphoid cells associated with the prepuce of infected deer. Such cells could be a source of contamination for semen and have the potential to allow for efficient transmission (Reid 2010).

In the absence of any direct evidence, it must be assumed that the risk of CWD entry in semen is non-negligible.

30.2.1.2. Embryos

The *Code* states that *in vivo*-derived embryos can be traded safely without any BSE (Article 11.6.1.) or scrapie risk (Article 14.9.1.). The safety of sheep embryos with respect to scrapie was recognised by the OIE in May 2010 following advice from the IETS which in February 2010 assessed all the peer-reviewed studies that have been published on the subject.

Extrapolating from what is known about the presence of scrapie infectivity in sheep embryos, it can reasonably be assumed that there is very little likelihood that CWD infectivity would be present in *in vivo*-derived deer embryos. However, in the absence of any direct evidence, it must be assumed that the risk of CWD entry in embryos is non-negligible.

30.2.2. Exposure assessment

By extrapolation from what is known about the related prion disease scrapie, it is reasonable to assume that CWD is unlikely be transmitted by germplasm. However, CWD is infectious and infectivity is widely distributed in many tissues. Therefore, since imported germplasm will be inseminated/transplanted directly into New Zealand deer the likelihood of exposure is assessed to be high.

30.2.3. Consequence assessment

CWD is a contagious disease that would be likely to spread in farmed and feral deer. Introduction would be likely to go unnoticed for a protracted time period in which the disease could become widespread. It would cause economic losses for deer farmers and cause mortalities in feral deer. There would be no implications for human health or the environment apart from effects on wild deer. Since there could be economically important consequences for deer farmers the consequence assessment is non-negligible.

30.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, the risk estimate for the introduction of the CWD agent in the commodity is non-negligible, and it is classified as a risk in the commodity. Therefore, risk management measures may be justified.

30.3. RISK MANAGEMENT

The following points were considered when drafting options for the management of CWD in the commodity:

- CWD occurs in North America and outbreaks have been reported from Korea.
- It is not known whether or not CWD can be transmitted by germplasm. In the absence of direct study, a risk-averse policy that assumes that it could, is appropriate.
- There are some initial attempts to identify and maintain disease free herds of deer in the USA but these are probably not yet well established.
- The continuing spread of the disease in the USA and Canada means that boundaries of infected areas cannot be reliably defined.
- Diagnostic tests for live animals are available but have not been adequately validated for deer, and adequate sensitivity cannot be assumed.
- There are no vaccines or treatments for the disease.

One or a combination of the following options could be used in order to manage the risk.

1. Germplasm from all countries except the USA, Canada, Mexico and Korea could be introduced provided CWD is notifiable and never been diagnosed.
2. Importation of germplasm from North American countries and Korea could be prohibited.
3. Germplasm donors from North American countries and Korea could be certified as having lived their entire lives in herds in which there have been no cases of CWD for at least the last 4 years and have been tested by an immunohistochemical test on samples of recto-anal mucosa-associated lymphoid tissue (RAMALT).
4. Germplasm donors from North American countries and Korea could be certified as having lived their entire lives in herds in which there have been no cases of CWD for at least the last 4 years and in which all adult deer have been tested by an immunohistochemical test on samples of recto-anal mucosa-associated lymphoid tissue (RAMALT).

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