

***Import Risk Analysis: Cattle  
germplasm from all countries***

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June 2008

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**Biosecurity New Zealand  
Ministry of Agriculture and Forestry  
Wellington  
New Zealand**



**June 2008**

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Policy and Risk  
Biosecurity New Zealand

*Import risk analysis: Cattle germplasm from all countries*

June 2008

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## EXECUTIVE SUMMARY

This risk analysis covers the import of frozen bovine semen and *in vivo* derived bovine embryos from all countries.

An initial list of 86 disease agents was compiled. The list did not include arthropod and nematode parasites as these cannot be carried by semen or embryos. Further consideration of these resulted in a *Preliminary Hazard List* of 37 disease agents or groups of disease agents, which were subjected to risk analysis. In some cases risk analysis was done on a group of agents rather than a single agent e.g. Simbu group viruses, *Salmonella* spp., mollicutes of cattle etc.

28 of these preliminary hazards were considered to be potential hazards and were subjected to a risk assessment. 12 potential hazards were assessed to be associated with a negligible risk and, in these cases, no risk management measures are required.

A non-negligible risk was identified with the following hazards:

- Borna disease virus
- Bovine viral diarrhoea virus type 2
- Crimean Congo haemorrhagic fever virus
- Foot and mouth disease virus
- Exotic bovine herpes viruses
- Lumpy skin disease virus
- Rift Valley fever virus
- Vesicular stomatitis virus
- Exotic *Brucella* spp.
- *Mycobacterium bovis*
- *Mycoplasma mycoides* subsp. *mycoides* SC
- Other exotic *Mycoplasma* spp.
- Exotic *Salmonella* spp.
- Exotic *Leptospira* spp.
- *Chlamydomphila abortus*
- *Coxiella burnetii*

Options for risk management measures in order to effectively manage the risk associated with each of these hazards have been presented.

# 1 INTRODUCTION

This risk analysis has been developed in response to a request from the Animals Import section of MAF Biosecurity New Zealand.

# 2 SCOPE

This risk analysis is limited to the description of the risks due to disease-causing organisms associated with the importation of cattle embryos and semen. Other risk factors that may be of commercial importance to importers (e.g. genetic diseases) have not been considered in the analysis.

The analysis is restricted to the risks posed by viral, bacterial, and protozoal diseases. Diseases caused by external and internal parasites are excluded because these parasites cannot be transmitted by semen or embryos.

# 3 COMMODITY DEFINITION

The commodities considered are frozen semen and *in vivo* derived frozen embryos from healthy cattle. Fresh semen, *in vitro* derived embryos, and cloned embryos are specifically excluded from this risk analysis. Semen and embryos are referred to collectively as germplasm. It is assumed that the commodities will:

- be collected and processed at suitable collection centres and laboratories approved for the purpose by the veterinary administration of the exporting country. The collection centres shall meet the standards for collection centres specified in sections 3.2.1 and 3.3.1 in the *OIE Terrestrial Animal Health Code*.
- only be collected from clinically healthy donors.
- undergo diagnostic testing at a laboratory approved by the veterinary administration of the exporting country.
- be processed, packaged, stored, and transported according to standards laid down in the *OIE Terrestrial Animal Health Code* (OIE 2006) and The Research Subcommittee of the International Embryo Transfer Society, Health and Safety Advisory Committee (IETS 2004), including the use of antibiotics and trypsin as recommended by IETS.
- be stored in the frozen state for at least 28 days before shipment to New Zealand, and that during this time the donor animals and all animals in contact with them will have remained healthy and free from any diseases that are considered to be of non-negligible risk in this risk analysis.

## 4 METHODOLOGY OF RISK ANALYSIS

The methodology used in this risk analysis follows the guidelines as described in *Import Risk Analysis: Animals and Animal Products* (Murray 2002)<sup>1</sup> and in section 1.3 of the *Terrestrial Animal Health Code* of the World Organisation for Animal Health (OIE 2006).

The risk analysis process used by the MAF is summarised overleaf in Figure 1.

### 4.1 HAZARD LIST

The first step in the risk analysis is hazard identification. The process begins with the collation of a list of organisms that might be associated with germplasm from cattle. The diseases of interest are those that could be transmitted in cattle germplasm and could infect domestic, feral or wild animals that occur in New Zealand, and man. In this case a list was made of all the cattle diseases that are classified as listed diseases in the year 2005 edition of the OIE *Terrestrial Animal Health Code* and diseases mentioned in the following sources:

Veterinary Medicine. Radostits OM, Blood DC, and Gay CC. 8<sup>th</sup> edition. Bailliere Tindall, London, Philadelphia, Sydney, Tokyo, Toronto. 1994.

Infectious Diseases of Livestock. Coetzer JAW and Tustin RC. 2<sup>nd</sup> edition. Oxford University Press, Cape Town, Oxford, New York. 2004.

Foreign Animal Diseases “The Gray Book”  
[www.vet.uga.edu/vpp/gray\\_book/FAD/SGP.htm](http://www.vet.uga.edu/vpp/gray_book/FAD/SGP.htm).

The MAF databases that contain complete listings of all diseases of cattle that appears in Import Health Standards (IHSs) and Overseas Market Access Requirements (OMARs) for all countries for which the information is available.

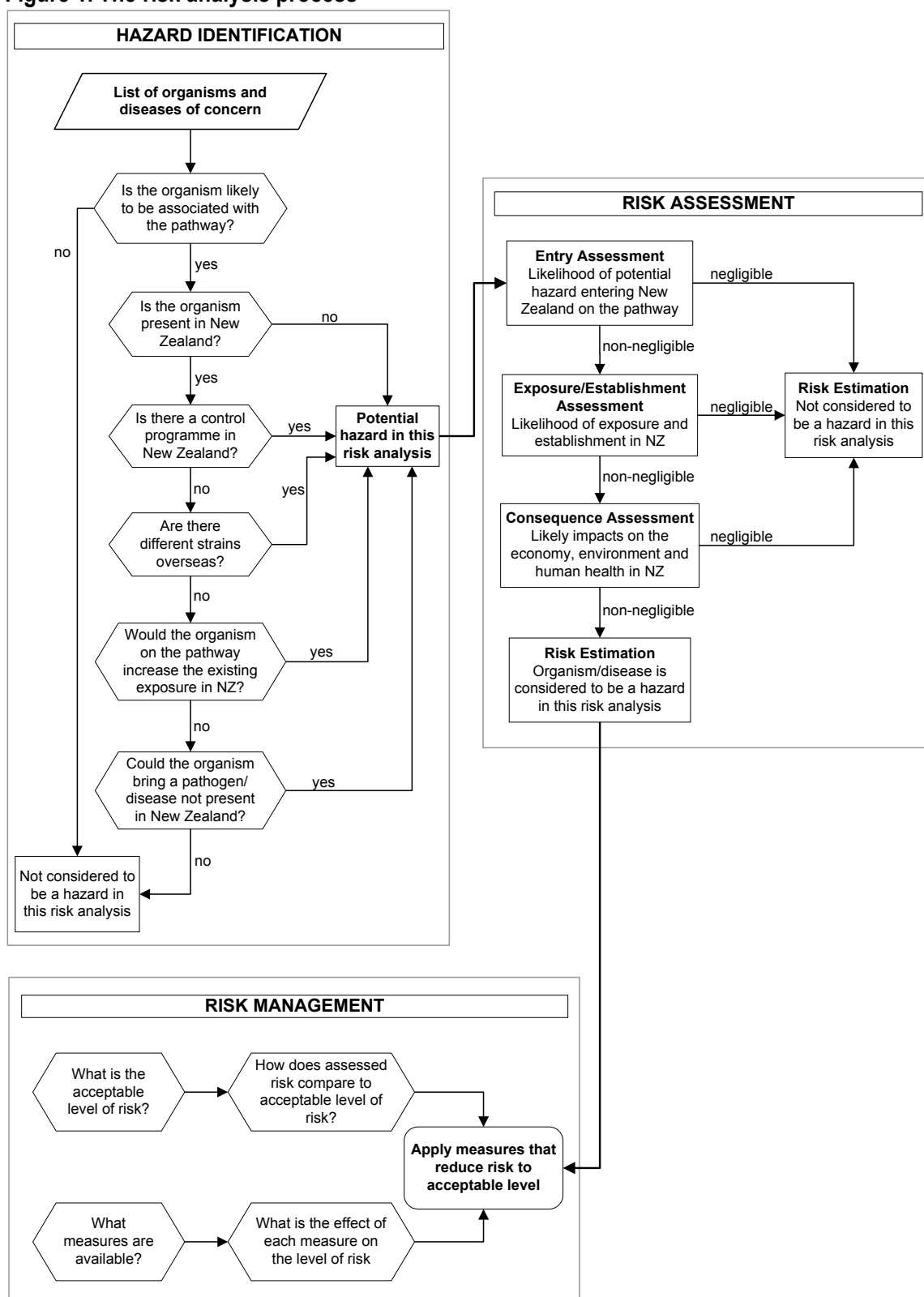
Organisms classified as unwanted by the New Zealand Ministry of Agriculture and Forestry (Ministry of Agriculture and Forestry 2005).

The diseases of cattle that were identified in these sources are listed in Table 1.

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<sup>1</sup> Risk analysis projects commenced after 12 April 2006 follow the guidelines documented in Biosecurity New Zealand’s *Risk Analysis Procedures – Version 1* (see: [www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf](http://www.biosecurity.govt.nz/files/pests-diseases/surveillance-review/risk-analysis-procedures.pdf))

Figure 1. The risk analysis process



**Table 1. Initial hazard list of organisms.**

<b>ORGANISM</b>	<b>OIE LIST</b>	<b>ZOONOTIC</b>	<b>NEW ZEALAND STATUS</b>	<b>NOTES</b>
<b>VIRUSES</b>				
Akabane (Simbu viruses)	No	No	Exotic	
Aujeszky's disease virus	Yes	No	Exotic	
Adenovirus virus	No	No	Endemic(Vermunt and Parkinson 2000a)	
Bluetongue virus	Yes	No	Exotic	24 serotypes
Borna disease virus	No	?	Exotic	
Bovine calicivirus	No	No	Unknown	
Bovine corona virus	No	No	Endemic(Durham et al 1979; Vermunt and Parkinson 2000a)	
Bovine herpes virus-1 (IBR/IPV)	Yes	No	BHV-1.2b endemic. BHV-1.1 and 1.2a exotic	
Bovine herpesvirus-2	No	No	Endemic(Vermunt and Parkinson 2000a; Vermunt and Parkinson 2000b)	
Bovine herpesvirus-5	No	No	Exotic	
Bovine parvovirus	No	No	Unknown	
Bovine papular stomatitis virus	No	No	Endemic (Vermunt and Parkinson 2000a)	
Bovine respiratory syncytial disease virus	No	No	Endemic (Motha and Hansen 1997)	
Bovine rhinovirus	No	No	Unknown	
Bovine viral diarrhoea virus	No	No	BVDV1 endemic BVDV 2 exotic (Horner 2000)	Two types
Crimean Congo haemorrhagic fever virus	No	Yes	Exotic	
Enzootic bovine leucosis virus	Yes	No	Endemic	
Ephemeral fever virus	No	No	Exotic	
Foot and mouth disease virus	Yes	No	Exotic	7 serotypes multiple strains
Ibaraki	No	No	Exotic	
Jembrana disease virus	No	No	Exotic	
Lumpy skin disease virus	Yes	No	Exotic	
Malignant catarrhal fever virus	Yes	No	Wildebeest type exotic Sheep associated virus endemic	Wildebeest and Sheep associated
Miscellaneous arboviruses	No	No	All exotic	
Palyam virus group	No	No	Exotic	Many strains
Parainfluenza virus	No	No	Endemic	
Pseudocowpox virus	No	No	Endemic (Hill 1994)	

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Rabies	Yes	Yes	Exotic	Related rhabdoviruses
Rift Valley fever virus	Yes	Yes	Exotic	
Rinderpest virus	Yes	No	Exotic	Strains vary in virulence
Ross River virus and Barmah Forest virus	No	Yes	Exotic	
Rotavirus	No	No	Endemic (Durham et al 1979; Vermunt and Parkinson 2000a)	
Vesicular stomatitis virus	Yes	Yes	Exotic	3 subtypes
West Nile disease virus	No	Yes	Exotic	

### BACTERIA INCLUDING MOLLICUTES

<i>Actinobacillus lignieresii</i>	No	No	Endemic	
<i>Arcanobacter pyogenes</i>	No	No	Endemic	
<i>Bacillus anthracis</i>	Yes	Yes	Exotic	
<i>Brucella abortus</i>	Yes	No	Exotic	
<i>Burkholderia pseudomallei</i>	No	Yes	Exotic	
<i>Campylobacter fetus</i> subsp. <i>venerealis</i>	Yes	No	Endemic (Loveridge and Gardner 1993)	Subsp venerealis and fetus
<i>Campylobacter jejuni</i>	No	Yes	Endemic	
<i>Clostridium</i> spp.	No	No	Endemic	
<i>Corynebacterium renale</i>	No	No	Endemic	
<i>Dermatophilus congolensis</i>	Yes	Yes	Endemic	
<i>Escherichia coli</i>	No	Yes	Endemic	Plasmid and virulence types
Footrot associated organisms	No	No	Endemic	Various species
<i>Haemophilus somnus</i> ( <i>Histophilus ovis</i> ?)	No	No	Endemic	
<i>Klebsiella</i> spp	No	No	Endemic	
<i>Listeria monocytogenes</i>	No	Yes	Endemic	
<i>Mannheimia</i> ( <i>Pasteurella</i> ) <i>haemolytica</i>	No	No	Endemic	
<i>Moraxella bovis</i>	No	No	Endemic	
<i>Mycobacterium bovis</i>	Yes	Yes	Endemic/ control programme	
<i>Mycobacterium avium</i> subsp. <i>avium</i>	Yes	Yes	Endemic	
<i>Mycobacterium avium</i> subsp. <i>paratuberculosis</i>	Yes	No?	Endemic	
<i>Mycoplasma mycoides</i> subsp. <i>mycoides</i> SC	Yes	No	Exotic	
Mollicutes (various)				
<i>Nocardia</i> spp.	No	No	Endemic	
<i>Pasteurella multocida</i> B and E	Yes	No	Exotic	
<i>Pasteurella multocida</i> other than B and E	No	No	Endemic	
<i>Salmonella</i> spp.	No	Yes	Some serotypes exotic	
<i>Staphylococcus</i> spp.	No	Variable	Endemic	



<i>Streptococcus</i> spp.	No	Variable	Endemic
<i>Yersinia</i> spp.	No	Yes	Endemic

**SPIROCHAETES**

<i>Leptospira</i> spp.	Yes	Yes	6 serovars are endemic (Midwinter 1999)	Over 200 serovars
<i>Borrelia burgdorferi</i>	No	Yes	Exotic	
<i>Borrelia theileri</i>	No	No	exotic	

**PROTOZOAL PARASITES**

<i>Babesia</i> spp.	Yes	No	Exotic
<i>Besnoitia besnoiti</i>	No	No	Exotic
<i>Cryptosporidium parvum</i>	No	Yes	Endemic
<i>Eimeria</i> spp.	No	No	Endemic
<i>Neospora caninum</i>	No	No	Endemic
<i>Sarcocystis</i> spp.	No	No	S hirsuta and S cruzi endemic. S hominis unknown
<i>Theileria</i> spp.	Yes	No	One species endemic.
<i>Trichomonas foetus</i>	Yes	No	Endemic
<i>Trypanosoma evansi</i>	Yes	No	Exotic
<i>Trypanosoma</i> spp. tsetse fly-borne	Yes	No	Exotic

**RICKETTSIAL AND CHLAMYDIAL ORGANISMS**

<i>Anaplasma marginale</i> , <i>A. centrale</i> , <i>A. caudatum</i>	Yes	No	Exotic
<i>Anaplasma phagocytophilum</i>	No	Yes	Exotic
<i>Chlamydia abortus</i>	Yes	Yes	Exotic
<i>Coxiella burnetti</i>	Yes	Yes	Exotic
<i>Ehrlichia ruminantium</i>	Yes	No	Exotic
<i>Ehrlichia chaffeensis</i>	No	Yes	Exotic
<i>Eperythrozoon</i> spp.	No	No	Endemic
<i>Haemobartonella bovis</i>	No	No	Unknown

**PRIONS**

Bovine spongiform encephalopathy	Yes	Yes	Exotic
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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 that are published in the MAF publication *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 (Ministry of Agriculture and Forestry 2005).

A preliminary hazard list was then compiled from the agents listed in Table 1 which included all disease agents exotic to New Zealand, 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, and are potentially harmful, and organisms that occur in New Zealand but for which an eradication programme administered by a Pest Management Strategy under the Biosecurity Act is in place.

In addition, the preliminary hazard list also included any disease agents that are already in New Zealand but because of the nature of the imports are likely to significantly increase existing hazards associated with them, and any disease agents that occur only in well defined geographically bounded areas of New Zealand.

Organisms transmitted exclusively by insect vectors, which therefore cannot be transmitted in semen or by embryo transfer, were excluded from the preliminary hazard list. The organisms excluded on these grounds were *Babesia bovis*, *Babesia bigemina*, and other *Babesia* spp. (De Vos et al 2004), *Borrelia theileri* (Bishop 2004), *Borrelia burgdorferi* (Hodzic and Barthold 2004), Ephemeral fever virus (St George 2004), *Trypanosoma congolense*, *Trypanosoma vivax*, and *Trypanosoma brucei* (Connor and Van den Bossche 2004), *Trypanosoma evansi* (Luckins 2004; Pham-Sy-Lang et al 2001), *Ehrlichia chaffeensis* (Long et al 2003; Varela et al 2004; Varela et al 2003), *Theileria Parva*, *Theileria annulata*, and other mildly pathogenic *Theileria* spp (Lawrence et al 2004a; Lawrence et al 2004b; Lawrence et al 2004c; Lawrence and Williamson 2004a; Lawrence and Williamson 2004b; Lawrence and Williamson 2004c; Lawrence and Williamson 2004d; Pipano and Shkap 2004).

Protozoal parasites that cannot be transmitted by germplasm because they have a complex life-cycle requiring an intermediate host, or intestinal parasites where transmission occurs by the faeco-oral route were excluded from the preliminary hazard list. The organisms excluded on these grounds were *Besnoitia besnoitii* (Bigalke and Prozesky 2004), *Cryptosporidium parvum* (Stewart and Penzhorn 2004), *Eimeria* spp. (Stewart and Penzhorn 2004), and *Sarcocystis* spp. (Marcus et al 2004).

Disease agents that are predominantly transmitted by insects that have been included in the preliminary hazard list include bluetongue virus, Palyam group viruses, and Simbu group viruses which have been demonstrated in semen (Gard et al 1989), and Ibaraki virus which is closely related to bluetongue virus.

The preliminary hazard list consisted of:

### Viruses

Akabane disease virus and other Simbu group viruses	IBR/IPV virus (genital form)
Aujeszky's disease virus	Ibaraki virus
Bluetongue virus	Jembrana virus
Borna disease virus	Lumpy skin disease virus
Borna disease virus	Malignant catarrhal fever virus
Bovine calicivirus	Palyam group viruses
Bovine parvovirus	Rabies virus
Bovine rhinovirus	Rift Valley fever virus
Bovine virus diarrhoea virus	Rinderpest virus
Crimea Congo haemorrhagic disease virus	Vesicular stomatitis virus
Foot and mouth disease virus	West Nile disease virus

### Bacterial diseases

<i>Bacillus anthracis</i>	Mollicutes of cattle
<i>Brucella abortus</i>	<i>Pasteurella multocida</i> B and E
<i>Burkholderia pseudomallei</i>	<i>Salmonella</i> spp. (exotic species)
<i>Mycobacterium bovis</i>	
<i>Mycoplasma mycoides</i> subsp. <i>mycoides</i> SC	

### Spirochaetes

*Leptospira* spp.

### Rickettsial and Chlamydial organisms

<i>Anaplasma marginale</i>	<i>Coxiella burnetii</i>
<i>Anaplasma centrale</i>	<i>Haemobartonella</i>
<i>Chlamydophila abortus</i>	

### Prion diseases

Bovine spongiform encephalopathy

Organisms in the preliminary hazard list were subjected to further analysis to determine whether they were considered potential hazards (see sections on hazard identification of individual diseases) and all organisms considered to be potential hazards were subjected to a full risk assessment.

## 4.2 RISK ASSESSMENT

Under the MAF Biosecurity New Zealand and OIE methodologies, risk assessment consists of:

- a) Entry assessment - the likelihood of the organism being imported in 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, exposure, establishment or spread of the organism.
- d) Risk estimation - a conclusion on the risk posed by the organism based on the release, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a hazard.

It is important to understand that not all of the above steps may be necessary in all risk assessments. The MAF Biosecurity New Zealand and OIE methodologies make it clear that if the likelihood of entry is negligible for a certain potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises where 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 where both entry and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

## 4.3 RISK MANAGEMENT

For each organism classified as a hazard, a risk management step is carried out, which identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a hazard, these are described alongside options of similar, lesser, or greater stringency where available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all hazards. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted. As obliged under Article 3.1 of the WTO Agreement on Sanitary and Phytosanitary Measures (the SPS Agreement) the measures adopted in IHSs will 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).

## 4.4 RISK COMMUNICATION

This draft import risk analysis is issued 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 they consider necessary or preferable.

Following this period of public consultation on this draft document, a review of submissions will be produced and a decision-making committee will determine whether any changes need to be made to this draft risk analysis.

Following this process of consultation and review, the Imports Standards team of MAF Biosecurity New Zealand will decide on the appropriate combination of sanitary measures to ensure the effective management of identified risks. These will be presented in a draft IHS which will also be released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to the draft IHS will be reviewed before a final IHS is issued.

## 4.5 SPECIAL CONSIDERATIONS

Importation of semen and particularly embryos is generally accepted as being much safer than importing live animals. However, for many diseases there is little information available in the literature relating to the ability of semen and embryos from infected animals to transmit diseases. In the case of bluetongue, cattle that are in the viraemic stage of the disease may excrete the infectious agent in their semen (Bowen et al 1983; Howard et al 1985). Callis reviewed the literature and found that foot and mouth disease virus may be found in semen for up to 10 days after experimental infection (Callis 1996) which correlates with the time the animals are likely to have been viraemic. The etiological agents of lumpy skin disease (Irons et al 2005) and Q fever (Kruszewska and Tylewska-Wierzbanska 1997) have been found in the semen of infected animals. Although seminal excretion of infectious agents may not have been demonstrated in many diseases it is assumed in this risk analysis that any animal that is in the viraemic or bacteraemic stage of an infectious disease may excrete the infectious organism in their semen. In principle, semen or embryos should never be collected from animals that are febrile or showing clinical signs of an infectious disease and semen collected from febrile animals may be of inferior quality. However, in some diseases e.g. foot and mouth disease (Sanson 1994) animals may excrete infectious agents before showing clinical signs of infection. In this risk analysis it is assumed that semen or embryos are collected only from animals that have been examined and found to be healthy. However, this does not exclude the possibility that they could be excreting infectious agents in semen since, in some cases, animals may show no clinical signs while viraemic.

Donors of germplasm should be kept on germplasm collection centres that meet the standards of the *OIE Terrestrial Animal Health Code* (appendix 3.2.2 and the applicable parts of appendix 3.3). The methods of preparation of embryos and semen should follow OIE recommended methods. Washing of embryos and inclusion of antibiotics or trypsin in washing fluids and addition of antibiotics to semen influences the survival of pathogens in prepared germplasm and the adherence of organisms to the zona pellucida.

Embryo transfer is generally regarded as the safest means of introducing new genetic material to a country (Thibier and Geurin 2000). However, in many cases data that conclusively show that the procedure is safe are not available. The Research Subcommittee of the International Embryo Transfer Society (IETS 2004) Import/Export Committee, produces data relating to the safety of embryo transfer procedures. Diseases for which information is available are classed in four categories of risk. This list (which was updated in 2004) is published in section 3.3.5 of the *OIE Terrestrial Animal Health Code*. In this risk analysis information additional to that supplied by IETS has been sought and used, where it could be found. In the case of viral and bacterial diseases where no evidence is available to indicate otherwise, it is assumed that the instillation of semen or embryos that are contaminated with infectious organisms into the uterus of a recipient animal will result in that animal becoming infected with the organism. However, this is not assumed for those organisms (particularly protozoa) that are known to be transmitted only by biological transmission involving arthropod vectors.

Donors of embryos are both the male and the female donors. It is assumed that male donors will be of equal health status to the female donor at the time of semen donation or natural mating.

The incubation period and the time for which an animal may remain viraemic are critical parameters for determining quarantine periods. An animal could have been infected with a disease on the day it goes into quarantine. After the incubation period for the disease, it could then be viraemic or bacteraemic for a period that differs for each disease. Before semen or embryos are collected, donor animals could be quarantined for the maximum known incubation period plus the maximum period for which viraemia can last. Ideally the maximum period would be the mean period plus three standard deviations. This would cover approximately 99% of cases. However, usually the true distribution of incubation period and viraemia is not known because data are not available from a sufficiently large number of cases or because of technical difficulties in obtaining accurate data. Data quoted for the period of viraemia or bacteraemia is also unreliable because of the small numbers of animals that can be used and because the presence of viraemia is not measured continuously but at discrete intervals. If viraemia was determined at ten day intervals and an animal was viraemic on day ten but not at day 20, this really means that viraemia could have continued between 10 and 20 days. The measurement of viraemia is also dependant on the accuracy and sensitivity of the method used to determine it. For these reasons a conservative margin of error may be added to the best available estimates when determining quarantine periods. The margin of error added cannot be scientifically determined but relies on judgement, taking into account such things as amount and perceived accuracy of the available data, type of disease, and methods that were used to

measure viraemia. Generally in this risk analysis, suggested quarantine periods are adjusted to whole weeks or months. When Import Health Standards are later written for particular cases these suggested periods may be modified.

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## 5 AKABANE DISEASE

### 5.1 HAZARD IDENTIFICATION

#### 5.1.1 Aetiological agent

Family: Bunyaviridae; Genus: Bunyavirus. Serogroup Simbu. Akabane disease virus and related viruses belong to a group known collectively as Simbu viruses (St George and Kirkland 2004). The group includes viruses such as Aino, Tinaroo, Peaton, and Cache Valley viruses that cause similar syndromes.

#### 5.1.2 OIE list

Not listed.

#### 5.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic unwanted organism.

#### 5.1.4 Epidemiology

Akabane and related viruses have been isolated from *Culicoides* spp. (midges) and mosquitoes. *Culicoides* spp. are assumed to be the vectors these viruses (St George and Kirkland 2004). Cattle and other ruminants including sheep; (St George and Kirkland 2004; Charles 1994; Haughey et al 1988) and goats (Han and Du 2003) are susceptible.

Viruses in the Simbu-group occur endemically in large areas of Africa, Asia, Australia, and the Middle East (Charles 1994; Haughey et al 1988; St George and Kirkland 2004) and the related Cache Valley virus occurs in the United States of America (Edwards 1994; Edwards et al 1989).

The incubation period (infection to start of viraemia) for Akabane virus is from 1-6 days (St George 1998). In non-pregnant animals infection does not lead to the development of any signs (Gard et al 1989). Akabane virus crosses from maternal to foetal circulation in infected pregnant females and causes the development of malformed calves, particularly cases of arthrogryposis and hydroencephaly (Charles 1994; Parsonson et al 1977; Parsonson et al 1988; St George and Kirkland 2004). In cattle maximal damage occurs when infection takes place at about the 12th to 16th week of gestation (St George and Kirkland 2004). Once a foetus has become immuno-competent it can mount an immune response and damage is less apparent or does not occur. Infected calves are usually non viable (Charles 1994). Calves born or aborted will not be contagious and will not infect vectors.

Epidemics of foetal malformations due to Akabane virus have been reported in Japan and Australia (St George and Kirkland 2004). However, animals that have been exposed to the infection become immune and this leads to the establishment of a mainly immune population of cattle in endemic areas. For this reason foetal abnormalities usually occur sporadically in endemically infected areas but sero-conversion is common (Cybinski and St George 1978; Cybinski et al 1978; Fukutomi et al 2003; St George and Kirkland 2004). There are no reports of the disease having a significant economic impact in endemically infected countries, but prior to the disease becoming endemic outbreaks of foetal malformations could occur.

### **5.1.5 Hazard identification conclusion**

In view of the above, Akabane and other Simbu viruses are classified as potential hazards in the commodity.

## **5.2 RISK ASSESSMENT**

### **5.2.1 Entry assessment**

#### **5.2.1.1 Semen**

The virus was not excreted in the semen of eight artificially infected bulls (Parsonson et al 1981), whilst another expert has stated that there is “some evidence that the risk of transmission is low” (Eaglesome and Garcia 1997). After being inoculated with semen from naturally infected bulls, two of 16 sheep developed antibody to Akabane virus indicating that this virus was present in the bull’s semen (Gard et al 1989). Therefore it is considered that the likelihood that semen of viraemic animals may contain these viruses is non-negligible. The viraemic period for Akabane virus lasts for 3-4 days (St George and Kirkland 2004) and animals that have recovered from the infection are immune. Long term carriers of the virus have not been described. Since the viraemic period is short, the likelihood of collecting semen from a viraemic animal is considered to be low, but non-negligible.

#### **5.2.1.2 Embryos**

Simbu viruses have not been reported in embryos collected for transplantation. However, if the viruses can be transmitted in embryos they would have to be collected during the viraemic phase of the disease. The likelihood of collecting embryos during a period of viraemia is low but non-negligible. IETS has classified Akabane as a category 4 disease i.e. one for which preliminary work has been conducted or is in the progress” (IETS 2004). The likelihood of the disease being transmitted in embryos is therefore considered to be low, but non-negligible.

### 5.2.2 Exposure assessment

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

### 5.2.3 Consequence assessment

No description was found of infection of foetuses during the very early stages of pregnancy. A recipient of infected germplasm could become viraemic for 3-4 days although it would not be infectious. These viruses could only be transmitted to other animals in New Zealand by competent insect vectors. Annual surveys reported in the MAF publication *Surveillance* have demonstrated that *Culicoides* spp. are not present in New Zealand. A typical report shows that no *Culicoides* spp. were found in 15,000 insects trapped and that serological conversion to arboviruses did not occur in sentinel cattle (Motha et al 1997). Since *Culicoides* spp. are the main vectors of the disease it is unlikely that New Zealand cattle would be exposed to the virus. The virus has also been isolated from mosquitoes but no work has been done to investigate whether New Zealand mosquitoes are competent vectors. Furthermore, published surveys provide good evidence that New Zealand is free of arbovirus vectors (Motha et al 1997). In the absence of a competent vector in New Zealand, disease would be unable to establish if introduced.

The virus does not infect people and therefore there are no consequences for human health.

Antibodies to the virus have been found in a variety of African wildlife but disease has not been described in them (St George and Kirkland 2004). Marsupials are not susceptible (St George and Kirkland 2004). The disease has not been described in animals that occur as wild or feral species in New Zealand. Therefore, there would be no consequences for the environment, resulting from the introduction of infected germplasm.

The likelihood of the disease establishing and the consequences of establishment are considered to be negligible.

### 5.2.4 Risk estimation

Because the consequence assessment is negligible, the risk estimate for Akabane and other Simbu group viruses is negligible and they are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

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## **6 AUJESZKY'S DISEASE**

### **6.1 HAZARD IDENTIFICATION**

#### **6.1.1 Aetiological agent**

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

#### **6.1.2 OIE list**

Listed.

#### **6.1.3 New Zealand status**

Listed on the unwanted organisms register as an exotic, notifiable organism.

#### **6.1.4 Epidemiology**

Aujeszky's disease (pseudo-rabies) 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. Cattle do not transmit the virus to other animals and are considered to be dead-end hosts (Baker et al 1982; Henderson et al 1995; Herweijer and de Jonge 1977; Van Oirschot 2004). In animals other than pigs the disease is characterized by acute neurological signs and is invariably fatal (Baker et al 1982; Henderson et al 1995; Herweijer and de Jonge 1977; Van Oirschot 2004).

#### **6.1.5 Hazard identification conclusion**

In view of the above, Aujeszky's disease virus is classified as a potential hazard in the commodity.

### **6.2 RISK ASSESSMENT**

#### **6.2.1 Entry assessment**

Aujeszky's disease is a rare disease in cattle and only occurs when they have been in close contact with pigs. When it occurs the 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. Under these circumstances the likelihood that semen or embryos would be collected from infected donors is negligible and the likelihood of release is considered to be negligible.



## 6.2.2 Risk estimation

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

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## **7 BLUETONGUE**

### **7.1 HAZARD IDENTIFICATION**

#### **7.1.1 Aetiological agent**

Family: Reoviridae; Genus: Orbivirus. Bluetongue virus (BTV). There are 24 known serotypes of BTV.

#### **7.1.2 OIE list**

Listed.

#### **7.1.3 New Zealand status**

Listed on the unwanted organisms register as an exotic, notifiable organism.

#### **7.1.4 Epidemiology**

Bluetongue virus can infect many ruminant species. It occurs in most tropical and sub-tropical countries. It is absent in countries south of 34° south, including New Zealand, and countries north of 50° north (OIE 2006). The virus causes disease mainly in sheep, occasionally in goats, and rarely in cattle and deer. 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. In cattle infection is usually subclinical and mortality negligible but viraemic cattle can act as a source of infection for *Culicoides* spp. (Verwoerd and Erasmus 2004).

#### **7.1.5 Hazard identification conclusion**

In view of the above, bluetongue virus is classified as a potential hazard in the commodity.

### **7.2 RISK ASSESSMENT**

#### **7.2.1 Entry assessment**

##### **7.2.1.1 Semen**

Bluetongue virus can be excreted in bull's semen (Parsonson et al 1981). However, the virus is only excreted in semen while animals are viraemic (Bowen et al 1983; Howard et al 1985). The incubation period varies from 2-15 days following experimental infection, and is usually about 7 days in natural infections (Verwoerd and Erasmus 2004). Infected cattle remain viraemic for about 50 days (Verwoerd and Erasmus 2004). In countries where many strains of virus are endemic a few strains usually dominate in any one season

but as the population becomes immune to these strains the dominant strains are replaced by other strains 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 considered to be non-negligible.

#### **7.2.1.2 Embryos**

The International Embryo Transfer Society has classified bluetongue in cattle as belonging to Category 1. This category indicates that “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” (IETS 2004). Therefore the risk of transmission of bluetongue virus by properly prepared embryos is considered to be negligible.

#### **7.2.2 Exposure assessment**

Imported semen will be inseminated into susceptible New Zealand recipients. Therefore the likelihood of exposure to infected semen is considered to be non-negligible.

#### **7.2.3 Consequence assessment**

Cattle inseminated with infected semen became infected and developed viraemia (Bowen et al 1985; Schlafer et al 1990; Bowen and Howard 1984). Animals that have been inseminated may be viraemic for up to 50 days, but since bluetongue is not a contagious disease they will not transmit the disease to other ruminants. Although no reference could be found for iatrogenic transmission of BTV, mechanical transmission of this disease is thought unlikely to be of major significance in disease epizootics (Radostits et al 2007).

BTV is transmitted by *Culicoides* vectors. A *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al 1991), under which around 15,000 insects collected from light traps are examined annually (Motha et al 1997) and 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.

The OIE *Terrestrial Animal Health Code* states that countries that are south of 34° S and are not adjacent to a country not having an bluetongue virus free status may be considered free from bluetongue. Furthermore, the OIE *Terrestrial Animal Health 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 zones.” (OIE 2006).

Bluetongue is not a zoonotic disease and the virus does not constitute a threat to human health.

It is a disease of ruminants and there is no threat to indigenous animals or birds. Some species of deer are susceptible to the infection. The effect the virus might have on that is not known. However, since vectors for the virus do not occur the consequences for the environment, of introducing the virus, would be negligible.

Because New Zealand is free of *Culicoides* spp., the likelihood that the virus could establish in New Zealand is considered 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 consequence assessment is considered to be negligible.

#### 7.2.4 Risk estimation

Since the consequence assessment for the importation of semen is negligible, and the entry assessment for the importation of embryos is negligible, the risk estimate for BTV is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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## **8 BORNA DISEASE**

### **8.1 HAZARD IDENTIFICATION**

#### **8.1.1 Aetiological agent**

Family: Bornaviridae; Genus: Bornavirus. Borna disease virus is the only member of this family.

#### **8.1.2 OIE list**

Not listed.

#### **8.1.3 New Zealand status**

Listed on the unwanted organisms register as an exotic, unwanted organism.

#### **8.1.4 Epidemiology**

Borna disease is a disease of horses, sheep, and a variety of other animals including goats, deer, rabbits (Rott et al 2004), lynx (Desgiorgis et al 2000), and foxes (Dauphin et al 2001). Cattle can be subclinically infected (Hagiwara et al 1996). Disease is rare, but acute nervous disease can occur .

A closely related virus has been found in mallards and jackdaws in Sweden (Berg et al 2001) and a related virus has been identified as the aetiological agent of wobbly possum disease in New Zealand (O'Keefe et al 1997). In sheep and horses, it typically presents as a disease of the nervous system, but infection with the virus is most commonly subclinical (Rott et al 2004). Antibody to Borna disease virus has been found in humans suffering from psychosomatic disorders (Bode et al 1996; Rott et al 1985). However, the exact role of the virus in human infections and as a cause of psychosomatic disorders remains controversial. The specificity of demonstrated antibody and the accuracy and reliability of the PCR test to demonstrate the presence of viral RNA has been questioned, but the issues remain unresolved (Carbone 2001; Staeheli et al 2000).

The disease occurs most commonly in Germany and Switzerland. However, serologically positive animals have also been found in Poland, the Netherlands, Switzerland, Iran (Rott et al 2004), and Japan (Hagiwara et al 1997; Hagiwara et al 1996; Hagiwara et al 2002; Inoue et al 2002; Nakamura et al 1996; Nakamura et al 1995; Okamoto et al 2002) and Borna virus RNA has recently been found in France (Dauphin et al 2001; Dauphin and Zientara 2003). Reports on the demonstration of antibodies in horses have also come from North America (Kao et al 1993) and Israel (Teplitsky et al 2003). The virus has been demonstrated in cats in Britain (Reeves et al 1998). Several authors have suggested that the disease is an emerging disease and that many species of animals may be infected (Boucher et al 1999; Ludwig and Bode 2000).

The incubation period is thought to vary from 4 weeks to several months (Ludwig and Kao 1990). In mice the disease enters the body through the olfactory epithelium and migrates along nerve axons to the brain (Carbone et al 1987; Morales et al 1988; Sauder and Staeheli 2003). The virus can be experimentally transmitted to rats by inoculation into the footpads. However, neurectomy prevents the disease occurring thus demonstrating that transfer of the virus to the brain is by the intra-axonal route (Carbone et al 1987). It is excreted in nasal secretions, saliva and urine (Rott et al 2004; Vahlenkamp et al 2002). In an experimental situation the disease was transmitted from persistently infected rats to naïve rats via the olfactory route. This has led to the suggestion that rats could be a source of infection for farm animals (Sauder and Staeheli 2003). Vertical transmission has not been reported. Most infections are thought to be sub-clinical (Ludwig and Kao 1990) and the virus persists in carriers for at least 2 years, as demonstrated by the presence of viral RNA in peripheral mononuclear cells (Vahlenkamp et al 2002). Viral RNA has been demonstrated in the peripheral mononuclear cells of cattle (Hagiwara et al 1996), sheep (Hagiwara et al 1997; Vahlenkamp et al 2000; Vahlenkamp et al 2002), horses (Nakamura et al 1995; Vahlenkamp et al 2002), cats (Nakamura et al 1996; Reeves et al 1998), and humans (Kishi et al 1995; Vahlenkamp et al 2000). Natural transmission is presumed to occur via direct contact, fomites and food, inhalation, and ingestion (Rott et al 2004).

Despite the fact that Borna disease has been known for more than 250 years (Rott et al 2004), knowledge about the disease is still fragmentary and incomplete. The interpretation of the results of diagnostic tests is problematical. Although viral RNA has been demonstrated in an increasing number of countries and animals species, the occurrence of the disease is still mainly confined to parts of Germany and surrounding countries. Studies using RT-PCR have not generally been confirmed by viral isolation.

The disease is not regarded by OIE as a disease that is important to trade and it only occurs sporadically in countries where it does occur. However in Germany it is a notifiable disease and is controlled by a slaughter-out policy (Rott and Herzog 1994).

The rapidly increasing literature on the disease suggests that it may be an emerging disease. However, increased interest in the disease since it was suggested that it might be zoonotic, may account for some of the current interest.

#### **8.1.5 Hazard identification conclusion**

In view of the above, Borna disease is considered to be a potential hazard in the commodity.

## **8.2 RISK ASSESSMENT**

### **8.2.1 Entry assessment**

#### **8.2.1.1 Semen**

There is nothing in the literature that indicates that Borna disease is spread venereally. Nothing is known about the potential for the virus to contaminate semen or embryos. Much of the available information on the disease is based on studies in rats. In rats infected as adults the virus multiplies only in neurons. However, in rats infected as neonates the virus is found in all organs and these animals remain persistent shedders of virus. Virus can be shed in various body secretions including nasal secretions, faeces, and urine. It is not known to what extent the pathogenesis in cattle parallels that of rats. However, peripheral mononuclear cells of cattle have been found to contain viral RNA (Hagiwara et al 1996).

The likelihood that semen could be contaminated with infected mononuclear cells cannot be ignored since in some infections such as *Brucella ovis* infection in sheep large numbers of cells are found in the semen. Concomitant bacterial infections and Borna disease virus might therefore result in the shedding of virus in the semen. In addition contamination of semen by urine could introduce the virus.

Until definite information is available, the likelihood of the entry of virus in semen is considered to be low but non-negligible.

#### **8.2.1.2 Embryos**

No information was found about embryos derived from Borna disease infected animals. However, since the virus is excreted in urine, saliva, and nasal secretions, and viral RNA is found in mononuclear cells, a conservative approach has been taken in this risk analysis and the likelihood of entry in embryos is considered to be non-negligible.

### **8.2.2 Exposure assessment**

Imported embryos and semen would be inseminated/transplanted into susceptible recipients in New Zealand. Therefore the likelihood of exposure is considered to be high.

### **8.2.3 Consequence assessment**

It is assumed that the agent could be transmitted by insemination or transplantation of infected germplasm and that infected recipients of germplasm would be contagious and could infect animals in contact with them. Although most infections of cattle are not apparent, clinical cases of disease do occur (Ludwig and Kao 1990). Introduction of Borna disease virus could result in the establishment of a production-limiting disease in cattle that could be transmitted to sheep, horses, and other species (possibly including man).



The association between viral infection and the occurrence of psychosomatic diseases in humans (Bode et al 1996; Rott et al 1985) remains speculative. The consequences of introducing the virus for human health are therefore, uncertain, but are considered to be non-negligible.

The virus is known to infect a wide variety of animals (Dauphin and Zientara 2003; Desgiorgis et al 2000; Rott et al 2004) and birds (Berg et al 2001) and could therefore cause sporadic cases of disease in wild and feral animals and birds in New Zealand. In particular ostriches (Ashash et al 1996) have been infected with the virus and ratites (including kiwis) might therefore be susceptible. The presence of a similar virus in possums has not had any effect on the New Zealand environment apart from the rare occurrence of wobbly possum disease in possums. The effects on the environment are likely to be minimal but in view of the uncertainty, particularly regarding kiwis it should be regarded as non-negligible.

Since the introduction of the virus could lead to the establishment of a production limiting and possibly zoonotic disease and because the effects the virus could have on kiwis is not known, the consequences are considered to be non-negligible.

## **8.2.4 Risk estimation**

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate for Borna disease virus is considered to be non-negligible and it is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

## **8.3 RISK MANAGEMENT**

### **8.3.1 Options**

Since Borna disease is not listed by the OIE, no international standards for risk management exist.

Diagnostic methods available include virus isolation (Ludwig and Kao 1990; Rott et al 2004) and demonstration of virus proteins or RNA (Vahlenkamp et al 2002) in tissues. Serology has been used in epidemiological surveys but it is not a reliable indicator of infection in individual animals. Two of six animals that were confirmed as being infected with Borna disease at post mortem were negative in both the ELISA and indirect immunofluorescence test (Allmang et al 2001) and one was positive in the IFA but not ELISA. These findings indicate that infection does not always result in detectable antibody production (Muller-Doblies et al 2003). Positive serology is common in sheep (Muller-Doblies et al 2003) and the position in cattle could be similar. The most sensitive method for the isolation of virus is the intracerebral inoculation of rabbits which become ill within 4 weeks (Rott et al 2004). The virus can be isolated in embryonic

rabbit or rat brain cells. It could therefore be specified that aliquots of semen or embryos should be tested by one of these methods. RT-PCR tests are now widely used in research projects and surveys for the detection of viral RNA. These tests could be used to screen cattle for the presence of virus.

Importations could be restricted to countries where disease does not occur.

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

- Germplasm donors could be required to be resident since birth in countries where the disease has never been reported.
- Donors could be selected from herds with a greater than 5 year history of freedom from the disease in countries in which the disease is notifiable or in which reliable histories are available.
- Aliquots of semen and embryos from each collection batch of germplasm could be inoculated intracerebrally into rabbits or cultured on cell cultures derived from embryonic rabbit or rat brain with negative results.
- Peripheral cells from donors could be tested by PCR with negative results.

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## 9 BOVINE CALICIVIRUS INFECTION

### 9.1 HAZARD IDENTIFICATION

#### 9.1.1 Aetiological agent

Family: Caliciviridae; Genus: Norovirus, bovine enteric calicivirus and possibly other calici-like viruses.

#### 9.1.2 OIE list

Not listed.

#### 9.1.3 New Zealand status

Not reported to occur in New Zealand.

#### 9.1.4 Epidemiology

Two genotypes of the virus, the Jena and Newbury agents, occur in Europe (Knowles and Clarke 2004) and a third type has been described in the USA (Oliver et al 2003).

Despite identification of the viruses in calves nearly 40 years ago (Woode and Bridger 1978), the role of bovine enteric caliciviruses in calf diarrhoea is not well understood. Experimental infection of gnotobiotic calves and new born calves caused diarrhoea and intestinal pathology (Hall et al 1984). However, in naturally occurring cases of diarrhoea, calves are often infected with several viruses including rotaviruses and coronaviruses that are isolated in higher numbers than the caliciviruses (Knowles and Clarke 2004). Descriptions of diarrhoea associated with the virus are restricted to calves. Adult animals are apparently resistant or immune to infection.

The virus has been described in England (Knowles and Clarke 2004; Woode and Bridger 1978), Germany (Deng et al 2003), the Netherlands (van Der Poel et al 2000), and the USA (Smiley et al 2003). Investigations to identify virus or virus antibodies in countries where the virus is known to occur generally indicated a high prevalence of infection. In Germany, virus was identified in 8.9% of 381 diarrhoeal samples from cattle and antibody was found in 99.1% of 824 diarrhoeal samples (Deng et al 2003). In the USA, 72% of 75 calf faecal samples were positive in a RT-PCR assay (Smiley et al 2003). In the Netherlands, 44% of pooled faecal samples from 75 veal farms were found to be

positive in a RT-PCR assay, and it was suggested that calves may be a source of infection for humans. However, a recent study suggests that calf strains differ from human isolates and calves are unlikely to be a source of infection for humans (Oliver et al 2003). The virus has been known for almost 40 years but attracts little attention from diagnostic laboratories and research workers. This suggests that it is of minor economic importance.

It is not known whether the virus occurs in New Zealand. However, since it is widely distributed in the world and is a trivial pathogen for which active surveys have not been done, it is likely that the virus may already be present in New Zealand.

#### **9.1.5 Hazard identification conclusion**

Since bovine caliciviruses have been described as causing calf diarrhoea and have not been isolated in New Zealand they are regarded as potential hazards in the commodity.

### **9.2 RISK ASSESSMENT**

#### **9.2.1 Entry assessment**

The role played by caliciviruses in the aetiology of calf diarrhoea is uncertain. Post infection excretion of the virus in the faeces only lasts for a few days and the involvement of other organs has not been reported (Woode 1990). Viraemia has not been reported and all descriptions of the disease syndrome are restricted to calves. Under these circumstances the likelihood that adult animals would excrete virus in germplasm is considered to be negligible.

#### **9.2.2 Risk estimation**

Because the likelihood of entry is considered to be negligible the risk estimate for bovine calicivirus is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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## **10 BOVINE PARVOVIRUS INFECTION**

### **10.1 HAZARD IDENTIFICATION**

#### **10.1.1 Aetiological agent**

Family: Parvoviridae; Genus: Parvovirus, bovine parvovirus.

#### **10.1.2 OIE list**

Not listed.

#### **10.1.3 New Zealand status**

Unknown. Bovine parvovirus has not been identified in New Zealand although it is considered likely to be ubiquitous (Thomson 2004).

#### **10.1.4 Epidemiology**

Isolation of the virus has been reported from the USA (Barnes et al 1982), Canada (Sandals et al 1995), Australia (Durham et al 1985a), Germany (Elschner 1995), and Japan (Inaba et al 1973), and it is considered likely to be ubiquitous (Thomson 2004). The virus was isolated from low numbers of calves with and without diarrhoea (Elschner 1995). Durham found that on three infected farms, calves became infected and developed antibody soon after birth but on only one farm was this associated with an outbreak of post weaning diarrhoea (Durham et al 1985a). Experimental infection of calves led to mild to moderate diarrhoea (Durham et al 1985c) and concurrent subclinical coccidiosis infestation exacerbated the clinical signs (Durham et al 1985b). In 29 herds in Canada the overall seroprevalence was 82% in cattle and herd prevalence was 100% (Sandals et al 1995).

There is one report of the virus crossing the placental barrier and resulting in foetal death. Reports on clinical disease associated with the virus are rare and generally the literature is dated. Even experimental infections are generally mild and antibody occurs widely in clinically normal animals. Thompson has stated that there is uncertainty as to the pathogenic potential of the virus in cattle (Thomson 2004).



### 10.1.5 Hazard identification conclusion

It is concluded that the virus occurs commonly in healthy cattle and is of doubtful significance as a pathogen. It may occur ubiquitously and could be present in New Zealand since no surveys have been reported to identify the virus or antibody to it. Therefore, bovine parvovirus is not considered to be a potential hazard in the commodity.

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## **11 BOVINE RHINOVIRUS INFECTION**

### **11.1 HAZARD IDENTIFICATION**

#### **11.1.1 Aetiological agent**

Family: Picornviridae; Genus: Rhinovirus, serotypes 1-3.

#### **11.1.2 OIE list**

Not listed.

#### **11.1.3 New Zealand status**

Unknown. Bovine rhinovirus has not been identified in New Zealand although it is considered likely to be ubiquitous.

#### **11.1.4 Epidemiology**

Bovine rhinoviruses are commonly isolated from the nasal cavities of cattle (Sellers 1990). The presence of the virus has been reported in Germany, England, the USA, Japan, and Sudan (Sellers 1990; Thomson 2004) but its distribution is suspected to be world-wide. There are no reports of the virus being recovered from other species. A study of 1,590 cases of respiratory infection found no significant association between the presence of bovine rhinovirus and disease (Stott et al 1980). 48% of cattle in a study by Mohanty (1973) were seropositive for bovine rhinovirus (Thompson 2004).

Experimental infection causes rhinitis and signs of infection include fever, inappetance, lacrimation, conjunctivitis and nasal discharge (Sellers 1990; Thomson 2004). Although lower respiratory infections may occur it is unproven that the virus is the primary cause of such syndromes and mixed infections with other respiratory viruses may be involved in these cases (Sellers 1990; Thomson 2004).

#### **11.1.5 Hazard identification conclusion**

It is concluded that bovine rhinovirus occurs in both healthy cattle and those showing signs of respiratory disease. There is no evidence that it is significant primary pathogen, but may play a role in some respiratory infections in conjunction with other respiratory pathogens. It is likely that it occurs ubiquitously and since no surveys have been reported to identify the virus or antibody to it, it may be already present in New Zealand. There is no evidence to suggest that it is a cause of economically important disease. It is not known to be a zoonotic virus. Therefore, bovine rhinovirus is not considered to be a potential hazard in the commodity.

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## 12 BOVINE VIRAL DIARRHOEA VIRUS

### 12.1 HAZARD IDENTIFICATION

#### 12.1.1 Aetiological agent

Family: Flaviviridae; Genus: Pestivirus, genotypes BVDV1 and BVDV2. In each genotype both cytopathic and non-cytopathic biotypes occur.

#### 12.1.2 OIE list

Listed, although not covered by a chapter in the *Code*.

#### 12.1.3 New Zealand status

Bovine viral diarrhoea virus genotype 1 (BVDV1) is endemic in New Zealand but genotype 2 (BVDV2) is exotic.

#### 12.1.4 Epidemiology

BVDV has a world-wide distribution, but 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).

BVDV1 infection of non-pregnant cattle usually results in a mild infection typified by pyrexia and leukopenia from about 3-7 days. Viraemia and nasal excretion of the virus occur during this period (Brownlie 2005). The clinical signs are often so mild that they are not observed, or only mild signs and occasionally diarrhoea is seen (Potgieter 2004). Since it is widely distributed in most cattle herds, cattle are commonly infected before they become pregnant, resulting in a population of cattle that is usually immune and does 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 a 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 be 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 following an initial infection 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 characterized by severe leucopenia and haemorrhagic disease (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). Super infection of persistently infected animals with a cytopathic

BVDV strain results in the development of mucosal disease (Brownlie 2005; Drew 2004; Potgieter 2004). The cytopathic strain that re-infects the persistent carrier animals 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 terminates 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).

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

### **12.1.5 Hazard identification conclusion**

BVDV1 is endemic in New Zealand. However, BVDV2 virus is exotic and can cause severe disease. Therefore BVDV2 is considered to be a potential hazard in the commodity.

## **12.2 RISK ASSESSMENT**

### **12.2.1 Entry assessment**

#### **12.2.1.1 Semen**

BVDV may be excreted in the semen of persistently infected or acutely infected bulls (Kirkland et al 1991; Lindberg 2005). It also persisted for several months in the semen of bulls that were experimentally infected with the virus. Virus isolation methods detected the virus for 21 days after infection but PCR tests were positive for up to 7 months, which was the duration of the experiment. Virus was detected in the semen of one bull 5 months after infection by sub-inoculation of semen into a susceptible calf (Givens et al 2003). It therefore seems that virus may be excreted in semen for up to at least 5 months after infection despite the fact that the bulls have developed antibody to the virus. Persistent excretion of virus in semen has also been reported from a bull that was antibody positive and non-viraemic (Niskanen et al 2002; Voges et al 1998). These findings indicate that semen from BVDV infected bulls may contain BVDV for long periods of time despite being serologically positive. Therefore the likelihood of entry in semen is considered to be non-negligible.

#### **12.2.1.2 Embryos**

The position with regard to BVDV infection of embryos is uncertain. The Research Subcommittee of the International Embryo Transfer Society (Trachte et al 1998), Health and Safety Advisory Committee, has placed BVDV in Category 3 which consists of, “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”.

It has been shown that washing procedures removed BVDV from embryos (Singh et al 1982) and that recipients did not become infected (Bak et al 1992; Brock et al 1997; Smith and Grimmer 2000). In contrast other investigations showed that embryos exposed *in vitro* to BVDV could not be consistently freed from the virus by washing or washing and trypsin treatment (Bielanski and Jordan 1996; Trachte et al 1998; Waldrop et al 2004a). 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 from *in vivo* derived embryos by the usual washing procedures (Waldrop et al 2004b). There was also strong circumstantial evidence that a BVDV2 strain of virus was introduced into Britain with an embryo imported from America (Smith and Grimmer 2000) and that reproductive problems and seroconversion to BVDV in a recipient herd in Sweden were caused by imported embryos (Lindberg 2005).

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

It is therefore concluded that the likelihood that embryos could be infected with BVDV is non-negligible.

### **12.2.2 Exposure assessment**

Imported embryos or semen would be inseminated or transferred into recipient females and therefore the likelihood of exposure of the recipients is very high and for the purposes of this risk analysis is considered to be non-negligible.

### **12.2.3 Consequence assessment**

BVDV2 is exotic to New Zealand and, if introduced, it would be expected to spread amongst susceptible cattle and even those immune to BVDV1 would not be protected. 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.

BVDV1 is known to infect deer and goats (Horner 2000). Antibody to the virus is known to develop in these species but disease has not been described. The consequences for these species are therefore considered to be negligible. The likelihood that there would be any other consequences for the environment is considered to be negligible.

The consequences for cattle are considered to be non-negligible. The consequences for the environment and human health are considered to be negligible.

#### **12.2.4 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 hazard in the commodity. Therefore, risk management measures can be justified.

### **12.3 RISK MANAGEMENT**

#### **12.3.1 Options**

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 (Drew 2004). 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 semen of bovines (Anonymous 2003) and the recommendations of the OIE *Code* (OIE 2006). 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 that they have produced since their last negative serological test, tested for BVDV. Some animals that had developed antibody titres continued to excrete the virus for at least 5 months after experimental infection (Givens et al 2003). Therefore it could be required that every batch of semen imported into New Zealand, regardless of the serological status of the donor should be tested by virus isolation or by RT-PCR for the presence of BVD virus or RNA. Embryo donors could be tested serologically and for viraemia. In the case of serologically negative donors they could be tested both before and after embryo collection to ensure that they were not infected during or shortly before the period of embryo collection. In addition all non-fertilized, degenerated and zona pellucida compromised embryos, collection fluid, and washing fluid could be collected (OIE 2006) and tested by virus isolation or RT-PCR for virus or viral RNA.

Since bovine foetal calf serum and other serum products may be contaminated with BVDV it could be specified that all products used in processing germplasm should be shown to be free from BVDV (see section 42).

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

- Donor bulls could be housed on a semen collection centre where bulls are maintained and tested as specified in appendix 3.2.1 of the *Code*.
- A straw from each batch of imported semen could be tested by virus isolation or RT-PCR for BVDV2 with negative results.
- Potential embryo donors could be tested serologically (by ELISA) and by RT-PCR before being placed in quarantine. In that case, any animals that are serologically negative and viraemic (PCR positive) would be considered unsuitable for use as donors. Those that are serologically positive and PCR negative would be considered suitable for use as donors. Animals that are serologically negative and non-viraemic could be held in quarantine for at least 3 weeks prior to embryo collection. After 3 weeks in quarantine donors could again be tested by a serological test (ELISA) and by RT-PCR. In that case, 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.
- All non-fertilized, 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 collected and tested by virus isolation or RT-PCR to demonstrate freedom from BVDV-2.

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## 13 CRIMEAN CONGO HAEMORRHAGIC FEVER

### 13.1 HAZARD IDENTIFICATION

#### 13.1.1 Aetiological agent

Family: Bunyaviridae; Genus: Nairovirus, Crimean Congo haemorrhagic fever virus (CCHFV).

#### 13.1.2 OIE list

Listed.

#### 13.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic unwanted organism.

#### 13.1.4 Epidemiology

CCHFV occurs in Africa, Asia, the Middle East, and Eastern Europe (Swanepoel and Burt 2004). The virus infects humans and a wide variety of ruminants and other smaller animals such as hares; it can also infect ostriches (Swanepoel and Burt 2004). Serological methods, including ELISA, can be used to detect antibody against CCHFV (Burt et al 1993; Qing et al 2003) 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 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 occurred in a South African hospital (Shepherd et al 1985). The virus has been isolated from at least 30 species of ixodid ticks (Swanepoel and Burt 2004) but not from argasid ticks (Durden et al 1993). 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).

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

incubation period in cattle will be up to 10 days. The viraemic period lasts for up to 7 days in ruminants and other animals (Swanepoel and Burt 2004). There are no descriptions of long term carriers.

#### **13.1.5 Hazard identification conclusion**

CCHFV causes a serious disease in humans. As it is not present in New Zealand but may be carried by infected cattle, it is classified as a potential hazard in the commodity.

### **13.2 RISK ASSESSMENT**

#### **13.2.1 Entry assessment**

No information was found on the transmission of the virus in semen or embryos. Since viraemia occurs for a period of around 7 days (Swanepoel and Burt 2004) it is assumed that germplasm collected during viraemia could be infected. The likelihood of collecting germplasm during a viraemic episode is considered to be low but non-negligible.

#### **13.2.2 Exposure assessment**

Any imported semen or embryos would be inseminated or implanted into susceptible New Zealand recipients. Therefore the likelihood of exposure is considered to be high.

#### **13.2.3 Consequence assessment**

If the disease were to become established in New Zealand it would have negligible effects on the livestock industries since infections in animals are invariably subclinical.

Transmission of the virus by insemination or implantation of germplasm has not been described. However, it is assumed that insemination or implantation of infected semen or embryos into susceptible New Zealand recipients would result in infection. Infection of cattle would not cause any signs of disease but the infected recipients of the germplasm would become viraemic for a short period. During the period of viraemia the animals would not be contagious but could infect competent vectors. At least 30 species of ixodid ticks have been found to carry the virus but the known distribution of the disease mirrors the distribution of *Hyalomma* spp. ticks (Swanepoel et al 1987). Therefore, the maintenance of the disease must depend on a cycle between mammalian hosts and *Hyalomma* spp. The New Zealand cattle tick *Haemaphysalis longicornis* is listed as being able to transmit the virus (Heath 2002). In addition, it is considered that there is a very low likelihood that a recipient of germplasm would be infested by a cattle tick while viraemic after insemination or transplantation. Overall, the establishment of the disease in New Zealand is therefore considered to be unlikely.

If the New Zealand cattle tick can act as a vector of the virus, establishment of the disease in New Zealand could lead to the rare occurrence of a serious and sometimes fatal disease in humans.

The virus might cause subclinical infections in feral ruminants and small mammals.

In conclusion, CCHFV would be unlikely to establish in New Zealand, and if it did there would be a negligible effect on the livestock farming industries or feral or wild animal populations. However, humans are susceptible to the virus and the possible effects on human health would be non-negligible.

#### **13.2.4 Risk estimation**

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

### **13.3 RISK MANAGEMENT**

#### **13.3.1 Option evaluation**

As there is no *Code* chapter for CCHFV, there are no international risk management standards for any commodity.

The disease has a short incubation period and long-term carriers do not occur. Therefore, quarantine of tick free cattle in tick free premises would be effective in preventing collection of infected germplasm. A quarantine period of 21 days would be adequate as the incubation period is 3-9 days (Swanepoel and Burt 2004) and the period of viraemia lasts about 7 days (Gonzalez et al 1998). Another option would be to test donor animals serologically before and at a suitable time interval after germplasm collection to ensure that they did not become infected during the period of semen collection.

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

- Donors could be required to have been resident for at least the 21 days before germplasm collection in a country or zone that is free from the disease.
- Donors could be scrupulously treated with a suitable acaricide and inspected to ensure that they are free from ticks and placed in isolation in tick-free germplasm collection premises. They could be kept in quarantine for a minimum of 3 weeks immediately before the start of and then during semen or embryo collection and regularly inspected and maintained in a tick-free state throughout the period of quarantine.
- Donors could be serologically tested within the 7 days prior to the start of germplasm collection and 3-8 weeks after germplasm collection is completed. Germplasm collected from animals that were serologically positive at the first test and did not have a rising titre at the second test could be considered suitable for

export. Germplasm from animals that are negative at both tests could be considered suitable for export. Germplasm from animals that sero-convert or have rising titres between the two tests could be disqualified from use as donors of germplasm for export to New Zealand.

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## **14 FOOT AND MOUTH DISEASE**

### **14.1 HAZARD IDENTIFICATION**

#### **14.1.1 Aetiological agent**

Family: Picornaviridae; Genus Aphthovirus, foot and mouth disease virus (FMDV). There are seven serotypes of the virus: O, A, C, SAT 1, SAT 2, SAT 3, and Asia 1.

#### **14.1.2 OIE list**

Listed.

#### **14.1.3 New Zealand status**

Listed on the unwanted organisms register as an exotic notifiable disease.

#### **14.1.4 Epidemiology**

Extensive reviews on FMDV are available (Sanson 1994; Thomson and Bastos 2004) and much of the information given below is taken from these. The disease is the most contagious and economically devastating known animal disease. It can infect all cloven hoofed animals. 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. Infected animals excrete the virus in saliva, faeces, urine, milk, semen, and in ocular and nasal discharges (Sanson 1994; Thomson and Bastos 2004). 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 aerosolized 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 by persistent carriers is low and the ability of persistently infected cattle to spread the disease is controversial (Thomson and Bastos 2004).

#### **14.1.5 Hazard identification conclusion**

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



## 14.2 RISK ASSESSMENT

### 14.2.1 Entry assessment

#### 14.2.1.1 Semen

The virus 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 infected semen (Callis 1996). The risk of release of virus in semen is considered to be non-negligible.

#### 14.2.1.2 Embryos

Foot and mouth disease of cattle is classified by IETS as a Category 1, i.e. 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” (IETS 2004). 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; Singh et al 1986). The likelihood that embryos from infected cattle would be contaminated with FMDV could therefore, reasonably be considered to be negligible if embryos are properly handled.

However, there is still a cause for concern since virus 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 correctly administered, virus could potentially contaminate *in vivo* derived embryos. In view of this doubt and because the disease is the most economically damaging disease known, a more conservative approach may be appropriate. Therefore the likelihood of release is considered to be non-negligible.

### 14.2.2 Exposure assessment

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

### 14.2.3 Consequence assessment

Infected semen or embryos would be likely to result in infection of the recipient (Callis 1996). The infected animals would develop disease and would become highly contagious and likely to infect any cloven hoofed animals they came in contact with or possibly transmit infection by aerosol to animals several kilometers from them. In extreme cases, particularly if large populations of pigs have been infected, the virus could spread by airborne infection over hundreds of kilometers (Gloster et al 1982).

Animals that become infected could become the focal point for a serious outbreak of foot and mouth disease. An outbreak of the disease 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 dramatically demonstrated by the losses that resulted from an outbreak of the disease in Britain where the costs to government were estimated at 3.1 billion pounds (Thompson et al 2002).

Foot and mouth disease 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 could constitute an ongoing source of infection for domestic stock.

Introduction of the disease could have extremely severe effects on individual farmers and the economy of the country. The consequences are considered to be non-negligible

#### **14.2.4 Risk estimation**

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

### **14.3 RISK MANAGEMENT**

#### **14.3.1 Options**

The following options could be considered to effectively manage the risk associated with FMDV.

- It is possible to continue with 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 was 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 OIE *Terrestrial Animal Health Code* gives conditions under which semen can be imported from infected countries into foot-and-mouth disease free countries. These conditions include the stipulation that animals are kept on foot and mouth disease free premises in an area where no foot and mouth disease has occurred within a radius of 10 kilometers for the 30 days before collection. Also unvaccinated animals could be tested for antibody not less than 21 days after collection of semen. Alternatively animals could have been vaccinated within 12 months prior to collection. These OIE recommendations for semen could be followed.

- Article 2.2.10.16 of the OIE *Terrestrial Animal Health Code* (OIE 2006) states that, irrespective of the FMD status of the exporting country, Veterinary Administrations 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 appendix 3.3.1 or appendix 3.3.3, as relevant. These OIE recommendations for embryos could be followed.
- The safety of embryo transfer procedures is fully reliant on the technical and ethical excellence of the individual in charge (Thibier 2006; Suttmoller and Wrathall 1997). Furthermore, the number of embryos used to determine the IETS classification of this disease was limited and the IETS Import/Export Committee did not define precisely what was meant by the term ‘negligible risk’ as referred to in 14.2.1.2 above (Suttmoller and Wrathall 1997). Therefore, in view of the extreme seriousness of the disease and the catastrophic consequences that could result from the introduction of FMDV, it could be considered that the OIE recommendations for bovine germplasm are not sufficient to provide the appropriate level of protection against this hazard and importation of germplasm from countries that are infected with foot-and-mouth disease could be prohibited.

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## 15 BOVINE HERPES VIRUSES

### 15.1 HAZARD IDENTIFICATION

#### 15.1.1 Aetiological agents

Family: Herpesviridae; Subfamily: Alphaherpesvirinae; Genus: Varicellovirus, bovine herpesvirus 1 (D'Arce et al 2002) is associated with infectious bovine rhinotracheitis (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. Strains formerly described as IBRV 1.3 that are associated with encephalitis are now classified as BHV5. 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 1995a).

**Table 2. Bovine herpesviruses**

Type	Syndrome			
	IBR	IPV/IPB	Abortion	Encephalitis
BHV1.1	+	-	+	-
BHV1.2a	+	+	+	-
BHV1.2b	+	+	-	-
BHV5	-	-	-	+

#### 15.1.2 OIE list

Infectious bovine rhinotracheitis and infectious pustular vulvovaginitis are listed by the OIE.

#### 15.1.3 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). However, at the present time identification of abortifacient strains of the virus from either subtype 1 or 2 strains would require experimental infection of pregnant cows. A more pragmatic approach is to regard BHV1.1 and BHV 1.2a as exotic organisms. Abortifacient strains are classified on the unwanted organisms register as unwanted notifiable organisms.

#### **15.1.4 Epidemiology**

IBR/IPV has a world-wide distribution. Only BHV1.2b and BHV5 occur in Australia. The virus is endemic in New Zealand and serological surveys have shown that it occurs very widely (Neilson and Grace 1988). Both the IBR and the IPV syndrome have been described in New Zealand (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 occasionally occur (van Oirschot 2004). Virus spreads to the conjunctiva and trigeminal ganglion by neuronal axonal transport (van Oirschot 2004). Many animals become chronically infected latent carriers of the virus in their trigeminal or sacral ganglia, and may excrete the virus periodically when they are stressed (Babuik et al 2004; van Oirschot 2004). Sub-clinically infected bulls may excrete virus in their semen (Babuik et al 2004).

BHV5 associated with encephalitis (Wentink et al 1993) has been described in Australia (Brake and Studdert 1985) but not in New Zealand.

#### **15.1.5 Hazard identification conclusion**

Abortifacient strains of IBR/IPV virus are exotic notifiable organisms and they are commonly present in carrier animals. These organisms are therefore classified as potential hazards. However, since practical tests are not available to identify abortifacient strains in the laboratory it is necessary to regard all BHV1.1 and BHV1.2a strains as potential hazards. BHV5 is also exotic and is regarded as a potential hazard.

### **15.2 RISK ASSESSMENT**

#### **15.2.1 Entry assessment**

##### **15.2.1.1 Semen**

Subclinically infected bulls may excrete the virus in their semen (Babuik et al 2004). There are many reports confirming that bulls can shed BHV1 in their semen (de Gee et al 1996; Smits et al 2000; van Oirschot 1995a; van Oirschot et al 1993). Excretion of BHV5 in bull semen has also been reported (D'Arce et al 2002; Gomes et al 2003). The likelihood of entry in semen is therefore considered to be non-negligible.

##### **15.2.1.2 Embryos**

IBR virus adheres to zona pellucida of intact embryos but is removed by trypsin treatment and washing (Stringfellow et al 1990; Wrathall et al 2006). 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” (IETS 2004). The IETS classification is dependant on the requirement that the embryos are trypsin treated. Therefore it is concluded that provided the embryos are trypsin treated according to the IETS recommendations the likelihood of transmission of BHV1 by embryo transfer is considered to be negligible. It is assumed that BHV5 would behave in a similar manner to BHV1.

### **15.2.2 Exposure assessment**

The likelihood that imported semen will be inseminated into recipients is very high. Therefore the likelihood of exposure is considered to be non-negligible.

### **15.2.3 Consequence assessment**

Semen infected with IBR/ IPV virus is infectious for susceptible recipients (Parsonson and Snowdon 1975; 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 1995b) and the likelihood that insemination will result in infection of the recipients is high. If imported semen is infected with a BHV1.1 or BHV1.2a strain, the likelihood that a new strain would be introduced into New Zealand that could cause abortion outbreaks is non-negligible. This would have economical consequences for affected cattle herds. Similarly, if BHV5 is introduced the virus could become established in New Zealand and sporadic cases of encephalitis may occur.

The virus does not infect humans and therefore the consequences for people are negligible.

Other ruminants can possibly be infected with BHVs since they may have antibody to the virus. However, the antibody may be cross-reacting antibody as in the case of deer infected with cervine herpesvirus (Motha and Jenner 2001; Tisdall and Rowe 2001). No significant disease has been described in other ruminants. The consequences for the environment are therefore assessed to be negligible.

Since abortifacient strains of BHV1 and BHV5 can be excreted in semen, the likelihood that importation of semen could cause significant consequences is non-negligible.

### **15.2.4 Risk estimate**

Since entry, exposure, and consequence assessments for exotic bovine herpesviruses in imported semen are all non-negligible, the risk estimate is non-negligible, and they are classified as a hazard. Therefore risk management measures can be justified for imported bovine semen.

The entry assessment for exotic bovine herpesviruses in imported embryos was considered to be negligible, so the risk estimate is non-negligible, and they are not classified as a hazard. Therefore, risk management measures are not justified for imported bovine embryos..

### 15.3 RISK MANAGEMENT

#### 15.3.1 Options

The OIE *Terrestrial Animal Health Code* does not discuss strains of bovine herpes viruses but instead considers the clinical syndromes of IBR and IPV. There are, therefore, no international risk management standards that are directly applicable although it is reasonable to extrapolate from the *Code* to the exotic strains of concern here. The *Code* recommends that semen should be taken from bulls kept in IBR/IPV free herds. Under paragraph 2a of article 2.3.5.3 of the *Code*, maintenance of herd freedom requires repeated blood testing of individuals at a maximum interval of 12 months together with (paragraph 2d) blood testing of all cattle that abort after more than 3 months gestation. However, subclinical IBR breakdowns of high health status herds and AI studs have been reported where no source of infection has been identified (Pritchard et al 2003; van Oirschot et al 1993).

The *Code* also recommends that the donor should be kept isolated during and for at least 30 days after semen collection and be tested serologically with negative results at least 21 days after semen collection. It can be assumed that animals infected with BHV5 will cross react in serological tests for BHV1 (Jianning Wang personal communication). However, 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), demonstrating that seronegative status does not eliminate the risk of virus transmission through semen.

Semen could also be subjected to a virus isolation test (OIE, 2006). Although the PCR has been reported to be more sensitive for virus detection than the virus isolation test (Smits et al 2000) and to yield quick results (de Gee et al 1996) a test has not yet been validated for international trade (de Gee et al 1996; van Oirschot 2004).

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

- Each batch of semen could be subjected to a virus isolation test with a requirement for negative results. When a suitable validated PCR test is available for both BHV1 and BHV5, it could replace the virus isolation test.
- Donor animals could come from herds or artificial breeding centres that are maintained free from IBR according to OIE recommended criteria.



- Donor animals could be isolated for at least 30 days after semen collection and tested by a validated serological test with negative results at least 21 days after semen collection.

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## 16 IBARAKI DISEASE

### 16.1 HAZARD IDENTIFICATION

#### 16.1.1 Aetiological agent

Family: Reoviridae; Genus: Orbivirus. Ibaraki virus belongs to the epizootic haemorrhagic disease (EHD) serogroup and is classified as belonging to serogroup 7 but may be re-classified as a serogroup 2 virus (Kitano 2004).

#### 16.1.2 OIE list

Not listed.

#### 16.1.3 New Zealand status

Classified on the unwanted organisms register as an “other exotic organism”.

#### 16.1.4 Epidemiology

Ibaraki disease sporadically affects cattle in Japan. The first outbreak in 1959 affected over 30,000 cattle with 4,000 mortalities. Further outbreaks occurred in 1982 and 1987 (Kitano 2004). In 1997 and 1998 limited outbreaks occurred (Uchinuno et al 2000). In 1982, 73 cases occurred in Korea (Bak et al 1983). There was one isolation of the virus from the brain of a calf with encephalitis in Taiwan (Liao et al 1996). The disease has not occurred elsewhere, although antibody to the virus has been found in several other countries (Kitano 2004).

The disease syndrome is characterized by an inability to swallow caused by degeneration of the muscles of the pharynx, larynx, oesophagus, and tongue. In most cases animals recover in a few days. In more severe cases the skin of the mouth and oral mucous membranes may become congested and necrotic and up to 40% of these severely affected cattle may die or are slaughtered (Kitano 2004). In the 1997 outbreak numerous abortions and stillbirths also occurred. The incubation period is 4-11 days (Kitano 2004). Virus can be isolated from blood or lymph nodes (Kitano 2004; Ohashi et al 1999). Reports describing persistence of viraemia were not found but since bluetongue virus persists for up to 2 months (Verwoerd and Erasmus 2004) it is likely that Ibaraki virus would persist for a similar period.

The disease occurs in late summer and autumn when biting insects occur in large numbers and the virus has been isolated from *Culicoides* spp. (Ohashi et al 1999).

### **16.1.5 Hazard identification conclusion**

Ibaraki virus is exotic and could cause mortality and morbidity in susceptible cattle. Therefore it is classified as a potential hazard in the commodity.

## **16.2 RISK ASSESSMENT**

### **16.2.1 Entry assessment**

No information was found about the transmission of the virus in germplasm. Therefore, it is assumed that the virus would behave in a similar manner to bluetongue virus and EHDV. It is therefore assumed that the disease could be transmitted in semen while animals are viraemic and that it will not be transmitted by properly washed and prepared embryos. However, the disease only occurs in sporadically and the likelihood that semen would contain virus is negligible except in years where there is an active outbreak of the disease in the area in which the donors are resident. Therefore the likelihood that semen will contain virus is considered to be low but non-negligible. Since the correctly prepared embryos do not transmit bluetongue it is likely that Ibaraki disease would also not be transmitted by embryos. However, since evidence is lacking it is assumed that the likelihood that embryos could transmit the virus is non-negligible.

### **16.2.2 Exposure assessment**

Since imported germplasm will be inseminated or transferred into susceptible recipients the likelihood of exposure is therefore considered to be non-negligible.

### **16.2.3 Consequence assessment**

No literature is available to indicate if insemination or embryo transfer with infected germplasm would infect recipients. However, extrapolation from bluetongue studies suggests that infection of recipients is likely in the case of semen and unlikely in the case of embryos.

Infection of recipients would be likely to lead to the presence of viraemic but not infectious animals and cases of clinical disease, but because there are no *Culicoides* in New Zealand the disease could not establish here.

The disease is not known to be zoonotic and therefore introduction of the virus would have negligible consequences for human health.

The disease has not been described in animals other than cattle and therefore consequences for the environment for the environment would be negligible.

Since the disease could not establish in New Zealand, the consequences are considered to be negligible.

#### 16.2.4 Risk estimation

Since the consequences of introducing the virus are considered to be negligible, the risk estimate for Ibaraki virus is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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## 17 JEMBRANA DISEASE

### 17.1 HAZARD IDENTIFICATION

#### 17.1.1 Aetiological agent

Family: Retroviridae; Genus: lentivirus, Jembrana disease virus.

#### 17.1.2 OIE list

Not listed.

#### 17.1.3 New Zealand status

Not listed on the unwanted organisms register.

#### 17.1.4 Epidemiology

Jembrana disease is a serious disease of Bali cattle (*Bos javanicus*) (Hartaningsih et al 2001; Hartaningsih et al 1993; Hartaningsih et al 1994) that occurs only in Indonesia. Bali cattle are the domesticated form of the wild banteng cattle. Ongole cattle (Soeharsono et al 1995), Friesian cattle, and Asian buffalo (*Bubalus bubalis*) have been experimentally infected. They became viraemic and developed antibody, but developed only very mild signs of disease and did not show overt clinical signs (Soeharsono et al 1990; Soeharsono et al 1995; Wilcox 1997).

The disease was first diagnosed in 1964 on the island of Bali. Within a year, 26,000 Bali cattle of a population of 300,000 died (Wilcox 1997). The aetiological agent has been identified as a lentivirus (Chadwick et al 1998; Wilcox et al 1992). The disease apparently spreads by contact and through mechanical transmission by biting flies (Wilcox 1997). Movement of Bali cattle has led to a limited spread of the disease to neighbouring islands (Hartaningsih et al 1993; Wilcox 1997). The incubation period is 4.5-12 days (Soeharsono et al 1990). Clinically, the disease is characterised by fever and lymphomegaly. During the febrile period there may be around  $10^8$  virus particles per ml of blood. Later in the course of the infection the number of virus particles in the blood drops to about 10 infectious doses per ml and this low level of viraemia is believed to be insufficient for mechanical transmission by blood sucking insects (Wilcox 1997). Bali cattle remain viraemic for at least 2 years. In other cattle following experimental infection signs of disease are mild and viraemia only persists for 3 months, and in buffalo virus persisted in the spleen for 9 months (Soeharsono et al 1990; Soeharsono et al 1995). The development of antibody to the virus is delayed for up to 11 weeks or longer but then persists for at least a year (Hartaningsih et al 1994).

### 17.1.5 Hazard identification conclusion

The disease has remained confined to Bali and a few surrounding islands of Indonesia. It has only occurred as an overt disease in Bali cattle (*Bos janvanica*). Since Bali cattle do not occur in New Zealand they could only be imported as a new species requiring approval from ERMA and the development of a risk analysis and import health standard. The virus could not be introduced by importation of germplasm from other cattle (*Bos indicus* or *Bos taurus*). Therefore the agent is not considered to be a potential hazard in the commodity.

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## 18 LUMPY SKIN DISEASE

### 18.1 HAZARD IDENTIFICATION

#### 18.1.1 Aetiological agent

Family: Poxviridae; Genus: Capripox, lumpy skin disease virus.

#### 18.1.2 OIE list

Listed.

#### 18.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic, notifiable organism.

#### 18.1.4 Epidemiology

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). Although very closely related it is probable that sheep and goat pox virus does not occur naturally in cattle and similarly lumpy skin disease virus does not transmit naturally to sheep and goats (Kitching and Carn 2004).

The disease has generally remained confined to Africa, although outbreaks occurred in Israel (Yeruham et al 1995) and possibly in Saudi Arabia in oryx (Arnaud et al 1992; Greth et al 1992). In the latter case there could be some doubt as to whether the causal agent was a sheep and goat pox virus. 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 (for review see Coetzer 2004). However, Chihota transmitted the disease mechanically with *Aedes aegypti* (Chihota et al 2001) but failed to transmit it with other potential vectors (Chihota et al 2003). Isolation from *Stomoxys calcitrans* and *Musca confiscata* has occurred but attempts to transmit the disease with these vectors were not successful (Coetzer 2004). Transmission of the disease by intradermal infection is inefficient but intravenous infection caused typical disease. Spread by contact did not occur (Carn and Kitching 1995b). Early work indicated that the disease can spread by very close contact such as sharing of drinking troughs but this is rare (Coetzer 2004).

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 between 1% and 20% and mortality was less than 10% (Bruckner according to (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 (Carn and Kitching 1995a). For the purposes of the OIE *Code* the incubation period is 28 days (OIE 2006). 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 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 saliva for 11 days, in semen for 22 days, and in skin lesions for 33 days (Weiss 1968 according to Coetzer 2004). In experimental infections viraemia persisted for 4 days (Coetzer 2004). Viral DNA has been demonstrated by PCR in semen up to 159 days and virus has been isolated from semen for up to 42 days after experimental infection (Irons et al 2005).

Interpretation of serological tests is problematical because some animals only develop low titres after infection (Coetzer 2004; Kitching and Carn 2004).

Immunity in recovered cattle is lifelong (Coetzer 2004) and long term carriers have not been described.

#### **18.1.5 Hazard identification conclusion**

Lumpy skin disease is an OIE listed disease that is exotic to New Zealand. Therefore it is classified as a potential hazard in the commodity.

### **18.2 RISK ASSESSMENT**

#### **18.2.1 Entry assessment**

##### **18.2.1.1 Semen**

Lumpy skin disease virus was isolated from the semen for 42 days and viral DNA demonstrated in semen for 159 days 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 bulls. Therefore the likelihood that the virus will contaminate imported semen is considered to be low but non-negligible.

##### **18.2.1.2 Embryos**

Nothing is known about the transmission of the disease by embryo transplantation. However, since lumpy skin disease is an economically important disease a conservative stance is justified and in this risk analysis it is considered that the likelihood of transmission by embryo transfer is non-negligible.

### **18.2.2 Exposure assessment**

Since germplasm is likely to be inseminated or transferred into susceptible recipients the likelihood of exposure is high and for the purposes of this risk analysis is considered to be non-negligible.

### **18.2.3 Consequence assessment**

Lumpy skin disease is known to be present in semen and is likely to be transmitted by insemination of infected semen (Hare 1985). There is no information concerning the transmission of the virus by embryo transfer. For the purposes of this risk analysis it is assumed that both insemination and embryo transfer could result in infection of recipients. Infected recipients would develop clinically overt disease. Since the disease has never spread beyond Africa and the Middle East the likelihood that competent vectors of the disease would be present in New Zealand is very low. The likelihood of the disease establishing in New Zealand is therefore extremely low. However, a significant number of our trading partners require that, as a condition of continuing trade in live 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 have been negotiated continued trade in live animals would not be possible, with some countries.

The disease is not a zoonotic disease and introduction of the virus would have no consequences for human health.

There are no reports of infection of sheep, goats, or deer. 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 antelope play a significant role in the epidemiology of the disease (Hedger and Hamblin 1983). There are no reports of infection of deer. Therefore the introduction of the virus would not have any consequences for the New Zealand environment.

Since there may be negative impacts on the trade in live animals and germplasm, the consequences of the introduction of the virus are considered to be non-negligible.

### **18.2.4 Risk estimation**

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate for lumpy skin disease virus is non-negligible and it is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

## 18.3 RISK MANAGEMENT

### 18.3.1 Options

OIE recommendations for the importation of semen from infected countries are that donors should be kept in an establishment or artificial insemination centre in which 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 (OIE 2006). However, it has been shown that lumpy skin disease virus may be present in semen for at least 42 days after infection and that viral DNA may be present for at least 159 days (Irons et al 2005). Therefore the OIE recommendations may be considered inadequate.

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.

There are no OIE recommendations regarding embryos. The same conditions that apply to semen donors could also be applied to embryo donors.

In the case of countries that are free from lumpy skin disease OIE recommends that the animals should have shown no signs of lumpy skin disease on the day of collection of semen or for the following 28 days and that the animals should be kept in a lumpy skin disease free country. These conditions could be applied to donors of both semen and embryos for importation into New Zealand.

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

- Donors could be required to be resident for the 6 months prior to germplasm collection in a country or zone that is free from lumpy skin disease according to the OIE definition of freedom (OIE 2006).
- In countries where lumpy skin disease occurs 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 animal could be required to be free from any sign of lumpy skin disease for at least 28 days after completion of germplasm collection.
- 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 lumpy skin disease virus DNA.

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## 19 MALIGNANT CATARRHAL FEVER

### 19.1 HAZARD IDENTIFICATION

#### 19.1.1 Aetiological agent

Family: Herpesviridae; Genus: Herpesvirus, alcelaphine herpesvirus 1 (AHV-1) and ovine herpesvirus 2 (OHV-2).

#### 19.1.2 OIE list

Malignant catarrhal fever (AHV-1 only) is an OIE listed disease.

#### 19.1.3 New Zealand status

AHV-1 is considered to be exotic, whilst OHV-2 is endemic in New Zealand.

#### 19.1.4 Epidemiology

AHV-1 is carried subclinically by three species of wildebeest; blue wildebeest (*Connochaetes gnou*), black wildebeest (*Connochaetes taurinus taurinus*), and white bearded wildebeest (*Connochaetes albojubatus*). The disease occurs in cattle closely associated with wildebeest, especially when calving. The disease is usually fatal but mild cases that are followed by recovery do sometimes occur (Reid and Van Vuuren, 2004). It is rarely transmitted between cattle but congenital transmission from recovered cows to their offspring has been described. Disease does not occur and the virus does not persist in cattle herds that have no contact with wildebeest.

It has been stated that “because MCF-affected cattle and other infected species are not infectious, they represent true dead-end hosts and are not a source of infection to other animals” (Reid and Van Vuuren, 2004).

#### 19.1.5 Hazard identification conclusion

Germplasm would not be collected from clinically ill donors. Infected cattle are considered to be dead-end hosts and the likelihood of introducing the virus into New Zealand in germplasm is negligible. Therefore AHV-1 is not considered to be a potential hazard.

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## 20 MISCELLANEOUS ARBOVIRUSES

### 20.1 HAZARD IDENTIFICATION

#### 20.1.1 Aetiological agent

Various arboviruses.

#### 20.1.2 OIE list

Not listed.

#### 20.1.3 New Zealand status

Exotic viruses, not listed as unwanted.

#### 20.1.4 Epidemiology

A large group of viruses that are transmitted by mosquitoes or *Culicoides* spp. have been identified. At least 65 different arboviruses are found in the Australian geographical region (Mackenzie et al 1994). Many of these result in sub-clinical or trivial infections of man and animals but more regularly stimulate antibody formation and are identified as circulating in the animals concerned in serological surveys. A few are associated with distinct and sometimes serious viral diseases. The following viruses are more commonly mentioned in the literature:

**Sinbis virus** is a mosquito-borne alphavirus for which the maintenance hosts are generally believed to be birds (Russell 1995). A closely related virus, Whataroa virus, occurs in New Zealand (Miles et al 1971). Humans have antibody to the virus in endemic areas. Antibody to the virus has been demonstrated in cattle. No reports about the virus in cattle more recent than 1977 have been found. No evidence could be found that the virus causes disease of cattle or that cattle are anything but dead-end hosts, or that the virus is excreted in semen or would contaminate embryos. Sinbis virus is not considered to be a potential hazard in the commodity.

**Epizootic haemorrhagic disease virus** is a *Culicoides*-borne orbivirus closely related to bluetongue and Palyam viruses (Maclachlan and Osburn 2004). It causes disease in deer in the United States. In Australia five sero-types of the virus have been isolated from non-clinically infected cattle (Parsonson and Snowdon 1985). The infection is not contagious and is transmitted by *Culicoides* spp. (Maclachlan and Osburn 2004). There is no evidence suggesting that it is transmitted in semen or embryos. It is not a zoonotic virus. Since *Culicoides* spp. are not present in New Zealand, the virus could not establish. It is not considered to be a potential hazard in the commodity.

**Gan Gan virus** is a mosquito-borne Bunyavirus, which has only been reported in New South Wales (Russell 1995). Antibody and rare cases associated with disease have been

reported in humans and antibody has been found in cattle. However, no reports could be found of clinical disease or viraemia in cattle. Therefore cattle are considered to be unable to transmit the infection to mosquitoes. There is no evidence that the virus is transmitted in cattle semen or embryos. Gan Gan disease is not considered to be a potential hazard in the commodity.

**Kunjin virus** is generally confined to Northern regions of Australia and sporadically occurs in central Australia in years of exceptional rainfall. It was absent from central Australia for 26 years before reappearing in 2000 (Brown et al 2002). However, it is rarely reported. In 2004 there were 4 cases in the Northern Territory (Liu et al 2005). According to Russell (1995), Whelan found cattle in the Northern Territory to be serologically positive. However, the main vertebrate hosts are believed to be water birds, particularly the Rufus night heron (Marshall 1988 according to (Russell 1995)). Experimental infection of sheep resulted in transient shedding of virus in lymph but virus disappeared with the production of antibodies within 3-4 days of infection (Pearson et al 1976). No evidence could be found to indicate that cattle become viraemic or act as maintenance hosts, or that the virus is transmitted in germplasm. Kunjin virus is not considered to be a potential hazard in the commodity.

**Murray Valley encephalitis virus** is an alphavirus that causes a disease of humans. The virus is active in the Northern Territory of Australia and some parts of western Australia from December till June, as indicated by a sentinel chicken programme (Broom 2003; Russell 1995). Human cases occur from February to July. Cattle seroconvert and are potential hosts but are poor amplifiers of the virus compared to rabbits and kangaroos (Kay et al 1985). In an experimentally infected sheep, the virus was cleared rapidly after the production of antibody 3-4 days after infection (Pearson et al 1976). Waterbirds, particularly night herons, are considered to be the major vertebrate hosts of the virus (Russell 1995). No evidence could be found that indicates that cattle play a role in the maintenance of the virus or that the virus is transmitted in germplasm. Therefore the virus is not considered to be a potential hazard in the commodity.

**Chikungunya virus** is an alpha virus which has a wide distribution in Africa, India, and South East Asia. It has recently spread to several Indian Ocean islands including Reunion and Mauritius (Chastel 2005). Serological studies have indicated a low prevalence of antibodies in cattle but no record could be found indicating that cattle are efficient amplifiers of virus or are maintenance hosts or that the virus is transmitted in cattle germplasm. Therefore the virus is not considered to be a potential hazard in the commodity.

**St Louis encephalitis virus** causes serious disease and occasional mortality in humans in the USA, and Central and South America. Sporadic cases have been recorded (Day and Stark 2000; Jones et al 2002). There is a considerable amount of evidence that indicates that birds are the maintenance hosts of the virus (Gruwell et al 2000; Reisen et al 2003; Shaman et al 2003). Although one study indicates that cattle seroconverted (Ulloa et al 2003) no evidence could be found that they had significant viraemias or that cattle are

maintenance hosts for the virus or transmit the disease in germplasm. St Louis encephalitis virus is not considered to be a potential hazard in the commodity.

**Japanese encephalitis virus** causes serious disease in humans. Between 30,000 and 50,000 cases occur annually in the Asian region (CDC 2006; WHO 2006). The disease has recently emerged in Australia in islands in the Torres Straits and the Cape York peninsular (Mackenzie 1999; Ritchie and Rochester 2001). Approximately 30 % of cases end fatally and serious complications are common in recovered patients. The disease is transmitted by mosquitoes of the *Culex* genus. The maintenance host for the virus are ardeid birds (herons and egrets) and the virus is amplified in pigs that are sub-clinically infected (CDC 2006; WHO 2006). However, cattle are not known to be involved in the maintenance or amplification of the virus and transmission in cattle germplasm has not been described, for this reason the virus is not considered to be a potential hazard in the commodity.

#### 20.1.5 Hazard identification conclusion

In view of the above, none of the arboviruses covered in this section are considered to be potential hazards in the commodity.

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## 21 PALYAM GROUP VIRUSES

### 21.1 HAZARD IDENTIFICATION

#### 21.1.1 Aetiological agent

Family: Reoviridae; Genus: Orbivirus, viruses belonging to the Palyam serogroup.

#### 21.1.2 OIE list

Not listed.

#### 21.1.3 New Zealand status

Considered exotic to New Zealand, not listed on the unwanted organisms register.

#### 21.1.4 Epidemiology

The Palyam serogroup of the orbiviruses are represented by a large number of viruses that occur in Australia, Africa, and Asia (Swanepoel 2004). There is some confusion about the identification of some of the viruses and further new viruses are likely to be found in the future. In one review 15 viruses were listed (Swanepoel 2004) and others have been reported (Doyle and Walton 1992). The viruses most commonly infect cattle, but neutralizing antibody has been found in sheep and goats (Swanepoel 2004). The main vectors for the viruses are *Culicoides* spp. but the Palyam viruses have also been isolated from ticks in Africa and mosquitoes in India (Swanepoel 2004). Large numbers of isolations of arboviruses including many Palyam viruses have been made from the blood of naturally infected cattle without clinical signs of disease and *Culicoides* midges in South Africa and Australia (Cybinski and St George 1982; Gard et al 1989; Gard et al 1988a; Gard et al 1988b; Littlejohns et al 1988; Nevill et al 1992; Theodoridis et al 1979).

Although the viruses usually cause mild or subclinical infections they have been associated with cattle abortions in Zimbabwe. Kasba virus was associated with congenital abnormalities such as hydraencephaly and cerebellar hypoplasia in calves in Japan (Goto et al 1988; Miura et al 1990). Similar congenital abnormalities were reported from Australia (Kirkland et al 1992). After infection with Kasba virus, Goto *et al* reported that cattle were consistently viraemic for 2 weeks and intermittently viraemic for 8 weeks (cited by Swanepoel 2004).

An arbovirus and *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al 1991). In a typical year serum samples were collected from 10 sentinel cattle from each of 17 herds, and a total of about 15,000 insects were collected from light traps (Motha et al 1997). No seroconversion has been detected in sentinel cattle and no *Culicoides* have been trapped to date.

### 21.1.5 Hazard identification conclusion

Although the Palyam virus group does not cause economically important diseases in endemically infected countries, they do occasionally cause abortions or foetal malformations and could have severe effects in a naïve population of cattle. Therefore these viruses are classified as a potential hazard in the commodity.

## 21.2 RISK ASSESSMENT

### 21.2.1 Entry assessment

#### 21.2.1.1 Semen

No information has been found regarding the transmission of Palyam viruses in semen. However, Palyam viruses belong to the *Orbivirus* genus and can be expected to behave in a similar manner to bluetongue. Bluetongue virus is excreted in semen only while animals remain viraemic (Bowen et al 1983; Howard et al 1985). According to Muria et al cattle remained viraemic with Kasba virus for up to 8 weeks (Swanepoel 2004). Therefore the likelihood of entry of Palyam viruses in semen is considered to be non-negligible.

#### 21.2.1.2 Embryos

No information has been found regarding the transmission of Palyam viruses by embryos. Therefore, as with semen, the likelihood of entry is assumed to be low but non-negligible.

### 21.2.2 Exposure assessment

Semen or embryos would be inseminated or transplanted into susceptible recipients. Therefore the likelihood of exposure is considered to be non-negligible.

### 21.2.3 Consequence assessment

It is assumed that insemination or transplantation of infected germplasm would lead to infection of the recipient. Infection would be subclinical and non-contagious to in-contact animals. A period of viraemia lasting up to 8 weeks could be expected in infected recipients and during this period they could infect competent vectors. *Culicoides* spp. are the natural host of the viruses (Swanepoel 2004) and other hosts are of doubtful significance. Since *Culicoides* spp. are not present in New Zealand the likelihood that Palyam viruses would be able to establish in New Zealand is considered to be negligible. Since none of New Zealand's trading partners have requirements regarding the presence of Palyam viruses in New Zealand, the consequences of individual animals being infected as a result of insemination or embryo transfer are considered to be negligible.

The viruses are not zoonotic and there are no consequences for human health.

The viruses have only been described in ruminants. They could infect feral goats and thar but infection of these species would have no consequences for their health. The closely related *Orbivirus*, epizootic haemorrhagic disease virus infects deer (Parsonson and Snowdon 1985) so it is probable that Palyam viruses could also infect deer but would be unlikely to affect their health. Therefore, there would be no consequences for New Zealand wild or feral animals or the environment.

In view of the above, the consequences of the introduction of Palyam viruses in germplasm are considered to be negligible.

#### 21.2.4 Risk estimation

Since the consequences of introduction of Palyam viruses in germplasm are considered to be negligible, the risk estimate for Palyam viruses is negligible and they are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

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## 22 RABIES

### 22.1 HAZARD IDENTIFICATION

#### 22.1.1 Aetiological agent

Family: Rhabdoviridae; Genus: Lyssavirus, rabies virus. There are a number of closely related lyssaviruses such as the European bat Lyssavirus which cause similar diseases.

#### 22.1.2 OIE list

Listed.

#### 22.1.3 New Zealand status

Listed on the unwanted organisms register as an unwanted and notifiable organism.

#### 22.1.4 Epidemiology

Rabies is a disease of all mammals including man. It is characterized by severe nervous signs and is invariably fatal.

Rabies occurs widely around the world but there are a number of countries including mainly island and peninsular countries that are free from the disease. In some countries such as Denmark and Australia that are free from true rabies virus, 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).

The virus is carried mainly by carnivores. In the final stages of the disease they excrete the virus in their saliva and transmit the disease 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 of virus in a bite wound the virus enters peripheral nerves and is transported through the nerves to the central nervous system. After entering the peripheral nerves the virus is not found in any other body tissues or in the blood. Amputation of limbs of mice experimentally infected in the foot pads has been shown to prevent the virus from progressing to the brain (Swanepoel 2004). The passage of virus through the nervous system is a slow process and depending on the site of infection, the dose of virus and the animal concerned, the incubation period before the appearance of clinical signs may vary from weeks to years. In cattle, 2-12 weeks has been reported but an incubation period of 87 weeks was reported in a case of experimental infection (Swanepoel 2004). In the *Terrestrial Animal Health Code* the incubation period is defined as 6 months. The

occurrence of viraemia is an exceptional event other than 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 generalized dissemination of infection in the brain. It then multiplies in the salivary glands and is excreted in the saliva. In the terminal stages of the disease animals become incoordinated and about 50% of infected cattle become aggressive. The disease lasts from a few days to a few weeks and invariably ends fatally. Typically, animals become incoordinated and aggressive and salivate excessively or develop a paralytic form of the disease (Swanepoel 2004). Cattle are generally dead-end hosts since they are unlikely to bite other animals or man. Although the disease is dramatic and a cause for serious concern, the actual prevalence in cattle is low. In South Africa over a period of 72 years 3,029 cases were reported in cattle (Swanepoel 2004). This is an average of 42 cases per year from a cattle population of approximately 10 million. Therefore, even if the disease was grossly under-reported, the prevalence was low.

### **22.1.5 Hazard identification conclusion**

Rabies virus can infect virtually all animals and man. It is an exotic, notifiable disease and is an important zoonosis. Therefore, it is classified as a potential hazard in the commodity.

## **22.2 RISK ASSESSMENT**

### **22.2.1 Entry assessment**

#### **22.2.1.1 Semen**

Infection of semen has not been described and the experiments would be dangerous to carry out and are unlikely to be done. However, viraemia in cases of rabies does not occur except in experimental infections of mice (Swanepoel 2004) 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 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 infected with rabies is considered to be negligible.

#### **22.2.1.2 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), and 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 occur. The likelihood of embryos being infected with rabies virus is considered to be negligible.

### 22.2.2 Risk estimation

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

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## 23 ROSS RIVER AND BARMAH FOREST VIRUSES

### 23.1 HAZARD IDENTIFICATION

#### 23.1.1 Aetiological agent

Family: Togaviridae; Genus: Alphavirus, Ross River virus and Barmah Forest viruses.

#### 23.1.2 OIE list

Not listed.

#### 23.1.3 New Zealand status

Exotic.

#### 23.1.4 Epidemiology

Ross River and Barmah Forest viruses are mosquito-borne alphaviruses that occur in Australia. They have not been reported in North America or Europe (Harley et al 2001; Russell 2002; Russell and Doggett 2006). They are zoonotic viruses but are not known to cause clinical disease in any domestic animals.

Approximately 5,000 human cases of Ross River fever (characterised by fever, polyarthritis, and rash) are notified annually in Australia (Harley et al 2001; Russell 2002; Russell and Doggett 2006). Ross River virus has been isolated from at least 30 species of mosquitoes and transmission has been demonstrated from at least 13 species (Harley et al 2001). The major mosquito vectors are *Culex annulirostris* in freshwater habitats, and *Aedes vigilax* and *Aedes camptorynchus* in northern and southern coastal regions. Other species involved in transmission include *Aedes normanensis*, *Coquillettidia linealis*, and *Aedes notoscriptus*. Based mainly on serological evidence, the reservoir hosts for the virus are believed to be large marsupials such as kangaroos and wallabies (Russell 2002; Russell and Doggett 2006; Vale et al 1991). However, antibodies to the virus have been found in a wide variety of placental and marsupial mammals, and viral isolations from naturally infected vertebrates have only been recorded in eight cases including two cases from macropods and two from horses (Harley et al 2001). Humans may also act as reservoirs of infection and a mosquito human cycle probably occurs during outbreaks of the disease.

Infections with Barmah Forest virus occur less commonly and little is known about the hosts of the virus (Russell and Doggett 2006). Effects of Ross River virus infection vary from subclinical infection, a transient rash and mild illness to polyarthritis and chronic illness. Clinical signs associated with Barmah Forest virus are similar. Recovery may occur in a few weeks but in some cases signs may persist for months or years (Harley et al 2001; Russell 2002).

The Ross River virus is normally confined to Australia, Papua New Guinea, and the Solomon Islands. In the latter two countries the virus may be introduced periodically from Australia (Russell 2002). A massive outbreak that occurred in the Pacific region in 1979-80 involved outbreaks in Fiji, American Samoa, the Cook Islands, and New Caledonia, and probably also Tonga, Kiribati, and Western Samoa. The outbreak seems to have been started by a single traveler from Australia infecting mosquitoes in Fiji (Harley et al 2001; Russell 2002). Since the virus is known to be transmitted by *Aedes aegypti* and *Aedes albopictus* the potential exists for outbreaks of disease to occur in countries where these species of mosquitoes are present.

Ross River and Barmah Forest viruses have not occurred in New Zealand. Two exotic species of mosquitoes *Aedes notoscriptus*, a probable vector of Ross River virus (Russell and Doggett 2006) and *Aedes camptorhynchus*, a known vector of the virus, have become established in New Zealand (Derraik and Calisher 2004). However, *Aedes camptorhynchus* is the subject of an eradication campaign, the outcome of which remains uncertain.

Antibody against the virus has been demonstrated in cattle but no isolations of virus have been reported (Harley et al 2001; Vale et al 1991). Kay and Aaskov failed to find viraemia in experimentally infected calves (Harley et al 2001). There have been no reports indicating that cattle could be linked epidemiologically with the disease in humans. No reports were found of the viruses infecting cattle semen or embryos.

### 23.1.5 Hazard identification conclusion

Since there is no indication that cattle can act as reservoirs of these viruses, they are not classified as potential hazards in the commodity.

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## **24 RIFT VALLEY FEVER**

### **24.1 HAZARD IDENTIFICATION**

#### **24.1.1 Aetiological agent**

Family: Bunyaviridae; Genus: Poliovirus, Rift Valley fever virus.

#### **24.1.2 OIE list**

Listed.

#### **24.1.3 New Zealand status**

Listed on the unwanted organisms register as an exotic notifiable organism.

#### **24.1.4 Epidemiology**

Rift Valley fever is an acute disease of sheep, goats, cattle, and people. The aetiological agent is an arbovirus that is carried by mosquitoes. It 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. Some cattle develop acute disease with fever, lacrimation, salivation and fetid diarrhoea. 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).

The infection was originally confined to sub-Saharan Africa but spread to Egypt (Balkhy and Memish 2003) in 1977 and to the Arabian peninsular in 2000 (Al-Afalet et al 2003; Anonymous 2000; Balkhy and Memish 2003; Jup et al 2002). There is evidence that the virus was not present in the Arabian peninsular before the outbreak in 2000 (Al-Afalet et al 2003). Epidemics occur in seasons associated with abnormally heavy rainfall and the expansion of the breeding sites of vector mosquitoes. Typically the disease is not seen in the years between epidemics (Swanepoel 1994). 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). Transovarial infection may occur in mosquitoes but is a rare occurrence and it is not known how the virus is maintained through inter epidemic periods (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 OIE specifies that the incubation period shall be 30 days (OIE, 2006). The disease usually follows an acute course in adult animals with abortion in pregnant females and a peracute course in neonates. Very high titers 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 contact with infected foetuses or other infected animal material or from mosquito bites. In humans there is fever, photophobia, and muscular weakness, and ocular problems complicate some cases. In less than 1% of cases, the haemorrhagic or encephalitic form of the disease may develop resulting in serious disease or death. In a recent outbreak in Saudi Arabia there were 882 confirmed cases and 124 deaths but the high proportion of deaths reported may have been influenced by under-reporting of mild cases (Balkhy and Memish 2003).

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

#### **24.1.5 Hazard identification conclusion**

The virus is an exotic, notifiable organism and is therefore classified as a potential hazard in the commodity.

### **24.2 RISK ASSESSMENT**

#### **24.2.1 Entry assessment**

##### **24.2.1.1 Semen**

There is no information available about the excretion of virus in semen. The organism has been listed as one that is likely to be present in semen and could be transmitted by semen (Hare 1985). It should be assumed that the virus would be excreted in semen during the viraemic period which lasts for up to 7 days (Swanepoel and Coetzer 2004). There is a more remote possibility that virus could be excreted in semen during the period of 21 days when blood is no longer infected but visceral organs are still infected (Swanepoel and Coetzer 2004). Therefore, the likelihood of virus being present in semen is considered to be non-negligible.

##### **24.2.1.2 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 (IETS 2004). It should be assumed that embryos could be infected at least during the period of viraemia (up to 7 days) and possibly during the 21 day period in which visceral organs remain infected (Swanepoel and Coetzer 2004). It is unlikely that an infected embryo would be viable, but in view of the lack of knowledge the likelihood of entry of virus is considered to be non-negligible.

#### 24.2.2 Exposure assessment

Imported semen or embryos would be inseminated or implanted into susceptible recipients. Therefore the likelihood of exposure is considered to be non-negligible.

#### 24.2.3 Consequence assessment

Although it is stated in the *OIE Terrestrial Animal Health Code* that commodities other than live animals and meat should “be considered as not having the potential to spread Rift Valley fever when they are the subject of international trade” (OIE, 2006), no evidence could be found to support or refute this statement. It is assumed that germplasm from viraemic animals 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.

At least 12 species of mosquitoes have been found to be infected with the virus (Swanepoel 1994) but it is not known whether mosquitoes indigenous to New Zealand could transmit the disease. The endemic mosquito *Aedes notoscriptus* is a laboratory vector of Rift Valley fever virus (Turell and Kay 1998). However, 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. Whether the disease could establish in *Aedes notoscriptus* in New Zealand is unknown. Because the disease has historically remained confined to Africa and the Middle East the likelihood of establishment in New Zealand is low. Pharo reviewed the literature and considered that it was unlikely that the disease could establish in New Zealand (Pharo 1999). However, since the competence of New Zealand mosquitoes to act as vectors for the virus is unknown the likelihood of establishment is non-negligible. Establishment of the disease in New Zealand could result in periodic serious losses to sheep and goat farmers and interference in international trade in animals. Additional certification and restrictions would apply to meat exported from an infected country (OIE, 2006).

The virus is a zoonotic organism and if it established in New Zealand it could be expected that people would become infected during disease outbreaks. They could become infected by mosquito bite or by contact with infected carcasses, abortion material, or meat (Swanepoel and Coetzer 2004). Most infections would result in a flu-like disease but a small percentage of cases could result in serious disease and death. In recent outbreaks of the disease in Saudi Arabia at least 882 confirmed cases of disease and 124 deaths occurred (Balkhy and Memish 2003). Therefore establishment of the disease would have serious consequences for human health.

The disease is one that is only known to infect domestic ruminants and possibly African buffalo 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.

Rift Valley fever is a zoonotic disease and if it were to establish it could cause serious economic consequences to the sheep industry and to a lesser extent to the cattle industry. Therefore the consequences of introduction are considered to be non-negligible.

#### **24.2.4 Risk estimation**

Since entry, exposure, and consequence assessments for semen and embryos are non-negligible, the risk estimate is non-negligible and Rift Valley fever virus is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

### **24.3 RISK MANAGEMENT**

#### **24.3.1 Option evaluation**

OIE makes no recommendations about the trade in germplasm from Rift Valley fever infected countries. Rift Valley fever has a short incubation period (12-36 hours) and the period of viraemia is of short duration (up to 7 days). However OIE specifies that for the purposes of the *Code* the incubation period shall be 30 days (OIE, 2006). Long-term carriers of virus do not occur and therefore quarantine of donors 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 *OIE Terrestrial Animal Health Code* refers to infected, disease free countries and recommends that live 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 Rift Valley fever (high summer rainfall), or were vaccinated, or held in mosquito free premises for 30 days prior to shipment (OIE, 2006). These recommendations could be applied directly to donors of germplasm.

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

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

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## 25 RINDERPEST

### 25.1 HAZARD IDENTIFICATION

#### 25.1.1 Aetiological agent

Family: Paramyxoviridae; Genus: Morbillivirus, rinderpest viruses.

#### 25.1.2 OIE list

Listed.

#### 25.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic, notifiable organism.

#### 25.1.4 Epidemiology

The OIE handistatus database reported only 1 outbreak of rinderpest in Kenya in wildlife in 2001 and one in cattle in 2003 (OIE 2006a). There have been no outbreaks of the disease in any other country since 1998. On 5 May 2005, 105 countries were recognized as free from rinderpest by OIE, another 40 countries had declared themselves provisionally free and 3 had declared provisionally free zones (OIE 2006b). All countries that New Zealand is likely to import cattle germplasm from are included in the list of countries officially free from rinderpest.

#### 25.1.5 Hazard identification conclusion

Since rinderpest is virtually a non-existent disease and does not occur in any of our likely trading partner's countries, the likelihood of importing rinderpest virus in germplasm is considered to be negligible, and it is not classified as a potential hazard in the commodity.

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## 26 VESICULAR STOMATITIS

### 26.1 HAZARD IDENTIFICATION

#### 26.1.1 Aetiological agent

Family: Rhabdoviridae; Genus: Vesiculovirus, vesicular stomatitis virus. Two main types (Indiana and New Jersey) are known. New Jersey contains only a single sub-type and Indiana has three sub-types.

#### 26.1.2 OIE list

Listed.

#### 26.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic, notifiable organism.

#### 26.1.4 Epidemiology

Vesicular stomatitis is considered to be a disease of horses, cattle, and pigs, and more rarely of sheep and goats (Schmidt 2004). In addition to being a virus of vertebrates, the virus 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).

Vesicular stomatitis 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 of the viral aetiology, 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 vesicular stomatitis, 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 in Georgia (Stallknecht 2000). In some seasons the disease spreads northward along riverbeds into locations in the northern USA (Schmidtman et al 1999) and even as far as Canada (Wilks 1994).

The most commonly held view is that the virus is transmitted by an insect vector. Virus has been isolated from sand flies (*Lutzomyia shannoni*) which are the most likely vectors (Braverman 1994; Comer et al 1994; Rodriguez et al 1996; Schmidtman et al 2002; Stallknecht 2000) but *Culicoides* spp. are also possible vectors and have been infected

experimentally (Nunamaker et al 2000). Blackflies (*Simulium* spp.) have also been incriminated in the transmission of the disease (Mead et al 2000). 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).

The maintenance hosts of the virus have not yet been conclusively established, but deer and raccoon (Stallknecht 2000) and the cotton rat *Sigmodon hispidus* (Jimenez et al 1996) have been found to have antibody to the virus. The white tailed deer has shown signs of infection and many other species of animals can be infected or develop antibodies against the virus (Blood et al 1989; Hanson and McMillan 1990).

The disease 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 the disease is 1-3 days (Wilks 1994), but for regulatory purposes a period of 21 days is given in the *OIE Terrestrial Animal Health Code* (OIE, 2006).

There is some controversy about the pathogenesis of the disease. 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 of the disease it is stated that virus replicates in the lower layers of the epidermis and there is no description 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 it has been stated that there is a primary viraemia with subsequent localization of virus in mucous membranes of the mouth and the skin around the coronets (Blood et al 1989). Viraemia was described in experimental infection of deer mice (Cornish et al 2001).

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).

### **26.1.5 Hazard identification conclusion**

Vesicular stomatitis virus is an important exotic pathogen of cattle. Therefore, it is classified as a potential hazard in the commodity.

## **26.2 RISK ASSESSMENT**

### **26.2.1 Entry assessment**

#### **26.2.1.1 Semen**

There is no information about the transmission of the disease by semen. Large ruminants were listed as likely to excrete the virus in semen and possibly able to transmit the virus (Hare 1985), but no evidence was quoted to support this view. Similarly, it has been listed in a category of diseases “with evidence that risk of transmission (by artificial insemination) is moderate or high”, but no evidence was given to support this categorization. In the same article vesicular stomatitis is listed as a disease for which isolation of the agent from semen has not been reported (Eaglesome and Garcia 1997). If viraemia is indeed absent in mammals, as seems likely (see section 2.1.4), then excretion of virus in semen is unlikely to occur. While this debate remains unresolved it should be assumed that virus could be excreted in semen during a viraemic period. It is unlikely that cattle would be viraemic and displaying no clinical signs at the time of semen collection, even in the most heavily infected countries. The likelihood of release of virus in germplasm is therefore unlikely but considered to be non-negligible.

#### **26.2.1.2 Embryos**

The virus adhered to the zona pellucida when cattle embryos were exposed to the virus and could not be removed by washing (Lauerman et al 1986). However, if viraemia does not occur in this disease contamination of embryos with virus is unlikely. IETS has classified the disease as a Category 4 organism in cattle and swine i.e. “a disease on which preliminary work has been conducted or is in progress”. It is concluded that, the likelihood of embryos being infected with the virus is low but non-negligible.

### **26.2.2 Exposure assessment**

Imported semen and embryos would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is considered to be non-negligible.

### **26.2.3 Consequence Assessment**

No data relating to the use of infected semen or embryos in susceptible recipients or other ruminants 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 vesicular stomatitis but would not be contagious and would not infect animals in contact with them. They could infect competent vectors while they are viraemic. Vectors of the disease are not known to occur in New Zealand. It seems unlikely that a suitable combination of competent vectors and maintenance hosts exists outside the endemic areas of the Americas as the disease has never established anywhere else. However, since no evidence exists to prove or disprove the possibility, the likelihood of establishment in New Zealand should be considered to

be non-negligible. The establishment of the disease in New Zealand would have serious consequences since it would create difficulties in distinguishing the disease from foot and mouth disease, would have some economic consequences for individual farmers and could have a negative impact on trade in live animals. The OIE does not recommend any restrictions associated with this disease for trade in meat, dairy products or semen but recommends that trade in embryos should be restricted to disease free countries or zones (OIE 2006). Therefore establishment of the virus in New Zealand could have implications for trade in embryos or live animals.

The 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). It is likely that the establishment of the disease in New Zealand would result in sporadic infections in humans during outbreaks of disease in livestock.

The exact host range of the virus 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 could be infected but indigenous birds are unlikely to be susceptible. Infections in feral and wild species are likely to be subclinical. Therefore the effects on the environment are likely to be negligible.

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

#### **26.2.4 Risk estimation**

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

### **26.3 RISK MANAGEMENT**

#### **26.3.1 Options**

The OIE gives recommendations for trade in live animals and embryos but not for semen (OIE 2006). It was concluded in the release assessment that the likelihood of viral contamination was very low but non-negligible. The OIE regulations for live animals and embryos could be combined to provide suitable options for semen and embryos. Germplasm donors could be restricted to animals from non-infected countries or zones or kept in insect free quarantine for a suitable length of time and tested by an OIE recommended serological test (OIE 2006; Schmidt 2004). The serological test could be

done after completion of germplasm collection instead of before germplasm collection as recommended by OIE for live animals.

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

- Donors could be required to be resident for at least the 30 days prior to germplasm collection and during germplasm collection, in a country or zone that is free from vesicular stomatitis.
- Donors could be required to be resident on a property where no cases of vesicular stomatitis were known to have occurred within 100 kilometers of the collecting centre during the period from 30 days before the first collection of semen until 30 days after the last collection of semen for the consignment.
- Donors could be required to be kept in an insect free quarantine station for at least the 30 days prior to and during germplasm collection and be subjected to an OIE recommended serological test with a negative result between 3-6 weeks after germplasm collection.

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## **27 WEST NILE DISEASE**

### **27.1 HAZARD IDENTIFICATION**

#### **27.1.1 Aetiological agent**

Family: Flaviviridae; Genus: Flavivirus, West Nile virus.

#### **27.1.2 OIE list**

Listed.

#### **27.1.3 New Zealand status**

Exotic organism, not listed as unwanted or notifiable by MAF.

#### **27.1.4 Epidemiology**

West Nile virus was originally isolated in Uganda in 1937. It is found all over Africa and has also been found in France (1962), Romania (1996), and Russia (1999) (Bunning et al 2004). The virus spread to the United States in 1999 and since then has spread throughout the USA (CDC 2003a) and adjoining countries. Disease is seen mainly in humans and horses but the virus also causes deaths in wild birds. Most cases in humans are asymptomatic, but there have been over 15,000 cases of disease and over 600 deaths in the epidemic in the USA (Higgs et al 2005).

The virus is transmitted by mosquitoes and maintained in a bird mosquito cycle (CDC 2003b). At least 43 species of mosquitoes have been suspected of acting as vectors of the disease (Gingrich and Williams 2005). The virus can be transmitted from infected mosquitoes to non-infected mosquitoes when they feed together on non-infected hosts (Higgs et al 2005).

No descriptions of clinical cases of disease in cattle have been reported, but there are several reports of cattle being positive for antibodies in serological surveys (Fontenille et al 1989; Karadzhov et al 1982; Koptopoulos and Papadopoulos 1980; Olaleye et al 1990). This indicates that the virus causes inapparent infections in cattle. Calves infected experimentally with West Nile virus developed antibody but no detectable viraemia was found (Ilkal et al 1988). According to CDC "People, horses, and most other mammals are not known to develop infectious-level viraemia very often, and thus are probably dead-end or incidental-hosts" (CDC 2003b). Infections in cattle are therefore subclinical and they do not develop viraemia and are dead-end hosts. Therefore the likelihood that the virus would be transmitted in imported cattle germplasm is considered to be negligible.



### 27.1.5 Hazard identification conclusion

Since cattle are dead end hosts for WNV, the likelihood that virus would be present in imported germplasm is considered to be negligible. Therefore, the organism is not considered to be a potential hazard in the commodity.

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## **28 ANTHRAX**

### **28.1 HAZARD IDENTIFICATION**

#### **28.1.1 Aetiological agent**

*Bacillus anthracis*.

#### **28.1.2 OIE list**

Listed.

#### **28.1.3 New Zealand status**

Exotic, notifiable disease last diagnosed in 1954.

#### **28.1.4 Epidemiology**

Anthrax is a bacterial disease of most warm-blooded vertebrates including man. The disease occurs in most countries but New Zealand has been free from the disease for 50 years (Gill 1992).

The infectious agent is a spore forming bacillus that can survive in the spore state in suitable soils for many decades. In 1999 an outbreak occurred in Australia on farms where the disease had not occurred for about 100 years. On these properties earthworks in relation to an irrigation scheme possibly resulted in disturbance of old burial sites of cattle (Turner et al 1999a; Turner et al 1999b). A related spore-forming bacillus has been cultivated from palaeozoic slate plugs believed to be 500 million years old (De Vos 1994). *Bacillus anthracis* is probably an obligate pathogen that only multiplies in animals although an alternative theory is that the organism can multiply in soil (De Vos and Turnbull 2004).

The organism multiplies in infected animals and on the death of the animal when a carcass is opened, it sporulates resulting in contamination of soil and the environment. In unopened carcasses the organism does not sporulate and is destroyed by putrefaction (De Vos and Turnbull 2004). The disease is not directly transmissible from animal to animal and infection is believed to be associated with ingestion of contaminated soil or other infected material. Biting flies may carry the infection but they were not considered to be important in the transmission of the disease in an outbreak in Australia (Turner et al 1999a). Blowflies may be important in the spread of the disease when they have been feeding on infected carcasses (De Vos and Turnbull 2004). Infection through skin wounds and abrasions may also occur and is a common route of infection for humans (De Vos and Turnbull 2004). In wool sorters disease and acts of terrorism infection can occur by inhalation, but this is not of importance in cattle. Carriers of the disease may occur in partially immunized cattle that recover from natural infection (De Vos 1994).

The incubation period probably varies from one to 14 days. In the peracute form in susceptible species, the course of the disease is only a few hours (De Vos and Turnbull 2004). In the acute form, death usually occurs within 48 hours (Blood and Radostits 1989). Sub-acute and chronic forms of the disease occur in less susceptible animals such as pigs and carnivores (De Vos and Turnbull 2004).

### 28.1.5 Hazard identification conclusion

Anthrax is an exotic, notifiable, and zoonotic disease and is therefore classified as a potential hazard in the commodity.

## 28.2 RISK ASSESSMENT

### 28.2.1 Entry assessment

Cattle suffer from the acute or peracute forms of anthrax and die quickly after they become infected. The *OIE Terrestrial Animal Health Code* states that “there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs” (OIE, 2006). Infection occurs as a result of the ingestion of spores and not from vegetative forms of the organisms (De Vos 1994). The organisms in an anthrax animal will only sporulate after death of the animals when the carcass is opened and the organisms are exposed to air. There is considered to be a negligible likelihood that semen or embryos collected from healthy donors in facilities that meet New Zealand requirements for collection centres, and processed according to IETS recommended methods could be infected with *Bacillus anthracis*. In addition, the vegetative form of *Bacillus anthracis* is sensitive to penicillin, streptomycin, and gentamycin, and it is common practice to include at least one of these antibiotics at bacteriocidal concentrations in semen diluents and embryo washing fluids.

### 28.2.2 Risk estimation

Since the likelihood of entry is negligible, the risk estimate for *Bacillus anthracis* is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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## 29 BRUCELLOSIS

### 29.1 Hazard Identification

#### 29.1.1 Aetiological agent

Primarily *Brucella abortus*, although *Brucella suis* and *Brucella melitensis* may occasionally infect cattle.

#### 29.1.2 OIE list

Listed.

#### 29.1.3 New Zealand status

*Brucella abortus*, *Brucella suis*, and *Brucella melitensis* are listed as unwanted notifiable organisms.

#### 29.1.4 Epidemiology

Brucellosis of cattle is a disease that formerly had a world-wide distribution, but has now been eradicated from many developed countries. New Zealand has been free from bovine brucellosis since 1989 (Hellstrom 1991; Mackereth 2003).

Information about the disease has been extensively reviewed (Godfroid et al 2004). *Brucella abortus* infects cattle and rarely other species of ruminants and causes a serious disease in humans. In cattle the disease is characterised by abortion in females and by orchitis, epididymitis, and infection of the accessory sexual glands in bulls. Infected animals remain chronically infected and females may excrete the organism in their milk and in their uterine discharges after calving or abortion. The uterine discharges contain enormous numbers of organisms. The disease is generally transmitted by ingestion of food or water and on fomites that have been contaminated by uterine discharges. The incubation period varies from weeks to years depending on how incubation period is defined, whether the animals were pregnant, and the stage of pregnancy when infected. Infection of bulls is less common than cows. Some calves born to infected dams may remain seronegative carriers of the infection and may excrete the organism when they calve.

The disease is diagnosed by serological tests such as the complement fixation test or ELISA and by isolation of the organism from uterine discharge, aborted fetuses, milk, and semen (Nielsen and Ewalt 2004). Diagnosis of the disease is more difficult in bulls in which the tests are less sensitive.

*Brucella abortus* is a zoonotic organism that causes a serious debilitating disease of humans. Humans can contract the disease by drinking unpasteurised milk or by contact with cows at calving.

### **29.1.5 Hazard identification conclusion**

The agents of bovine brucellosis are exotic, notifiable organisms that cause serious diseases of cattle and humans. Bovine brucellosis is therefore considered to be a potential hazard in the commodity.

## **29.2 RISK ASSESSMENT**

### **29.2.1 Entry assessment**

#### **29.2.1.1 Semen**

Orchitis, epididymitis, and infection of the accessory glands occurs in bulls (Godfroid et al 2004). Semen from infected bulls may be contaminated by *Brucella abortus* (Plant et al 1976; Rankin 1965). Therefore the likelihood that semen would contain *Brucella abortus* is considered to be non-negligible.

#### **29.2.1.2 Embryos**

There is a considerable body of evidence that shows that *Brucella abortus* is not carried by properly prepared and washed 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). *Brucella abortus* has also been shown to be sensitive to the antibiotics used in preparation of embryos (Stringfellow et al 1986). These and other findings have led IETS to classify *Brucella abortus* as a Category 1 disease i.e. 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” (IETS 2004). Therefore, provided embryos are properly processed and treated with appropriate antibiotics, the likelihood that they would contain *Brucella abortus* is considered to be negligible.

### **29.2.2 Exposure assessment**

Since semen may contain *Brucella abortus* and will be inseminated into susceptible recipient the likelihood of exposure is non-negligible.

### **29.2.3 Consequence assessment**

Infected semen inseminated into susceptible recipients is likely to cause infection in the recipient (Manthei et al 1950). If a recipient becomes pregnant she would be likely to abort and could contaminate the environment and infect other susceptible stock. This could lead to a breakdown in New Zealand’s status of freedom from brucellosis and lead to an expensive campaign to eradicate the new infection. There could also be consequences for trade in live animals since New Zealand would no longer have country freedom status.

Since brucellosis is a serious disease of humans, re-establishment of the disease in New Zealand cattle would be expected to lead to some cases of infection in people. Therefore, the consequences for human health are considered to be non-negligible.

As *Brucella abortus* infection has been described in wapiti and elk, it is possible that New Zealand red deer could be infected. However, descriptions of serious consequences of infection in these animals are lacking. There were no reports of infection in New Zealand deer when the disease was endemic. The infection in wildlife has been described as only “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 considered to be negligible.

In conclusion, the consequences of introducing infected 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.

## **29.2.4 Risk estimation**

### **29.2.4.1 Semen**

Since entry, exposure, and consequence assessments for *Brucella abortus* in semen are non-negligible, the risk estimate for semen is non-negligible. *Brucella abortus* is classified as a hazard in bovine semen and risk management measures can be justified.

Since the entry assessment for *Brucella abortus* in embryos was negligible, the risk estimate for embryos is negligible. *Brucella abortus* is not classified as a hazard in bovine embryos and risk management measures are not justified.

## **29.3 RISK MANAGEMENT**

### **29.3.1 Options**

The OIE *Code* recommends that, for the importation of semen, donor bulls should be from a semen collection centre at which the testing programme for the bulls includes serological testing with both the buffered *Brucella* antigen agglutination test and the complement fixation test. When the semen is collected from donors that are not resident at a semen collection centre, they should be from a country or zone that is free from brucellosis or kept in a herd that is officially free from brucellosis and tested serologically with a buffered *Brucella* antigen test (OIE, 2006). It could be specified that New Zealand only accept semen from a semen collection centre.

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

- Donor bulls could be required to be kept since birth in a country or zone that is officially free from brucellosis.
- Donor bulls could be housed at an artificial breeding centre where the testing programme for bulls includes testing with both the complement fixation test and the buffered antigen agglutination test.
- Donor bulls could be required to be 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.
- Donor bulls could be required to be 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.

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## 30 MELIOIDOSIS

### 30.1 HAZARD IDENTIFICATION

#### 30.1.1 Aetiological agent

*Burkholderia pseudomallei* (formerly *Pseudomonas pseudomallei* and *Malleomyces pseudomallei*).

#### 30.1.2 OIE list

Not listed.

#### 30.1.3 New Zealand status

Listed on the unwanted organisms register as an unwanted exotic organism.

#### 30.1.4 Epidemiology

Melioidosis is a disease of man and animals that occurs predominantly in the tropical and subtropical regions of Asia and northern Australia and in some foci in Africa (Groves and Harrington 1994; Inglis 2004; Inglis et al 2004). It occasionally spills over into temperate regions and a case has occurred in New Zealand (Corkill and Cornere 1987). The aetiological agent occurs in the environment and is widely distributed in water and soil (Sprague and Neubauer 2004). It has been transmitted to animals via oral mucosa, nasal mucosa, ingestion, parental inoculation, and skin scarification (Groves and Harrington 1994). Infection in natural cases is probably by contact with infected water and mud especially through abrasions and wounds. Water was implicated as a possible source of infection in six locations in one study (Inglis et al 2004). A case of presumed sexual transmission has been described from a man with *Burkholderia pseudomallei* prostatitis (Groves and Harrington 1994).

In animals, clinical melioidosis is most commonly seen in sheep, goats, and swine. Cattle are thought to be resistant to infection (Groves and Harrington 1994). In one investigation isolations were made from pigs, goats, sheep, and birds but not from cattle (Thomas 1981). In animals the agent may cause a wide variety of signs varying from septicaemia and acute respiratory infections to localized abscesses. There is no evidence that the infection is transmitted by semen or embryos.

#### 30.1.5 Hazard identification conclusion

*Burkholderia pseudomallei* is an organism found very widely in the environment in tropical and subtropical areas, but has not established in temperate climates. It appears to be an opportunistic pathogen and there are no descriptions of it being transmitted by semen or embryos in animals. Therefore, it is not considered to be a potential hazard in the commodity.

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## **31 BOVINE TUBERCULOSIS**

### **31.1 HAZARD IDENTIFICATION**

#### **31.1.1 Aetiological agent**

*Mycobacterium bovis*.

#### **31.1.2 OIE list**

Listed.

#### **31.1.3 New Zealand status**

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

#### **31.1.4 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 feral possums, pigs, goats, and ferrets.

The lesions of the primary complex of infection are localized to the organ of entry and/or the associated lymph node. In many cases the infection remains localized to the primary complex. Sometimes it spreads to infect other organs or becomes generalized 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 epithelioid granulomas with abscessation and sometimes calcification. Transmission is by contact with other infected animals and is usually by the respiratory route but can be by ingestion of infected material. 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 seldom reported and often involves *Mycobacterium tuberculosis* infection of bovines (Adeniran et al 1992).

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

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 is still the intradermal tuberculin test (Cousins et al 2004). A more

recently developed test that is used in some circumstances is the interferon-gamma test (Wood et al 1991).

The organism can be cultured by standard methods or bacterial DNA can be identified by PCR analysis (Palmer 2004).

### **31.1.5 Hazard identification conclusion**

*Mycobacterium bovis* is an endemic organism that is the subject of a national eradication campaign administered by the Animal Health Board under a pest management strategy as defined in the Biosecurity Act. It causes severe disease in a number of animal species including cattle and it may affect humans. Therefore, *Mycobacterium bovis* is classified as a potential hazard in the commodity.

## **31.2 RISK ASSESSMENT**

### **31.2.1 Entry assessment**

#### **31.2.1.1 Semen**

*Mycobacterium bovis* has been listed as an organism that is known to be excreted in bull semen (Hare 1985). It was shown to be regularly excreted in the semen of a bull (Niyaz Ahmed et al 1999). The organism has also been isolated from a typical granulomatous lesion in the prepuce of a bull (Thoen et al 1977). The occurrence of animals that are excreting the organism in their semen is assumed to be low as reported cases in the literature are rare. It is concluded that the likelihood of the entry of the organism in semen is very low but non-negligible.

#### **31.2.1.2 Embryos**

The infection of embryos with *Mycobacterium bovis* has not been described. However, the uterus and genital tract of cattle can be infected by *Mycobacterium bovis* (Biolatti et al 1989; Cousins et al 2004; Muscarella et al 1974). *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). It is therefore likely that infection of the genital tract is possible in cattle and that in these cases the organisms could adhere strongly to the zona pellucida of ova. However, infections of the genital tract are rare in cattle. The likelihood of entry of the organism in embryos is therefore considered to be low but non-negligible.

### **31.2.2 Exposure assessment**

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

### 31.2.3 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 the recipients. Infected cattle 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 campaign to eradicate bovine tuberculosis. 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.

*Mycobacterium 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 *Mycobacterium bovis* infections in humans are rare and the increase in the number of cases caused by introducing infected germplasm is likely to be immeasurably 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 considered to be non-negligible.

### 31.2.4 Risk estimation

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

## 31.3 RISK MANAGEMENT

### 31.3.1 Options

OIE defines conditions for recognition of a herd that is officially free from tuberculosis and for the export of bovine embryos and semen (OIE 2006). These recommendations could be adopted for the importation of germplasm into New Zealand.

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

- Semen donors could be required to show no clinical sign of bovine tuberculosis on the day of collection of the semen.
- Semen donors could be required to be kept in an artificial insemination centre free from bovine tuberculosis in a country, zone or compartment free from bovine

tuberculosis and which only accepts animals from free herds in a free country, zone or compartment as defined by the OIE.

- Semen donors could be required to show negative results to tuberculin tests carried out annually and be kept in a herd free from bovine tuberculosis as defined by the OIE.
- Embryo donors and all other susceptible animals in the herd of origin could be required to show no clinical sign of bovine tuberculosis during the 24 hours prior to embryo collection.
- Embryo donors could be required to have originated from a herd free from bovine tuberculosis in a country, zone or compartment free from bovine tuberculosis;
- Embryo donors could be required to be kept in a herd free from bovine tuberculosis, be isolated in the establishment of origin for the 30 days prior to departure to the collection centre, and be subjected to a tuberculin test for bovine tuberculosis with negative results.

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## 32 CONTAGIOUS BOVINE PLEUROPNEUMONIA

### 32.1 RISK ASSESSMENT

#### 32.1.1 Aetiological agent

*Mycoplasma mycoides* subsp. *mycoides* SC.

#### 32.1.2 OIE list

Listed.

#### 32.1.3 New Zealand status

Listed on the unwanted organisms register as an exotic notifiable organism.

#### 32.1.4 Epidemiology

Contagious bovine pleuropneumonia (CBPP) (Aliyu et al 2000) is a chronic disease of cattle. Asian buffalo and goats may also be infected but their role as a reservoir of infection is doubtful (Thiaucourt et al 2004). It is confined to parts of Africa (OIE 2006). CBPP is a serious disease causing significant economic losses where it does occur.

The incubation period of the disease is between 3 weeks and 3 months (Brown 1998; Thiaucourt et al 2004) and for the purposes of the *OIE Terrestrial Animal Health Code* is given as 6 months (OIE 2006a). Disease spreads by droplet infection over distances of 20 meters or more (Thiaucourt et al 2004). It 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 such as the complement fixation test or ELISA (Thiaucourt 2004). A high specificity and sensitivity is claimed for serological tests and PCR.

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 interesting to note that the organism has been isolated from semen of clinically healthy European bulls and bulls with seminal vesiculitis (Goncalves 1994; Stradaoli et al 1999).

### **32.1.5 Hazard identification conclusion**

*Mycoplasma mycoides mycoides* SC causes an exotic notifiable disease. Therefore for the purposes of this risk analysis it is classified as a potential hazard in the commodity.

## **32.2 RISK ASSESSMENT**

### **32.2.1 Entry assessment**

#### **32.2.1.1 Semen**

There are no reports of the 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 (Stradaoli 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. However, the likelihood that semen could be contaminated with virulent *Mycoplasma mycoides mycoides* SC is considered to be non-negligible.

#### **32.2.1.2 Embryos**

Nothing is known about the ability of embryos to transmit CBPP. It is therefore assumed in this risk analysis that the likelihood of transmission by embryo transfer is unlikely but non-negligible.

### **32.2.2 Exposure assessment**

Since imported germplasm would be inseminated or transferred to susceptible recipients the likelihood of exposure is considered to be non-negligible.

### **32.2.3 Consequence assessment**

The insemination or transfer of germplasm infected with *Mycoplasma mycoides mycoides* 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; Stradaoli et al 1999). Furthermore, *Mycoplasma mycoides mycoides* LC adheres to and infiltrates the zona pellucida (Sylla et al 2005) of embryos. The organism has been listed as one that could be transmitted by semen (Hare 1985). It is therefore assumed that intrauterine exposure to the organism could result in infection of the recipients.

Infection of recipients could result in cases of CBPP developing after an incubation period that could be prolonged for several months (Brown 1998; OIE 2006a; Thiaucourt et al 2004). The disease could spread by contact between cattle and lead to the establishment of an economically important disease in New Zealand, which could affect both individual farmers and trade in live animals. It would also be likely to result in an expensive campaign to eradicate the disease.

The organism is not known to cause any disease in humans and there would be no consequences for human health.

The organism causes CBPP only in cattle and mild infections in buffalo (Thiaucourt et al 2004). Therefore there would be no consequences for wild or feral animals or the environment.

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

#### **32.2.4 Risk estimation**

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and *Mycoplasma mycoides* subsp. *mycoides* SC is classified as a hazard in the commodity. Therefore, risk management measures are justified.

### **32.3 RISK MANAGEMENT**

#### **32.3.1 Options**

The OIE *Code* gives recommendations for the importation of embryos from infected and non-infected countries. It gives no recommendations regarding semen, however it specifies that the semen used to fertilise embryos should be from donors of equal health status to the embryo donors (OIE 2006a). The OIE recommendations for embryos could be used as a basis for formulating the risk management measures that should be used for the importation of 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. The donors could 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.

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

- Donors of germplasm could be required to originate from a country or zone that is free from CBPP.
- 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.
- 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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## 33 OTHER MOLLICUTES INFECTIONS

### 33.1 HAZARD IDENTIFICATION

#### 33.1.1 Aetiological agent

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

Genus: *Mycoplasma*

Genus: *Ureaplasma*

Genus: *Acholeplasma*

#### 33.1.2 OIE list

Not listed.

#### 33.1.3 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), *Mycoplasma alkalescens* (Brookbanks et al 1969), *Mycoplasma arginini* (Belton 1990; Belton 1996), *Mycoplasma 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 *Mycoplasma 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 are considered to be exotic:

*Mycoplasma bovis*, *Mycoplasma bovis*, *Mycoplasma mycoides*, *Mycoplasma californicum*, *Mycoplasma canadense*, *Mycoplasma group 7*, *Acholeplasma axanthum*, *Acholeplasma modicum*, and *Ureaplasma diversum*

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

### 33.1.4 Epidemiology

There are at least 124 species in the *Mycoplasma* genus, 8 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 some 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 e.g. *Mycoplasma mycoides mycoides* SC in cattle and *Mycoplasma capricolum capripneumoniae* in goats. Many species are best thought of as opportunistic pathogens. In addition to these problems they are sometimes difficult to culture and to classify and there have been some confusing changes to the taxonomy of the organisms. The number of organisms in the group is gradually increasing and it is unclear whether these are truly new organisms or were present in the past but wrongly typed or not typed. For these reasons older literature cannot always be accepted as being completely reliable. Basic information such as incubation periods, how long animals remain carriers for etc is often not available. Finally since the amount of work done to diagnose these infections in New Zealand may not be optimal, a statement that "the organism has not been described in New Zealand", has a clearly different meaning from a statement that "an organism is absent from or exotic to New Zealand".

*Acholeplasma* spp. are not significant veterinary pathogens (Anonymous 2004). Therefore, *Acholeplasma* spp. are not considered further in this document.

*Mycoplasma mycoides mycoides* SC is exotic in Australia, Canada, the USA and the EU and therefore not considered further.

*Ureaplasma* spp. have been isolated in New Zealand from bovine semen, sheath washings, and the female genital tract (Hodges and Holland 1980; Thornton and Wake 1997), but were not identified to species level. *Ureaplasma diversum* will therefore be regarded as an exotic species in this risk analysis.

*Mycoplasma bovis* is a common isolate of the urogenital tract of cows and bulls (Trichard and Jacobsz 1985). The organism has been associated with granular vulvovaginitis, necrotizing endometritis, seminal vesiculitis, and poor sperm motility but it is also commonly isolated from the lower reproductive tract of normal animals (Irons et al 2004).

*Mycoplasma bovis* was first isolated in the USA in 1961 and spread to many countries between 1970 and 2000 (Nicholas and Ayling 2003a). It was the *Mycoplasma* species most commonly isolated in Britain between 1990 and 2000 (Ayling et al 2004). Most isolations were from the lung or upper respiratory tract. It also occurs commonly in France (Le et al 2002). 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 2003a). It has been associated

with mastitis (Gonzalez et al 1992; Kirk et al 1997; Pfitzner 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).

*Mycoplasma canadense* has frequently been associated with mastitis but has also been isolated from normal milk (Ball and Mackie 1986; Baungartner 1999; Infante-Martinez et al 1999; Kaur and Garg 2000; Kirk et al 1997; Mackie et al 2000). Mastitis has been produced by experimental infection with this organism (Ball and Mackie 1986). It has also been isolated from semen and preputial washings (Ball 1990; Ball et al 1987b) and was associated with vulvitis in a heifer (Gilbert and Oettle 1990). However, intrauterine inoculation of the organism into adult cows did not cause lesions or lasting infections (Ball et al 1990; Ball et al 1987a).

*Mycoplasma californicum* has been associated with mixed infections of *Mycoplasma canadense* and *Mycoplasma californicum* in cases of mastitis (Infante-Martinez et al 1999; Mackie et al 2000). It has also been isolated from udders of dry cows (Mackie et al 1986), bovine fetuses (Boughton et al 1983), and from bull semen (Friis and Blom 1983).

*Mycoplasma* group 7 organisms have been associated with polyarthritis, mastitis and aborted fetuses (Hum et al 2000; Shiel et al 1982), particularly in Australia. They have also been isolated from cervicovaginal mucous and uterine discharge in buffaloes with a history of abortion (Pal et al 1984) and from preputial washings of male buffaloes (Katoch et al 1984). The organisms have also been isolated from urogenital tracts of cattle and aborted fetuses and from normal cows (Irons et al 2004).

*Ureaplasma diversum* has been associated with granular vulvovaginitis, endometritis, salpingitis, seminal vesiculitis, granular balanoposthitis, and aborted fetuses, but has also been isolated from normal cattle (Irons et al 2004). It was isolated from five aborted fetuses and four calves that were born prematurely and died. The isolated strain was inoculated onto the vulva of a virgin heifer and caused profuse purulent discharge (Ruhnke et al 1984). In Denmark, *Ureaplasma* spp. was the most frequent isolate from the urogenital tract in outbreaks of granular vulvovaginitis (Friss and Krog 1983). Le Grande isolated the *Ureaplasma diversum* from 74% of semen samples and 40% of normal cattle and found no association between granular vulvovaginitis or breeding performance and infection with the organism (Le Grand et al 1995). In a large experiment in a group of beef heifers, most showed signs of vulvovaginitis before breeding and 44% were positive for *Ureaplasma diversum* (Rae et al 1993).

Other organisms listed in Section 33.1.3 are considered less pathogenic. *Mycoplasma alkalescens*, *Mycoplasma arginini* and *Acholeplasma* spp. do not cause clinical disease. In attempts to transmit the organism experimentally, *Mycoplasma verecundum* did not infect gnotobiotic calves and *Mycoplasma arginini* and *Mycoplasma alkalescens* infected the lower respiratory tract of gnotobiotic calves but caused no signs of disease (Gourlay et al 1979).



### 33.1.5 Hazard identification conclusion

In the cases discussed above the relationship between the organism and the disease syndromes associated with them are usually only clear for the more pathogenic species such as *Mycoplasma bovis*. There appears to be a gradation of pathogenicity going from primary pathogens such as *Mycoplasma mycoides mycoides* SC to organisms which are clearly non pathogenic commensals such as *Acholeplasma laidlawii*, in between there are gradations of pathogens and opportunistic pathogens. The diseases or syndromes can be classified as erosion diseases causing a decline in economic efficiency which may vary from significant to minimal depending on the species and the circumstances. Since there is no justification for importing organisms that may be opportunistic pathogens, it would be reasonable to consider excluding all exotic Mollicutes that are known to infect animals. The following organisms are therefore considered to be potential hazards in the commodity:

*Mycoplasma bovis genitalium*  
*Mycoplasma bovis*  
*Mycoplasma verecundum*  
*Mycoplasma californicum*  
*Mycoplasma canadense*  
*Mycoplasma* group 7  
*Ureaplasma diversum*

## 33.2 RISK ASSESSMENT

### 33.2.1 Entry assessment

A number of studies have demonstrated that *Mycoplasma* species readily attach to zona pellucida and are not efficiently removed by washing (Bielanski et al 2000; 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 *Ureaplasma* spp. (Bielanski et al 2000; Bielanski et al 1989). *Ureaplasma* spp. have also been demonstrated to attach to zona pellucida and to morula (Britton et al 1989; Britton et al 1987). Embryos of mice that had been infected intraperitoneally with *Mycoplasma pulmonis* were contaminated and washing of the embryos did not remove the organism (Hill and Stalley 1991). The likelihood that germplasm will contain Mollicutes is considered to be non-negligible.

### 33.2.2 Exposure assessment

Since imported germplasm will be inseminated or transplanted into susceptible females the likelihood of exposure is considered to be non-negligible.

### 33.2.3 Consequence assessment

Heifers inseminated with semen contaminated with *Mycoplasma bovis* became infected with the organism and developed granular vaginitis and their fertility was reduced (Saed and Al-Aubaidi 1983). *Ureaplasma diversum* was transmitted to heifers by insemination (Gale 1987). Ewes served by a ram infected with an *Ureaplasma* spp. became infected with the organism (Livingstone and Gauer 1982). *Ureaplasma urealyticum* was transmitted to a woman by artificial insemination (Barwin 1984). Babies born from women with vaginal infections of *Mycoplasma hominis* or *Ureaplasma urealyticum* were colonised with the organisms. The infection was transmitted from vagina to amniotic fluid and then to the child (Dinsmoor et al 1989).

In view of the above, the likelihood that Mollicute infections could be transferred from semen or embryos to susceptible New Zealand heifers or cows is considered to be non-negligible.

The effects of the introduction of new Mollicutes into New Zealand would depend on the organism introduced. *Mycoplasma bovis* is a pathogen of economic importance that causes widespread respiratory disease in calves in Europe. *Mycoplasma bovis* is involved in a variety of conditions involving the genital tracts of cattle. *Mycoplasma canadense*, *Mycoplasma bovis*, *Mycoplasma californicum*, and probably other *Mycoplasma* spp. are the cause of mastitis in dairy cows. Other species are possibly opportunistic pathogens that are involved in single or mixed infections. Therefore the introduction of new Mollicutes could have significant economic effects on the New Zealand cattle industry.

There is no evidence to suggest that the introduction of Mollicutes in semen would adversely effect the environment. The species found in cattle are not found in birds but could cause infection of wild ruminants. However, they have not been described as causing significant diseases of deer or wild goats.

*Mycoplasmas* of cattle do not infect humans. The likelihood that species introduced in cattle germplasm would have deleterious effects on human health is considered to be negligible.

In conclusion, although the introduction of new species of Mollicutes would not have deleterious effects on human health or the environment, the likely effects on bovine health and the cattle industry are considered to be non-negligible.

### 33.2.4 Risk estimation

Since the entry, exposure, and consequence assessments are non-negligible, the risk estimate for these exotic Mollicutes is non-negligible, and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

### 33.3 RISK MANAGEMENT

#### 33.3.1 Options

Testing donor animals for all of these Mollicutes is not possible because of the variety of organisms that may be involved and the lack of a suitable range of tests to detect possible carriers.

The total ban on the introduction of germplasm is not an acceptable option since the introduction of new genetic material is essential for the improvement of the New Zealand genetic base.

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

- The current IHS allows the importation of semen from Europe, North America, and Australia with no safeguards other than reliance on antibiotics in the germplasm. However, mollicutes are susceptible to only a limited number of antibiotics. The range of antibiotics that various Mollicutes are sensitive to may vary and no comprehensive data is available that covers all possibilities. Most testing has been done on *Mycoplasma bovis*. Bielanski et al found that combinations of penicillin, streptomycin, lincomycin, spectinomycin, gentamycin, and tylosin failed to inactivate *Mycoplasma bovis* on contaminated embryos (Bielanski et al 1989). In another study the sensitivity of *Mycoplasma bovirhinis*, *Mycoplasma alkalescens* and *Mycoplasma bovis* to 12 antibiotics was measured. Tiamulin was most effective and erythromycin had no effect (Hirose et al 2003). In another study many strains of *Mycoplasma bovis* were found to be resistant to tylosin, spectinomycin, lincomycin, tetracycline, and oxytetracycline (Thomas et al 2003). Twenty one field isolates of *Mycoplasma 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 *Mycoplasma bovis* enrofloxacin was found to be efficacious but acquired resistance was demonstrated to tetracycline, spectinomycin, azithromycin, and clindamycin (Francoz et al 2005). 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, negates the use of antibiotics in extender and wash solutions as a completely reliable method for sanitizing germplasm. Since information regarding the mollicutes infections is constantly changing MAF could remain flexible and regularly update recommendations as new information becomes available. In addition MAF could regularly check the literature to see whether resistance to various antibiotics has been reported, and revise the requirements for the antibiotics to be used in semen extender and embryo wash solutions as necessary.

- Although PCR methods are available for some Mollicute organisms, validated methods are not available for all organisms. For this reason culturing of germplasm and identification of any isolated organisms could be used for germplasm testing. The germplasm could remain frozen until the results of the cultural examination are complete and a decision is made about whether to allow the importation to proceed. Testing of semen and embryos could be done using several different media (Irons et al 2004). Normally germplasm would be cultured from an aliquot taken before the addition of antibiotics.
- Another less rigorous option would be to culture germplasm after addition of antibiotics. In this case cultures are likely to be positive from germplasm that contained organisms that are resistant to the antibiotics. However, when antibiotics are bacteriostatic growth of organisms in culture may be suppressed but they could survive in the germplasm. This latter option would allow the importation of frozen semen that has already been processed and is available “on shelf”, while providing a significant increase in biosecurity standards compared to the present practices.

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## **34 HAEMORRHAGIC SEPTICAEMIA**

### **34.1 HAZARD IDENTIFICATION**

#### **34.1.1 Aetiological agent**

*Pasteurella multocida* types B and E.

#### **34.1.2 OIE list**

Listed.

#### **34.1.3 New Zealand status**

Listed on the unwanted organisms register as an unwanted notifiable organism.

#### **34.1.4 Epidemiology**

Haemorrhagic septicaemia occurs predominantly, but not entirely, in tropical and sub-tropical countries of Asia and Africa. It has also been described in the USA, South America, and non- tropical Africa (Bastianello and Henton 2004). In Africa it is caused by *Pasteurella multocida* types B and E and in Asia by Type B (Bastianello and Henton 2004; Carter 1998).

It is predominantly a disease of cattle and buffaloes. The incubation period in naturally acquired infections is from 1-3 days (Bastianello and Henton 2004; Carter 1998; de Alwis 1992). The course varies from peracute to subacute and inapparent infections also occur. Peracute infections are characterized by sudden death while acute cases show fever, profuse salivation, nasal discharge and rapid respiration. Firm subcutaneous swellings in the submandibular region are seen in subacute cases. Untreated cases usually end fatally (Bastianello and Henton 2004). Animals that survive infection may be active carriers for 4-6 weeks and then become latent carriers. In herds recently exposed to the infection up to 23% of animals may be latent carriers and these animals may remain carriers for at least 229 days (Bastianello and Henton 2004; de Alwis et al 1990). In carriers the organism is harboured in the nasopharynx, retropharyngeal lymph nodes, and tonsils and carrier animals may periodically become active shedders of the disease (Bastianello and Henton 2004; de Alwis et al 1990). Reactivation of the active carrier state may be stress related. The organism is excreted in respiratory aerosols, saliva, urine, faeces, and milk. Transmission is by the respiratory route or on fomites.

Resistance to antibiotics has not been described and treatment with sulphonamides and antibiotics is effective in controlling outbreaks of the disease (Bastianello and Henton 2004). However, treatment is ineffective for eliminating the carrier state (de Alwis et al 1990).



### 34.1.5 Hazard identification conclusion

*Pasteurella mutocida* types B and E are unwanted notifiable organisms that cause serious disease in cattle and are therefore considered to be potential hazards in the commodity.

## 34.2 RISK ANALYSIS

### 34.2.1 Entry assessment

Germplasm would not be collected from animals that are showing signs of haemorrhagic septicaemia. Since the incubation period is 1-3 days, animals that were in the incubation period at the time of germplasm collection would become clinically apparent before germplasm was exported to New Zealand. In carriers of infection the organism is located in the nasopharynx, adjacent lymph nodes and tonsils. Reactivation is unlikely to result in septicaemia or excretion in the germplasm and no references describing excretion of the agent in germplasm, by carriers, were found.

Resistance to antibiotics has not been described and antibiotics are added to semen extenders and used in washing fluids during the preparation of embryos.

For the above reasons the likelihood that the agent would be present in imported germplasm is considered to be negligible.

### 34.2.2 Risk estimation

Since the likelihood of the entry of the organism in germplasm is considered to be negligible, the risk estimate is negligible and *Pasteurella mutocida* types B and E are not considered to be hazards in the commodity. Therefore, risk management measures are not justified.

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## 35 SALMONELLOSIS

### 35.1 HAZARD IDENTIFICATION

#### 35.1.1 Aetiological agent

There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2004). Most of these belong to the species *enterica* and the subspecies *enterica* and if correct conventions are used, the names such as *dublin* and *typhimurium*, which do not have species status, 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 italicised and abbreviated as though the serovar had species status e.g. *Salmonella typhimurium*.

This analysis is concerned mainly with two important serovars: *Salmonella dublin* and *Salmonella typhimurium* but it also covers other exotic serovars.

Within each serovar there are multiple strains which can be identified by phage typing. In the case of *Salmonella typhimurium*, only the definitive phage type (DT) 104 is specifically considered in this analysis. *Salmonella typhimurium* DT104 is of particular significance because it exhibits multiple resistance to the common mainline antibiotics and is a threat to human and animal health (Hogue et al 1997; Jones et al 2002). It is now widely distributed in the world.

#### 35.1.2 OIE list

Bovine salmonellosis is not a listed disease in the *OIE Terrestrial Animal Health Code*. However, 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 2004).

#### 35.1.3 New Zealand status

*Salmonella dublin* is listed on the unwanted organisms register as an unwanted notifiable organism. *Salmonella typhimurium* is endemic in New Zealand but phage type 104 has only occurred rarely in humans and once in a dog and is classified in the category of “other unwanted organisms”. *Salmonella* spp. exotic to New Zealand are classified as other exotic species on the unwanted organisms register.

#### 35.1.4 Epidemiology

*Salmonella* spp. isolated in New Zealand from humans and animals are identified to serovar and phage type by the Environmental Science and Research (ESR) laboratory and recorded on a database (ESR 2003 and 2004b).

Information in this section relates mainly to *Salmonella typhimurium* and *Salmonella dublin* which are the most common *Salmonella* spp. isolated from cattle overseas.

*Salmonella dublin* has not been isolated in New Zealand. In other countries, it occurs most commonly in cattle but also occurs in sheep.

*Salmonella typhimurium* is endemic in New Zealand in both animals and humans, but DT104 has only been isolated from humans four times in 2003 and twice in 2004 (ESR 2003 and 2004a; ESR 2003 and 2004b). It has also been isolated from three dogs in a household where the owners suffered from diarrhoea after returning from an overseas visit (Julian 2002). The sporadic occurrence of *Salmonella typhimurium* DT104 in a few cases in humans and once in dogs does not suggest that it has become established in the New Zealand animal population.

*Salmonella dublin* and *Salmonella typhimurium* are common infections in cattle in England (Davies 2004; Hogue et al 1997; Jones et al 2002). Infection occurs mainly in calves but also occasionally in adult cattle.

*Salmonella* infection is mainly 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 their ingestion (Blood et al 1994). The intestine is initially infected and an 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 *Salmonella dublin* but also with other serovars. Animals that recover from *Salmonella dublin* infections frequently become carriers and may remain carriers for life, shedding organisms sporadically in their faeces. Animals infected with *Salmonella typhimurium* may be carriers of infection for 3-4 months.

Excreted organisms contaminate the environment and become a source of infection (Blood et al 1994). Young animals are more often affected by the disease than adults and very young animals may die after a short period of bacteraemia.

Carriers of infections can be detected by culturing faeces samples but because excretion is intermittent repeated sampling and culture is necessary (Davies 2004). Serology may be useful but is best applied on a herd basis (Davies 2004). However, it has been claimed that infections with *Salmonella dublin* can be detected in individual cattle by the ELISA (Nielsen and Ersboll 2004; Nielsen et al 2004).

### 35.1.5 Hazard identification conclusion

*Salmonella dublin* is an exotic, notifiable, zoonotic organism and *Salmonella typhimurium* type DT104 is an unwanted and zoonotic organism. Therefore these organisms are classified as potential hazards in the commodity. Other exotic *Salmonella* spp. are also considered to be potential hazards in the commodity.

## **35.2 RISK ASSESSMENT**

### **35.2.1 Entry assessment**

#### **35.2.1.1 Semen**

There is little information on the infection of semen by *Salmonella* spp. In poultry infection of semen has frequently been described and infection of the oviduct and eggs is common. However, extrapolation should not be made from birds to cattle. Infection of bulls with *Corynebacterium pyogenes* resulted in secondary infection of the reproductive tract with *Salmonella morbificans* which had been present in the alimentary tract (Boryczko and Furowicz 1971). Since septicaemia occurs during *Salmonella* infections the organism may infect semen. Semen could also become contaminated by faeces particularly in animals that have diarrhoea and have soiled skin and hair or wool with infected faeces.

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

#### **35.2.1.2 Embryos**

*Salmonella* spp. are excreted in vaginal discharges following abortions. Furthermore since *Salmonellae* are frequently excreted in faeces, contamination of semen or embryos with faeces is possible. IETS does not list *Salmonella* spp. in any risk category, thereby indicating that work on the transfer of the organism by embryo transfer has not been done (IETS 2004). 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 germplasm. The likelihood of entry of *Salmonella* spp. in embryos is therefore considered to be non-negligible.

### **35.2.2 Exposure assessment**

Imported germplasm would be inseminated or implanted into susceptible cattle and the likelihood of exposure is considered to be non-negligible.

### **35.2.3 Consequence assessment**

The introduction of infected germplasm would be likely to result in infection of recipients, which could become carriers and excretors of organisms that may infect other in contact animals and people. The introduction and establishment of any new *Salmonella* spp. could result in spread of the organisms in New Zealand and the establishment of production limiting diseases of livestock.

The establishment of *Salmonella typhimurium* DT 104 in animal populations would constitute a source of infection for people and be of particular concern to human health because of its resistance to antibiotics (Davies 2004; Hogue et al 1997). *Salmonella dublin* is also a zoonotic organism that could cause disease in people.

There would be no particular consequences for the environment other than possibly sporadic cases of salmonellosis in wild or feral animals such as feral deer and goats. Infected feral animals could be a source of infection for domestic animals. Infection of wild birds with *Salmonella* spp has been described (Alley et al 2002; Pennycott 2001). Therefore wild and feral birds could become infected, but infection with newly introduced species is no more likely to occur than with species already present in the country.

In conclusion, the introduction of infected 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 considered to be non-negligible.

#### **35.2.4 Risk estimation**

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for *Salmonellae* is non-negligible, and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

### **35.3 RISK MANAGEMENT**

#### **35.3.1 Options**

Many strains of *Salmonella* spp. are resistant to a wide range of commonly used antibiotics (Jones et al 2002; Wray et al 1991) and therefore the use of antibiotics in semen diluents or embryo wash fluids cannot be relied upon to eliminate *Salmonella* spp. from semen or embryos. Extenders used to dilute turkey semen failed to eliminate *Salmonella* (Donoghue et al 2004). Repeatedly culturing of faeces from donors to ensure that they are not carriers is a laborious and probably not completely reliable procedure. Since culture of *Salmonella* spp. from a variety of sample types is well documented (Davies 2004), 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 will be necessary to culture germplasm samples with added antibiotics. In this case it must be assumed that failure to culture organisms indicates that they are not present or have been inactivated by the antibiotics. This system is not ideal because antibiotics that are bacteriostatic may suppress growth of organisms in culture, without eliminating them. However, use of pre-enrichment medium will assist the isolation of damaged organisms by dilution of any antibiotics present and resuscitating damaged organisms (Davies 2004). This represents a practical compromise to the problems and increases biosecurity compared with the systems presently in place.

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

- 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.
- Aliquots of semen and embryos and (if available) the sediment of wash fluid from embryo processing could be cultured according to OIE recommended culture methods (Davies 2004). 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. Embryos that are substandard for use as embryos for transplantation could be used for culturing. If no substandard embryos are available then an aliquot of embryos could be used for culturing. Where pathogenic *Salmonella* spp., exotic to New Zealand are isolated importation of germplasm could be prohibited.

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## 36 LEPTOSPIROSIS

### 36.1 HAZARD IDENTIFICATION

#### 36.1.1 Aetiological agent

There are over 200 *Leptospira* serovars classified into 23 serogroups (Bolin 2004). 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*, *Leptospira pomona* etc.

#### 36.1.2 OIE list

Leptospirosis is listed by the OIE although the current *Terrestrial Animal Health Code* chapter for this disease is “under study”.

#### 36.1.3 New Zealand status

*Leptospira hardjo*, *Leptospira pomona*, *Leptospira balcanica*, *Leptospira copehageni*, *Leptospira ballum*, and *Leptospira tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). A single isolation of *Leptospira australis* has been reported from a human (Thompson 1980). In humans serological diagnosis indicates that five of the species endemic in farm animals infect humans but *Leptospira balcanica* which is associated with possums has not been diagnosed in man (Anonymous 2004). Other *Leptospira* spp. are classified by MAF as “other exotic organisms”.

#### 36.1.4 Epidemiology

Leptospirosis is not a single disease but a complex of diseases caused by at least 200 different organisms. Many of the *Leptospiras* are adapted to a particular host species (Farina et al) in which an almost symbiotic relationship has been formed. Species other than the maintenance host may be more resistant to infection but if infected are more susceptible to disease. *Leptospira hardjo*, for example, infects most cattle in an endemic situation but only causes occasional cases of disease in cattle. However, it may be responsible for causing sporadic cases of disease in other species such as man (accidental hosts). In maintenance hosts, *Leptospira* may localise in the kidneys and the animals may continue to excrete the organism in their urine for years. Cattle can remain carriers of *Leptospira hardjo* for at least 450 days (Hunter 2004). In New Zealand the prevalence of the disease in humans is relatively high for a temperate climate country and *Leptospira hardjo* accounts for nearly half the cases (Thornley et al 2002).

Leptospire spread in water and mud contaminated with infected urine. Infection can occur by mouth or through the skin particularly through abrasions and wounds. Diseased animals 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. The disease can be diagnosed by the isolation of the organism, but because this is a difficult process it is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low level titre indicating resolution or a chronic infection. The microscopic agglutination test is still the most commonly used test but a number of variations of ELISAs are also available. ELISAs generally lack serovar specificity (Bolin 2004). Leptospirosis is seldom the cause of economically serious disease in animals and is mainly of concern because it is a zoonotic disease that occasionally causes serious disease in humans (Thornley et al 2002).

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

### **36.1.5 Hazard identification conclusion**

*Leptospira* spp. other than the 6 endemic species are exotic, zoonotic organisms and are classified as potential hazards in the commodity.

## **36.2 RISK ASSESSMENT**

### **36.2.1 Entry assessment**

#### **36.2.1.1 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). However, *Leptospira* spp. are sensitive to the antibiotics normally used in the preparation of diluted semen and properly prepared semen is unlikely to infect recipients. Therefore, for the purposes of international trade treatment of animals or animal germplasm with suitable antibiotics provides an efficient means of controlling the spread of exotic serovars. OIE recommendations for international trade for ruminants, pigs and horses are that live animals should be treated for leptospirosis with a suitable antibiotic and that germplasm and semen should be prepared according to OIE recommendations which include the use of suitable antibiotics (OIE 2003). For many years New Zealand has successfully adopted these policies with regard to importation of live animals and germplasm. The risk of release is dependant upon the efficacy of the antibiotics used in semen preparation rather than the absence of the organism in the semen. The likelihood of entry is therefore considered to be low but non-negligible.

#### **36.2.1.2 Embryos**

*Leptospira* were found in the genital tract of heifers experimentally infected with *Leptospira hardjo* (Bielanski et al 1998), but *Leptospira* could not be cultivated from in

*vitro* fertilized embryos from the heifers. *Leptospira hardjo* were found to adhere to and penetrate into the pores of the zona pellucida of embryos exposed to them *in vitro* (Bielanski and Surujballi 1996). However, when cultured in antibiotic containing medium, *Leptospira hardjo* could not be isolated from the embryos whereas they could be isolated from controls cultured in medium containing no antibiotics. When embryos were transplanted into recipient heifers *Leptospira hardjo* was not transmitted to the recipients or their progeny (Bielanski and Surujballi 1996). The risk of release is dependant upon the efficacy of the antibiotics used in embryo preparation rather than the freedom of the embryos from infection. The likelihood of entry is therefore considered to be low but non-negligible.

### 36.2.2 Exposure assessment

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

### 36.2.3 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 dependant on the particular *Leptospira* serovar being one to which cattle are susceptible. If an infected recipient is able transmit the organism to suitable maintenance hosts during the period it is 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 for contact with infected animals. Some serovars are not important as human pathogens e.g. in New Zealand *Leptospira 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 (Anonymous 2004).

There are not likely to be noticeable consequences for feral or wild animals but some species such as *Leptospira gipputyphosa*, *Leptospira canicola*, *Leptospira sejroe*, and *Leptospira saxkoebing* are species that 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 considered to be non-negligible.

### 36.2.4 Risk estimation

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

## 36.3 RISK MANAGEMENT

### 36.3.1 Options

Because of the occurrence of long term carriers of infection, quarantine is not a suitable option. The following options could be considered in order to effectively manage the risk.

- 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.
- 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 recognize all serovars has not yet been achieved.
- Antibiotics could be used in the preparation of semen and embryos or for the treatment of donors. Germplasm could be prepared according to the recommendations of *OIE Terrestrial Animal Health Code* (OIE 2006a; OIE 2006b) and IETS (IETS 2004) including the use of suitable antibiotics in semen diluents and embryo washing media.

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## 37 ANAPLASMOSIS

### 37.1 HAZARD IDENTIFICATION

#### 37.1.1 Aetiological agents

*Anaplasma marginale*, *Anaplasma centrale*, and *Anaplasma caudatum*.

#### 37.1.2 OIE list

Listed.

#### 37.1.3 New Zealand status

Listed on the unwanted organisms register as exotic, notifiable organisms.

#### 37.1.4 Epidemiology

Anaplasmosis is a tick-borne disease of cattle caused by *Anaplasma marginale*. *Anaplasma centrale* is of very low pathogenicity and is widely used as a vaccine (Potgieter and Stoltz 2004). *Anaplasma caudatum* is found in North America and causes mild to severe disease.

The disease is widespread in the world but does not occur in most of Europe apart from the Iberian peninsula (OIE 2006). It is mainly confined to areas where suitable tick vectors are present but the role played by mechanical vectors may vary from one area to another.

Anaplasmosis is transmitted predominantly by ixodid ticks (hard ticks) but can also be transmitted by the argasid tick (soft tick) *Ornithodoros savignyi* (Potgieter and Stoltz 2004). Transmission in ticks is mainly transstadial but there have been occasional reports of transovarial transmission (Potgieter and Stoltz 2004). As many as 14 tick species from the genera *Boophilus*, *Rhipicephalus*, *Hyalomma*, *Ixodes*, and *Dermacentor* have been described as capable of transmitting the disease, but the validity of all cases has been questioned (McElwain 2004). The disease can also be transmitted mechanically by biting flies such as *Stomoxys calcitrans*, Tabanidae and mosquitoes of the genus *Psorophora* and other biting insects (McElwain 2004; Potgieter and Stoltz 2004). It is believed that for successful mechanical transmission to occur the time lapse between feeds on different animals should not be longer than a few minutes and that mechanical transmission is inefficient (Potgieter and Stoltz 2004). Endemic areas of anaplasmosis are therefore generally restricted to areas where vector ticks are present.

Young calves from both infected and non-infected cows are highly resistant to the infection up to the age of 6 months. Recovered animals remain life-long carriers of the organism and immune to reinfection. This allows immune populations of cattle to develop in endemic areas. Spillover of vectors from these areas into neighbouring areas

in favourable seasons, may result in outbreaks of disease in susceptible cattle (Potgieter and Stoltz 2004). Transmission of the organism can also occur iatrogenically when instruments or needles become contaminated with blood (e.g. when inoculating, castrating, dehorning, ear tagging etc.).

If autosterilisation occurs, animals again become susceptible to infection. Animals that are cleared of infection by chemotherapy remain resistant to clinical disease following exposure for variable periods up to 30 months (Potgieter and Stoltz 2004). The incubation period (prepatent period) following intravenous inoculation of infected blood may be as short as 4 days but is usually 3-5 weeks and may exceed 3 months (Potgieter and Stoltz 2004).

### **37.1.5 Hazard identification conclusion**

*Anaplasma* spp. are exotic notifiable organisms that may be carried by and cause disease in cattle. Therefore, *Anaplasma* spp. are considered to be potential hazards in the commodity.

## **37.2 RISK ASSESSMENT**

### **37.2.1 Entry assessment**

#### **37.2.1.1 Semen**

*Anaplasma marginale* caused a marked deterioration in semen quality and loss of libido in bulls but *Anaplasma marginale* was not found in the semen (Swift et al 1979). Similarly infection of rams with *Anaplasma ovis* caused deterioration of semen quality in rams which resolved after treatment of the disease (Kumi-Diaka et al 1988). However, *Anaplasma* spp. are not known to be excreted in or transmitted by semen. Transmission is only known to occur through insect vectors or accidental or experimental transfer of blood. The likelihood that *Anaplasma* spp. will be introduced in imported semen is considered to be negligible.

#### **37.2.1.2 Embryos**

The organism can be transmitted from cow to calf *in utero* (Potgieter and van Rensburg 1987). Calves infected *in utero* are born as immune carriers of infection. However, no literature was found that indicates that the organism can be transmitted by embryo transfer. The only known methods of transmission of the organism are transmission by vectors, *in utero* transmission or iatrogenic transmission by blood. The likelihood that the organism could be transmitted in embryos is therefore considered to be negligible.

### **37.2.2 Risk estimation**

Since the likelihood of entry of the organism in imported semen or embryos is considered to be negligible, risk estimated is negligible and *Anaplasma* spp. are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

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## 38 CHLAMYDIOSIS

### 38.1 HAZARD IDENTIFICATION

#### 38.1.1 Aetiological agent

*Chlamydomphila abortus* and *Chlamydomphila pecorum*.

#### 38.1.2 OIE list

Ovine chlamydiosis is listed.

#### 38.1.3 New Zealand status

*Chlamydomphila abortus* is listed on the unwanted organisms register as an unwanted notifiable organism.

*Chlamydomphila pecorum* has been isolated in New Zealand (Mackereth and Stanislawek 2002).

#### 38.1.4 Epidemiology

Enzootic abortion caused by *Chlamydomphila abortus*, is primarily a disease of sheep and goats (Aitken 1983), but it also infects cattle, causing a disease termed epizootic bovine abortion.

Transmission probably occurs by the faecal-oral and venereal routes. Persistent infections are common. Storz described persistent infection of male accessory glands and presence of *Chlamydomphila abortus* in semen (Andersen 2004). Ewes that have aborted remain long term intestinal carriers (Aitken 1983) and may also be chronically infected in their reproductive tracts (Andersen 2004; Papp et al 1994; Papp et al 1998). It is likely that the position is similar in cattle. Bulls may remain carriers for at least 18 months (Domeika et al 1994).

In *Chlamydomphila abortus* infections the incubation period is variable. Some animals become infected in one season and 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 (Aitken and Longbottom 2004; Andersen 2004; Dagnall and Wilsmore 1990; Domeika et al 1994; Szeredi and Bacsadi 2002; Thomas et al 1990). *Chlamydomphila abortus* and *Chlamydomphila pecorum* can 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 *Chlamydomphila abortus* and *Chlamydomphila pecorum* and some Gram negative organisms (Aitken and Longbottom 2004).

### **38.1.5 Hazard identification conclusion**

*Chlamydomphila abortus* is an exotic, notifiable disease of cattle and is considered to be a potential hazard in the commodity.

## **38.2 RISK ASSESSMENT**

### **38.2.1 Entry assessment**

#### **38.2.1.1 Semen**

Bulls and rams may excrete the *Chlamydomphila 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 semen imported into New Zealand could contain *Chlamydomphila* spp. is considered to be non-negligible.

#### **38.2.1.2 Embryos**

It was 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). Since similar experiments do not seem to have been done in cattle it is justifiable to extrapolate from experiments in ewes. However, small numbers of animals were involved in the experiment and it cannot be taken as a definitive finding. IETS has classified the organism as a Category 4 organism for which “preliminary information has been conducted or is in progress” (IETS 2004). The safety of embryo transfer remains to be conclusively proved. Therefore, the likelihood of introducing infection with embryos is considered to be low but non-negligible.

#### **38.2.2 Exposure assessment**

Imported germplasm would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is considered to be non-negligible.

#### **38.2.3 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 this time it was likely to have been *Chlamydomphila abortus*. In addition, insemination with infected semen resulted in sero-conversion and a recovery of the organism from three out of ten ewes (Appleyard et al 1985).

Preliminary evidence suggests that insemination of ewes with properly prepared embryos derived from infected ewes, did not result in the transmission of the disease. However, since the numbers of animals used was small and similar experiments have not been done in cattle it cannot be assumed that embryo transfer using embryos from infected animals is a safe procedure.

Introduction of the organism would be likely to result in the establishment of a production limiting disease in cattle. As there appears to be little evidence for diversity between strains of *Chlamydophila abortus* associated with cattle or sheep, it is reasonable to suggest that infection could also spread from cattle to sheep, where it causes the economically important disease, enzootic abortion, in sheep.

*Chlamydophila abortus* is a zoonotic organism that may cause sporadic cases of abortion in women that have been in contact with infected ewes during the lambing season (Aitken and Longbottom 2004). Although no descriptions of transmission from cattle to women were found it is assumed that women could also be infected directly from cattle. Therefore, introduction of the disease would have consequences for human health.

As the organism infects goats and deer, feral goats, deer, and thar could be infected. However, the consequences for the environment are likely to be minor since it is a disease that is associated with intensive farming and is unlikely to become a problem in free ranging wildlife. It is not known whether the organism could infect any of New Zealand's indigenous or feral animals but because it is a disease associated with intensive farming, the consequences are therefore likely to be negligible.

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 considered to be non-negligible.

#### **38.2.4 Risk estimation**

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

### **38.3 RISK MANAGEMENT**

#### **38.3.1 Options**

The *OIE Terrestrial Animal Health Code* relating to *Chlamydophila abortus* provides guidelines for safe trade of sheep semen but not for trade in embryos or for trade in cattle germplasm. IETS has classified *Chlamydophila abortus* (*Chlamydia psittaci*) in Category 4 which is a category of organism for which "Preliminary information has been conducted or is in progress" (IETS 2004). Therefore it is an organism for which the

likelihood of transmission by embryos is non-negligible. No other information on embryo transfer, relating to this organism, could be found. No information was found about transmission by cattle embryos. Therefore similar precautions need to be taken for both semen and embryo donors.

Since infected animals may remain long term carriers of infection, quarantine of donors is not considered to be a viable option.

Although criteria have been defined by OIE for sheep flocks that are considered to be free from enzootic abortion, no such definition is available for cattle herds and it is unlikely that cattle herds will be located in endemic countries that could be classified as *Chlamydia*-free. However, the disease is not of major economic importance in cattle and not considered in the OIE *Code*.

For these reasons testing of individual animals by a serological test could be used. Individual donors could be tested serologically using a sensitive serological test, 3 weeks after germplasm collection. Alternatively, aliquots of semen and embryos/washing fluid could be tested for *Chlamydia* by culture, PCR or antigen detection ELISA (Aitken and Longbottom 2004).

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

- Donors could be selected from animals that have been resident since birth or for the previous 2 years in a country or zone that is free from *Chlamydia abortus* based on no laboratory confirmation of infection in any species for at least two years.
- Individual donors could be tested serologically using an OIE recommended test for *Chlamydia abortus*, 2-3 weeks after germplasm collection.
- Aliquots of semen and embryos could be tested for *Chlamydia abortus* by PCR or antigen detection ELISA, with negative results. In the case of embryos, wash fluid and embryos that are substandard and not suitable for export, could be used for testing.

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## 39 Q FEVER

### 39.1 HAZARD IDENTIFICATION

#### 39.1.1 Aetiological agent

*Coxiella burnetii*.

#### 39.1.2 OIE list

Listed.

#### 39.1.3 New Zealand status

Listed on the unwanted organisms register as an unwanted notifiable organism.

#### 39.1.4 Epidemiology

Q fever occurs worldwide with the exception of New Zealand (Worthington 2001) 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 the infections are of minimal economic importance and rarely cause disease, but it is a zoonotic organism that 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 flu-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). It causes sporadic abortions in both humans and animals (Hatchette et al 2003; Raoult et al 2002).

Transmission frequently occurs from contact with infected uterine discharges and placentae 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 ticks from 11 genera can be infected (Kelly 2004) and their dried faeces forms dust that can contaminate animals' coats. Infected cattle shed the organism after successive parturitions 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 cattle will develop antibody within a similar time interval after infection.

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

#### **39.1.5 Hazard identification conclusion**

*Coxiella burnetii* is an exotic, notifiable, and zoonotic organism. Therefore, for the purposes of this analysis it is considered to be a potential hazard in the commodity.

### **39.2 RISK ASSESSMENT**

#### **39.2.1 Entry assessment**

##### **39.2.1.1 Semen**

The organism is excreted in semen of bulls and mice (Kruszewska and Tylewska-Wierzbanowska 1997; Kruszewska and Tylewska-Wierzbanowska 1993). Therefore the likelihood of entry is considered to be non-negligible.

##### **39.2.1.2 Embryos**

No reports were found about Q fever transmission by embryo transfer. Since *Coxiella burnetii* is frequently isolated from placentas and fetuses (Hatchette et al 2003; Marin and Raoult 1999; Marrie 1990), it is possible that the genital tract of female animals could be infected and that embryos could be contaminated. The likelihood that embryos could be infected with *Coxiella burnetii* is considered to be low but non-negligible.

#### **39.2.2 Exposure assessment**

Imported germplasm would be inseminated or transplanted into susceptible recipients. Therefore, the likelihood of exposure is considered to be non-negligible.

#### **39.2.3 Consequence assessment**

*Coxiella burnetii* can be transmitted venereally in mice (Kruszewska and Tylewska-Wierzbanowska 1993) and probably in humans and cattle (Kruszewska et al 1996; Milazzo et al 2001; Tylewska-Wierzbanowska et al 1991). Therefore, it is probable that sexual transmission can occur in cattle and insemination or transplantation of infected germplasm could result in infection of the recipients. Infected recipients would remain carriers for long periods and excrete large numbers of organisms in their birth products at parturition and in milk (Marrie 1990).

Establishment of the infection in New Zealand would be likely to have a negligible effect on the cattle and sheep industries as infection of these species is usually subclinical. It might have more important effects on the goat industry as up to 30% abortions have been recorded in some goat 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 animals including birds, and fish could be infected although these infections are likely to be sub-clinical. 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 considered to be non-negligible.

### 39.2.4 Risk estimation

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

## 39.3 RISK MANAGEMENT

### 39.3.1 Options

There are no recommendations relating to Q fever in the *OIE Terrestrial Animal Health Code*. Infected cattle would be long term carriers of infection and quarantine would not prevent the entry of the organism.

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

- 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.
- 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 of disease, any donors which are known to have previously tested positive for *Coxiella burnetii* could be excluded.
- It is noted that work is presently being done on the development of a PCR for testing semen. If this test is validated and becomes available, testing of individual batches of semen may become a possibility and could replace serological testing of bulls.

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## **40 BOVINE HAEMOBARTONELLOSIS**

### **40.1 HAZARD IDENTIFICATION**

#### **40.1.1 Aetiological agent**

*Haemobartonella bovis*.

#### **40.1.2 OIE list**

Not listed.

#### **40.1.3 New Zealand status**

Exotic (Thompson 1998). Not listed as notifiable or unwanted.

#### **40.1.4 Epidemiology**

The organism is not listed as a parasite that occurs in New Zealand (Thompson 1998). The related parasite of dogs is found in New Zealand by the same laboratories that carry out surveillance for all blood parasites. There are therefore no grounds to suspect that the organism would have been missed in the regular examination of blood smears that occurs in New Zealand laboratories. However, the organism could have been overlooked because it is usually only apparent in splenectomised cattle.

The organism is virtually a harmless organism that is only of concern where it causes mild disease in splenectomised animals. Its occurrence may be confused with *Anaplasma* spp. when examining blood smears (Potgieter 2004). Little is known about the natural transmission of the organism but it is generally assumed that it is transmitted by arthropod vectors (Potgieter 2004).

#### **40.1.5 Hazard identification conclusion**

Since *Haemobartonella bovis* causes inconsequential infections in cattle only and is of no economic importance, it is not considered to be a potential hazard in the commodity.

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## 41 BOVINE SPONGIFORM ENCEPHALOPATHY

### 41.1 HAZARD IDENTIFICATION

#### 41.1.1 Aetiological agent

A prion, which is widely accepted as being an infectious protein that contains no genetic material.

#### 41.1.2 OIE list

Listed.

#### 41.1.3 New Zealand status

Listed on the unwanted organisms register as an unwanted notifiable organism.

#### 41.1.4 Epidemiology

A major epidemic of bovine spongiform encephalopathy (BSE) began in the United Kingdom in 1986 (Hillerton 1998). The epidemic peaked in 1992 with a total of 37,490 cases (Hillerton 1998). The total number of cases in the outbreak had reached 184,131 by December 2004 but the number of annual cases had declined to 199 in 2005. Of these, 39 were confirmed cases from 156 suspects and the rest were detected in the targeted surveillance programme in which 551,000 cattle were tested (Burke 2006). This dramatic drop in case numbers indicates that the eradication methods and the premises on which they have been based are sound. The disease has spread to several European countries (Anonymous 2004). The numbers of confirmed cases that have been reported in the European Union countries varied from none in Estonia, Hungary, Latvia, and Lithuania (Anonymous 2004) to 935 in Portugal and 1,474 in Ireland. More recently a single case has been reported in Sweden (Anonymous 2006b). Cases have occurred in the USA (Anonymous 2006c) and in Canada (Anonymous 2006a).

BSE is a progressive disease of the nervous system of cattle. The disease agent is a prion which is an infectious protein that lacks any genetic material (RNA or DNA). It is a food-borne disease that is associated with feeding of protein derived from cattle to cattle. Other forms of transmission are believed to be unlikely although a few cases may be associated with vertical transmission from cow to calf (Braun et al 1998; Donnelly 1998; Donnelly et al 1997; Wilesmith and Ryan 1997; Wilesmith et al 1997).

Wells and his co-workers reported that the minimum time from experimental oral infection to detection of lesions in the brain was 32 months and the time to clinical signs developing was 35 months (Bradley and Verwoerd 2004). The incubation period can be much longer than this, with a probable upper limit of around 8 years. All cases end fatally, with the duration of signs lasting from 7 days to 14 months, but usually from 1-2 months.

The disease affects several species of animals including cats, kudu, nyala, and several species of oryx, cheetah, and puma (Kirkwood and Cunningham 1994). In man infection with the BSE agent causes variant Creutzfeldt Jakob disease (vCJD). Up to 4<sup>th</sup> November 2005 there had been 152 deaths due to or probably due to vCJD in the UK and 6 clinical cases were still alive (Anonymous 2005).

#### **41.1.5 Hazard identification conclusion**

BSE is an important exotic notifiable disease of cattle. Therefore, it is classified as a potential hazard in the commodity.

### **41.2 RISK ASSESSMENT**

#### **41.2.1 Entry assessment**

The disease occurs in the European Union countries and at extremely low prevalence in North America. However, a very extensive investigation has proved conclusively that the prion is not transmitted in either semen or embryos (Wrathall et al 2002). In the case of embryos this view has been endorsed by IETS who have classified the agent as a Category 1 agent which is “a disease or pathogenic 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” (IETS 2004). Therefore the likelihood of entry of the agent resulting from importation of semen or embryos is considered to be negligible.

#### **41.2.2 Risk estimation**

Since the likelihood of entry is assessed to be negligible, the risk estimate for BSE is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

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## 42 PREPARATION OF GERMLASM

Products derived from bovine serum such as foetal calf serum are often contaminated with bovine viral diarrhoea virus (BVDV) (Makoschey et al 2003; Yanagi et al 1996). Therefore, the likelihood that embryos that have been prepared using washing fluid that contains products derived from bovine serum may be contaminated with BVDV is non-negligible. Semen diluents are less likely to contain products derived from bovine serum.

### 42.1 SANITARY OPTIONS

The requirements of article 3.3.1.6 of the OIE *Code* could be certified. Namely, any biological product of animal origin used in the media and solutions for collection, processing, washing or storage of embryos could be required to be free of pathogenic micro-organisms. Media and solutions used in the collection, freezing and storage of embryos could be sterilized by approved methods according to the IETS Manual and handled in such a manner as to ensure that sterility is maintained.

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