

***Import Risk Analysis:***  
**Sheep and Goat**  
**Genetic Material**

10 October 2005

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***Import risk analysis: Sheep and Goat Genetic Material***

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



**10 October 2005**

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Ministry of Agriculture and Forestry  
Te Manatu Ahuwhenua, Ngaherehere  
ASB Bank House  
101-103 The Terrace  
P O Box 2526  
Wellington  
New Zealand

Telephone: +64 4 894 0100  
Facsimile: +64 4 894 0133  
Internet: <http://www.maf.govt.nz>

Pre-Clearance  
Biosecurity New Zealand

*Import risk analysis: Sheep and Goat Genetic Material*

10 October 2005

Approved for general release

Debbie Pearson  
Director Preclearance  
Biosecurity New Zealand

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## CONTRIBUTORS TO THIS RISK ANALYSIS

### 1. Primary Authors

|                 |                                                 |                                                      |
|-----------------|-------------------------------------------------|------------------------------------------------------|
| Bob Worthington | Contractor to Biosecurity<br>New Zealand        | Animal Health Consultant,<br>Wellington, New Zealand |
| Katie Owen      | Senior Adviser, Animal<br>Kingdom Risk Analysis | Biosecurity New Zealand,<br>Wellington               |

### 3. Internal Peer Review

|                 |                                                    |                                        |
|-----------------|----------------------------------------------------|----------------------------------------|
| Howard Pharo    | Team Manager, Animal<br>Kingdom Risk Analysis      | Biosecurity New Zealand,<br>Wellington |
| Erin Daldry     | Senior Adviser, Animal<br>Kingdom Risk Analysis    | Biosecurity New Zealand,<br>Wellington |
| Gillian Mylrea  | Senior Adviser, Animals<br>Import Health Standards | Biosecurity New Zealand,<br>Wellington |
| Joanne Thompson | Senior Adviser, Animals<br>Import Health Standards | Biosecurity New Zealand,<br>Wellington |
| Toni Tana       | Senior Adviser, Animal<br>Kingdom Risk Analysis    | Biosecurity New Zealand,<br>Wellington |
| Sandy Toy       | Senior Adviser, Indigenous<br>Fauna Risk Analysis  | Biosecurity New Zealand,<br>Wellington |
| Jose Derraick   | Technical Adviser, Human<br>Health Risk Analysis   | Biosecurity New Zealand,<br>Wellington |
| Doug Lush       | Senior Adviser, Human<br>Health Risk Analysis      | Biosecurity New Zealand,<br>Wellington |

### 4. External Scientific Review

|                          |                                 |                                               |
|--------------------------|---------------------------------|-----------------------------------------------|
| Judith Bourne AFFA       | Principal Veterinary<br>Officer | Animal Biosecurity<br>Biosecurity Australia   |
| Professor Colin<br>Wilks | Microbiologist                  | University of Melbourne Veterinary<br>Faculty |



## 1. EXECUTIVE SUMMARY

This risk analysis considers the risk of introduction of disease-causing organisms through the importation of sheep and goat genetic material (semen or embryos).

Eighty five disease agents that were considered in the analysis are listed in Table 2. Forty five endemic agents and one exotic agent (*Acholeplasma oculi*) that was considered to be unlikely to be pathogenic and not an economically significant disease, were excluded from further consideration. Scrapie was not included in this risk analysis as it has been the subject of a previous MAF risk analysis. Diseases caused by ectoparasites such as insects, ticks and mites and endoparasites such as roundworms and tapeworms were not considered because these parasites cannot be transmitted by semen or by embryos.

All organisms classified as exotic were subjected to more detailed analysis. For this purpose in some cases groups of disease agents from the same genera were grouped together and considered as a single group e.g. *Ehrlichia* spp., *Salmonella* spp. etc. In addition, bovine tuberculosis was considered to be a disease of concern as it is under official control in New Zealand. This resulted in a total of 34 analyses being carried out.

For each disease agent, a conclusion was reached as to whether the risk posed by the importation of semen or embryos was considered to be negligible or non-negligible. For all diseases that were posed a non-negligible risk, recommendations for risk management were made.

In 12 cases it was concluded that importation of germplasm would involve negligible risk. Many of these cases involved diseases that are transmitted exclusively by vectors that are not present in New Zealand. For the remaining cases risk management measures have been proposed. These measures generally involve quarantine procedures and or test procedures to ensure that the donors of germplasm are free from infection.

## 2. INTRODUCTION

Sheep and goat breeders have an on-going need to broaden the genetic base of New Zealand flocks. Importations of sheep and sheep germplasm have in the past generally been restricted to importation from Australia, and introductions from a limited number of other countries were permitted subjected to detailed sanitary measures that were designed to ensure that scrapie and other slow virus diseases would not be introduced, based on previous MAF risk analysis work. The importations of sheep and goat germplasm from countries other than Australia that have occurred after 1976 are summarised in Table 1.

**Table 1.** Importations of sheep and goat germplasm into New Zealand since 1976.

| Country of origin           | Date of import | Date of release | Breeds of sheep or goats                                          |
|-----------------------------|----------------|-----------------|-------------------------------------------------------------------|
| Denmark and Finland         | April 1984     | November 1990   | Oxford Down, Finnish Landrace, Texel                              |
| Denmark and Finland         | February 1986  | November 1990   | Texel, Oxford Down, Gotland, White Headed Marsh, Finnish Landrace |
| Zimbabwe                    | February 1986  | April 1993      | Angora goats, Boer goats                                          |
| Zimbabwe                    | 1989           | 1994            | Karakul                                                           |
| Israel                      | 1991           | 1994            | Awassi                                                            |
| Sweden                      | 1992           | 1996            | East Friesian                                                     |
| South Africa                | 1995           | 1999            | Angora goats                                                      |
| United Kingdom <sup>1</sup> | 1997           |                 | Transgenic sheep                                                  |
| Singapore <sup>2</sup>      | 2002           |                 | Argali                                                            |

**Notes:**

1. The importers abandoned this project and all sheep were slaughtered while still in quarantine.
2. Shipment of semen proved to be sterile when used in recipient ewes in quarantine. The remaining semen is still being held in quarantine.

### 2.1 COMMODITY DEFINITION

This risk analysis is limited to the description of the risks involved in the importation of sheep and goat embryos and semen. Risk due to importation of live animals has been specifically excluded. The analysis is limited to disease-causing organisms as defined in the Biosecurity Act. Genetic diseases and other risk factors that may be of commercial importance to importers have not been considered in the investigation. The risk analysis is qualitative.

The analysis is restricted to the risks imposed by viral, bacterial and protozoal diseases. Diseases caused by external and internal parasites are excluded because the parasites cannot be transmitted by semen or embryos. The disease scrapie has not been included in the analysis as this disease represents a special case which has been addressed separately by MAF in a previous risk analysis (MacDiarmid, 1996).

The commodities to be introduced are frozen semen and *in vivo* derived embryos from sheep (*Ovis aries*) and goats (*Capra hircus*). Semen and embryos are referred to collectively as germplasm. The commodities will:

- Be collected and processed at suitable collection centres and laboratories that have been approved for the purpose by the veterinary authority of the exporting country.
- Be processed, packaged and transported according to standards laid down in the OIE Terrestrial Animal Health Code (Anonymous, 2004) and The Research Subcommittee of the International Embryo Transfer Society (IETS) Import/Export Committee (IETS, 2002).

## **2.2 RISK ANALYSIS METHODOLOGY**

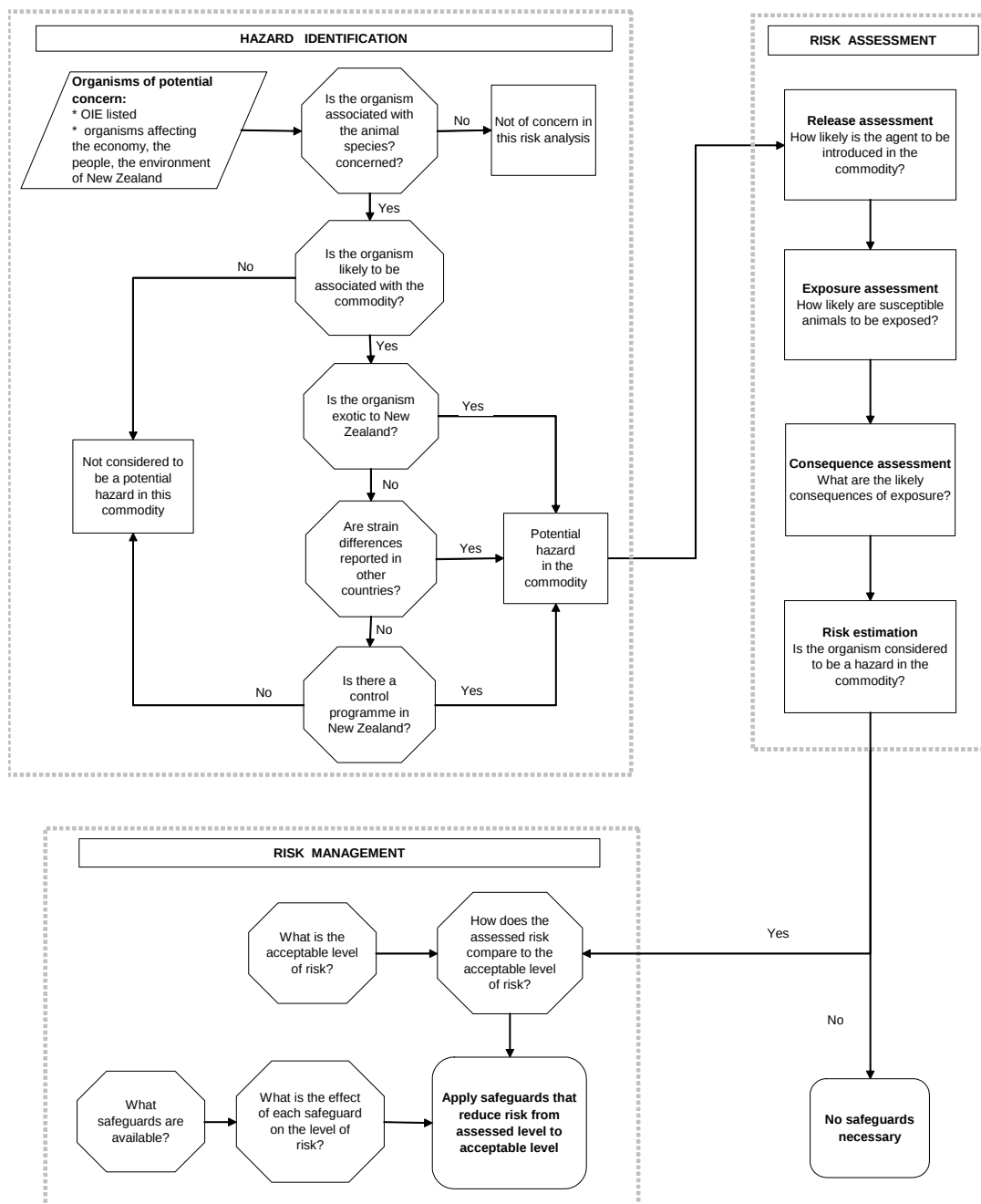
The methodology used in this risk analysis follows the guidelines in Section 1.3 of the Terrestrial Animal Health Code of the Office International Des Epizooties (Anonymous, 2004). In New Zealand, the OIE risk analysis framework is applied as described in *Import Risk Analysis. Animals and Animal Products* (Murray, 2002).

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

### **2.2.1 Hazard identification**

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 sheep or goat semen or embryos. The diseases of interest are those that could be transmitted in sheep or goat germplasm and could infect domestic, feral or wild animals that occur in New Zealand and man.

**Figure 1. The risk analysis process.**



For this risk analysis a list was made comprising all the diseases of sheep and goats that were listed by OIE in the year 2004 as well as other diseases mentioned in the following sources:

Diseases of sheep. WB Martin (Ed), Blackwell Scientific Publications, 1983, ISBN 0-632-01008-8.

Diseases of sheep. R Jensen, Lea and Febiger, 1974, ISBN 0-8121-0471-4.

Veterinary Medicine. DC Blood and OM Radostits, 7th edition, 1989, Bailliere Tindall, ISBN 0-7020-1286-6.

Infectious Diseases of Livestock. JAW Coetzer and RC Tustin (Eds), 2<sup>nd</sup> edition, 2004, Oxford University Press, Cape Town, ISBN 0-19-578202 X.

The MAF database that contains a complete listing of all diseases that appears in IHSs or in the listings of *Overseas Market Access Requirements* (OMARS) for all countries for which the information is available.

Each organism in the list was then considered and the following categories of organisms were retained in a *Preliminary Hazard List* (Table 2). .

- Organisms that were the subject of a control programme undertaken by a government department or a national, regional or small scale control programme under the Biosecurity Act.
- Organisms for which, after careful consideration the authors of the risk analysis believed that there was a valid reason to include them in the analysis. In practice this criterion resulted in the addition of only the Palyam viruses to the list.

For each of the organisms “Of concern” in Table 2, the hazard identification discusses key aspects of epidemiology, including a consideration of the following questions:

- 1) whether the various commodities could potentially act as a vehicle for the introduction of the organism,
- 2) whether it is exotic to New Zealand but likely to be present in exporting countries,
- 3) if it is present in New Zealand,
  - a) whether it is "under official control", which could be by government departments, by national or regional pest management strategies or by a small-scale programme, or
  - b) whether more virulent strains are known to exist in other countries.

For any organism, if the answers to questions one and either two or three are ‘yes’, it is classified as a potential hazard.

### 2.2.2 Risk Assessment

Under the OIE methodology, for each potential hazard, the following analysis is carried out:

#### Risk Assessment

- a) Release 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, 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 OIE methodology makes it clear that if the likelihood of release 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 release 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 release and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

### 2.2.3 Risk Management

Risk management consists of the following steps:

- a) Risk evaluation - a determination is made as to whether sanitary measures are necessary.
- b) Option evaluation - the options available for managing the risk are identified, and their risk reduction effects are considered.

- c) Recommended measures - the appropriate option or combination of options is recommended to achieve a negligible likelihood of entry, spread or establishment, while minimising negative trade effects.

Risk Communication, the final step of a complete risk analysis, is included in this document. MAF has standard procedures for consultation with the public and interested parties on all risk analyses. The OIE Terrestrial Animal Health Code, Chapter 1.3.2, Guidelines for Risk Analysis, also includes evaluation of Veterinary Services, zoning and regionalisation and surveillance and monitoring of animal health. (Anonymous, 2004) These considerations apply to individual countries and are not covered in this risk analysis as it is written for all countries. They will be taken into consideration by MAF at the time of writing an IHS for an individual country.

## 2.3 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 (Weiss as quoted by (Coetzer, 2004)), Q fever (Kruszewska and Tylewska-Wierzbanska, 1997), and maedi visna (De La Concha et al., 1996) have been found in the semen of infected animals. Although seminal excretion of infectious agents may not occur 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. However, this statement does not apply to those protozoal diseases which are known to be transmitted only by arthropod vectors.

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 are asymptomatic 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. A subcommittee of OIE, the Research Subcommittee of the International Embryo Transfer Society (IETS) 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 last updated in 2002 is published in Section 3.3.5 of the OIE Terrestrial Animal Health Code. (IETS, 2002) 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 case 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 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 should 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. In one case in the interval between publishing two editions of an authoritative text book recognised authorities changed their estimate for the incubation period for jaagsiekte from 2-3 years to 5-6months. (Verwoerd et al., 1994; Verwoerd et al., 2004) Data quoted for the period of viraemia or bacteraemia is also unreliable because of the small number 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 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 for error should 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 recommended quarantine periods are adjusted to whole weeks or months. When Import Health Standards are written for particular cases these recommended periods may be modified.

**Table 2. Preliminary hazard list.**

| Organism                                        | OIE List | Zoonotic | NZ Status                                     | Notes                                                                          | Of Concern |
|-------------------------------------------------|----------|----------|-----------------------------------------------|--------------------------------------------------------------------------------|------------|
| <b>Viruses</b>                                  |          |          |                                               |                                                                                |            |
| Akabane and related simbu viruses               | No       | No       | Exotic                                        | Many related viruses                                                           | Yes        |
| Aujeszky's disease virus                        | Yes      | No       | Exotic,                                       | -                                                                              | Yes        |
| Bluetongue virus                                | Yes      | No       | Exotic,                                       | 24 serotypes                                                                   | Yes        |
| Border disease virus                            | No       | No       | Endemic*                                      | -                                                                              | No         |
| Borna disease virus (unclassified)              | No       | Yes      | Exotic                                        | -                                                                              | Yes        |
| Bovine respiratory syncytial virus              | No       | No       | Endemic (Motha and Hansen, 1997)              | -                                                                              | No         |
| Caprine arthritis encephalitis virus            | Yes      | No       | Endemic                                       | Maedi visna virus-like strains occur - (Shah et al., 2004; Grego et al., 2005) | No         |
| (Capripoxvirus) Sheep/goat pox                  | Yes      | No       | Exotic,                                       | Various strains                                                                | Yes        |
| Coronavirus                                     | No       | No       | Endemic (Durham et al., 1979; Vermunt, 2000b) | -                                                                              | No         |
| Crimean-Congo haemorrhagic fever virus          | No       | Yes,     | Exotic                                        | 6 serogroups,                                                                  | Yes        |
| FMD virus                                       | Yes      | Yes      | Exotic,                                       | 7 serotypes multiple strains                                                   | Yes        |
| Ovine pulmonary adenocarcinoma virus            | Yes      | No       | Exotic                                        | -                                                                              | Yes        |
| Louping ill and related viruses                 | No       | Yes      | Exotic                                        | Multiple serotypes                                                             | Yes        |
| Maedi-visna lentivirus                          | Yes      | No       | Exotic                                        | CAE-like strains occur (Shah et al., 2004; Grego et al., 2005)                 | Yes        |
| Nairobi sheep disease virus and related viruses | Yes      | No       | Exotic,                                       | Ganjam, Dugbe viruses related antigenically                                    | Yes        |
| Ovine and caprine papillomaviruses              | No       | No       | Endemic (Shortridge and Cordes, 1971)         | -                                                                              | No         |
| Palyam serogroup viruses                        | No       | No       | Exotic                                        | Many serogroups                                                                | Yes        |
| Parainfluenza virus 3                           | No       | No       | Endemic*                                      | -                                                                              | No         |
| Peste des petits ruminants virus                | Yes      | No       | Exotic,                                       | Pathogenicity variation                                                        | Yes        |
| Rabies serogroup                                | Yes      | Yes      | Exotic,                                       | Several related Lyssaviruses                                                   | Yes        |
| Rift valley fever virus                         | Yes      | Yes      | Exotic,                                       | Pathogenicity variation?                                                       | Yes        |
| Rinderpest virus                                | Yes      | No       | Exotic,                                       | Pathogenicity variation                                                        | Yes        |

|                                                           |     |          |                                          |                                        |     |
|-----------------------------------------------------------|-----|----------|------------------------------------------|----------------------------------------|-----|
| Rotavirus                                                 | No  | No       | Endemic (Vermunt, 2000a)                 | -                                      | No  |
| Sheep/goat pox (Capripoxvirus)                            | Yes | No       | Exotic,                                  | Various strains                        | Yes |
| Vesicular stomatitis virus                                | Yes | No       | Exotic,                                  | 3 subtypes                             | Yes |
| Wesselsbron disease virus                                 | No  | Yes      | Exotic                                   | Multiple strains                       | Yes |
| <b>Bacteria including <i>Mycoplasma</i> spp.</b>          |     |          |                                          |                                        |     |
| <i>Acholeplasma laidlawii</i>                             | No  | No       | Endemic (Belton, 1990, 1996)             | -                                      | No  |
| <i>Acholeplasma oculi</i>                                 | -   | No       | Exotic                                   | Insignificant disease.<br>? pathogenic | No  |
| <i>Actinobacillus lignieresii</i>                         | No  | No       | Endemic *                                | -                                      | No  |
| <i>Actinobacillus seminis/Histophilus ovis</i>            | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Arcanobacter pyogenes</i>                              | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Bacillus anthracis</i>                                 | Yes | Yes      | Exotic                                   | -                                      | Yes |
| <i>Brucella melitensis</i>                                | Yes | Yes      | Exotic,                                  | Biovars 1,2,3                          | Yes |
| <i>Brucella ovis</i>                                      | Yes | No       | Endemic                                  | -                                      | No  |
| <i>Bordetella parapertusis</i>                            | No  | No       | Endemic (Anonymous, 1975a; Shrubb, 1998) | -                                      | No  |
| <i>Branhamella ovis</i>                                   | No  | No       | Endemic (Shrubb, 1998)                   | -                                      | No  |
| <i>Campylobacter fetus</i> subsp. <i>intestinalis</i>     | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Campylobacter fetus</i> subsp. <i>jejuni</i>           | No  | No       | Endemic*                                 | —                                      | No  |
| <i>Clostridium tetani</i> .                               | No  | Yes      | Endemic*                                 | strain differences                     | Yes |
| <i>Clostridium botulinum</i>                              | No  | Yes      | Endemic*                                 | strain differences                     | Yes |
| <i>Corynebacterium ovis</i>                               | No  | No       | Endemic *                                | -                                      | No  |
| <i>Corynebacterium renale</i>                             | No  | No       | Endemic. (Anonymous, 1975b, a)           | -                                      | No  |
| <i>Dermatophilus congolense</i>                           | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Dichelobacter nodosus</i>                              | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Erysipelothrix rhusiopathiae</i>                       | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Escherichia coli</i> (virulence plasmids)              | No  | Variable | Endemic*                                 | -                                      | No  |
| <i>Fusobacterium necrophorum</i>                          | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Haemophilus somni</i>                                  | No  | No       | Endemic*                                 | —                                      | No  |
| <i>Listeria monocytogenes</i>                             | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Moraxella bovis</i>                                    | No  | No       | Endemic*                                 | -                                      | No  |
| <i>Mycobacterium avium</i> subsp. <i>paratuberculosis</i> | Yes | No?      | Endemic*                                 | -                                      | No  |
| <i>Mycobacterium bovis</i>                                | Yes | Yes      | Endemic Control programme                | -                                      | Yes |
| <i>Mycoplasma agalactiae</i>                              | Yes |          | Exotic                                   | -                                      | Yes |

|                                                                |     |          |                                  |                              |     |
|----------------------------------------------------------------|-----|----------|----------------------------------|------------------------------|-----|
| <i>Mycoplasma arginini</i>                                     | No  | No       | Endemic (Belton, 1990, 1996)     | -                            | No  |
| <i>Mycoplasma. capricolum subsp. capripneumoniae</i>           | Yes | No       | Exotic,                          | -                            | Yes |
| <i>Mycoplasma conjunctivae</i>                                 | No  | No       | Endemic (Motha, 2003)            | -                            | No  |
| <i>Mycoplasma mycoides subsp. mycoides LC</i>                  | No  | No       | Endemic (Jackson and King, 2002) | -                            | No  |
| <i>Mycoplasma ovipneumoniae</i>                                | No  | No       | Endemic (Belton, 1990, 1996)     | -                            | No  |
| <i>Other Mollicutes</i>                                        | No  | No       | Exotic                           | Complex not fully understood | Yes |
| <i>Pasteurella haemolytica</i>                                 | No  | No       | Endemic*                         | -                            | No  |
| <i>Pasteurella multocida B and E</i>                           | Yes | No       | Exotic,                          | -                            | Yes |
| <i>Pasteurella multocida other than B and E</i>                | No  | No       | Endemic*                         | -                            | No  |
| <i>Pseudomonas pyocaena</i>                                    | No  | Variable | Endemic*                         | -                            | No  |
| <i>Salmonella abortus ovis</i>                                 | Yes | No       | Exotic,                          | -                            | Yes |
| <i>Salmonella. dublin,</i>                                     | No  | Yes      | Exotic,                          | -                            | Yes |
| <i>S. typhimurium DT 104</i>                                   | No  | Yes      | Exotic, rare imported cases      | -                            | Yes |
| <i>Salmonella sp</i>                                           | No  | Yes      | Endemic*                         | -                            | No  |
| <i>Staphylococcus spp.</i>                                     | No  | Variable | Endemic*                         | -                            | No  |
| <i>Streptococcus spp.</i>                                      | No  | Variable | Endemic*                         | -                            | No  |
| <b>Spirochaetes</b>                                            |     |          |                                  |                              |     |
| <i>Borrelia burgdorferi</i>                                    | No  | Yes      | Exotic                           | -                            | Yes |
| <i>Leptospira spp</i>                                          | Yes | Yes      | Exotic, 6 endemic                | >200 serovars                | Yes |
| <b>Protozoal parasites</b>                                     |     |          |                                  |                              |     |
| <i>Babesia ovis</i>                                            | No  | No       | Exotic                           | -                            | Yes |
| <i>Cryptosporidium spp.</i>                                    | No  | ?        | Endemic*                         | -                            | No  |
| <i>Eimeria spp.</i>                                            | No  | No       | Endemic*                         | -                            | No  |
| <i>Toxoplasma gondii</i>                                       | No  | Yes      | Endemic*                         | -                            | No  |
| <i>Theileria spp.</i> (sheep species)                          | No  | No       | Exotic,                          | Confused taxonomy,           | Yes |
| <i>Trypanosoma spp.</i> (Tsetse transmitted)                   | Yes | Yes      | Exotic,                          | Several species              | Yes |
| <b>Rickettsial and Chlamydial organisms</b>                    |     |          |                                  |                              |     |
| <i>Anaplasma ovis</i><br><i>A. mesaeterum</i> (Sheep species ) | No  | No       | Exotic                           | -                            | Yes |
| <i>Chlamydophila abortus</i>                                   | Yes | Yes      | Exotic,                          | -                            | Yes |
| <i>Coxiella burnetti</i>                                       | Yes | Yes      | Exotic,                          | -                            | Yes |
| <i>Ehrlichia ruminatum</i>                                     | Yes | No       | Exotic                           | -                            | Yes |
| <i>Eperythrozoon ovis</i>                                      | No  | No       | Endemic (Gill, 1990)             | -                            | No  |
| <i>Other Ehrlichia spp. of sheep</i>                           | No  | Yes      | Exotic                           | -                            | Yes |

| Fungi                    |    |    |                          |   |    |
|--------------------------|----|----|--------------------------|---|----|
| <i>Trichopyton spp.</i>  | No | No | Endemic*                 | - | No |
| <i>Zygomycosis group</i> | No | No | Endemic (Vermunt, 2000b) | - | No |

**Note:** Organisms marked with an asterisk 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 of the organisms as endemic. Two organisms (Palyam viruses and *Acholeplasma oculi*) 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, 2004).

Organisms classified as organisms of concern in Table 2 are considered in the risk analysis.

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## 3. AKABANE DISEASE

### 3.1 Hazard Identification

*3.1.1 Aetiological Agent:* Family: Bunyaviridae. 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.

*3.1.2 OIE List:* Not listed.

*3.1.3 New Zealand Status :* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004)

#### *3.1.4 Epidemiology*

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

Viruses in the Simbu-group occur endemically in large areas of Africa, Asia and the Middle East (Haughey et al., 1988; Charles, 1994; St George and Kirkland, 2004) and the related Cache Valley virus occurs in the United States of America (Edwards et al., 1989; Edwards, 1994). The incubation period (infection to start of viraemia) is from 1-6 days (St George, 1998). In non-pregnant animals infection does not lead to the development of any signs of disease and virus has been isolated from naturally infected asymptomatic sentinel cattle (Gard et al., 1989). Virus crosses from maternal to foetal circulation in infected pregnant females and causes the development of malformed lambs and kids, particularly cases of arthrogryposis and hydraencephaly (Parsonson et al., 1977; Parsonson et al., 1988; Charles, 1994; St George and Kirkland, 2004). In cattle maximal damage occurs when infection takes place at about the 12<sup>th</sup> to 16<sup>th</sup> week of gestation (St George and Kirkland, 2004). Once a foetus has become immuno-competent it can mount an immune reaction and damage is less apparent or does not occur. Infected calves are usually non viable (Charles, 1994). It can be assumed that sheep and goats will be maximally affected from some time before mid gestation until the foetus becomes immuno-competent at about the 65-70<sup>th</sup> day of gestation (St George and Kirkland, 2004). Lambs or kids born or aborted will not be contagious and will not infect vectors.

Major epidemics of foetal malformations 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 in asymptomatic animals 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.

### *3.1.5 Hazard identification conclusion*

Since Akabane and other Simbu viruses are not present in New Zealand and are unwanted organisms (Ministry of Agriculture and Forestry, 2004), they are classified as potential hazards for the purposes of this risk analysis.

## **3.2 Risk Assessment**

### *3.2.1 Release Assessment*

#### 3.2.1.1 Semen (sheep and goats)

The virus was not excreted in the semen in eight artificially infected bulls (Parsonson et al., 1981). However, this finding does not appear to have been confirmed by other workers and has not been repeated for other Simbu viruses. Therefore it is considered that the likelihood that semen of viraemic animals may contain these viruses is not negligible. However, the viraemic period for Akabane virus lasts only 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 low, but non-negligible.

#### 3.2.1.2 Embryos (sheep and goats)

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 or for an additional short period, if the uterus remains infected after virus ceases to circulate in the blood. 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, 2002). The likelihood of the disease being transmitted in embryos is therefore, low but non-negligible.

### *3.2.2 Exposure Assessment*

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

### 3.2.3 Consequence Assessment

#### 3.2.3.1 Introduction of semen and embryos from sheep and goats

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 and during this period it would not be contagious but could infect competent vectors if they exist in New Zealand. *Culicoides* spp. are not present in New Zealand (Motha et al., 1997) but it is not known whether competent mosquito vectors occur. New Zealand has remained free from the virus despite the importation of many cattle and sheep from Australia over many years. Therefore it seems likely that competent mosquito vectors may not exist although this matter is unresolved. If vectors do exist, the likelihood that a recipient of germplasm would infect competent mosquitoes during a short viraemic period is low but non-negligible.

If the disease were to become established in New Zealand it would cause outbreaks of disease characterized by the birth of deformed calves. However, as the disease became endemic and the majority of the population became immune these episodes would tend to become sporadic and the economic impact would be low as is the case in endemically infected countries. The disease is not listed by OIE as being significant for trade (Anonymous, 2004).

The consequences of infection are low but non-negligible.

#### 3.2.3.2 Other consequences

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.

#### 3.2.3.3 Consequence assessment conclusion

The risk of the disease establishing in New Zealand and the consequences of establishment are also likely to be low. Therefore overall the consequences are low but non-negligible.

### 3.2.4 Risk Estimation

Since the likelihood of release and exposure and the consequences of exposure are estimated to be non-negligible for all commodities the risk is considered to be non-negligible.

### **3.3 Risk Management**

#### *3.3.1 Risk Evaluation*

Since the risk estimate is non-negligible, risk management measures are justified.

#### *3.3.2 Option Evaluation*

##### 3.3.2.1 Risk management objective

The objective is to ensure that donors are not viraemic at the time of germplasm collection.

##### 3.3.2.2 Options available

Germplasm could be sourced from donors resident in countries that are free from Simbu viruses.

Alternatively germplasm could be collected from donors that have been resident in an area that is free from Akabane disease for 21 days or held in insect free isolation facilities for 21 days. This period will adequately cover the viraemic (3-4 days) and incubation (1-6 days) periods.

Alternatively animals could be tested serologically before and 3 weeks after germplasm collection. Animals that are positive before collection will be immune and will not excrete virus. Animals that are negative before and after collection will not have been exposed to the virus and will not excrete virus. Animals that sero-convert from negative to positive or have steeply rising titres may have been viraemic at the time of germplasm collection and their germplasm would be unsuitable for importation.

##### 3.3.2.3 Recommended sanitary measures

Donors should

- i. be resident for at least 21 days immediately before germplasm collection in a country or zone that is free from Simbu viruses; or
- ii. be held in a disease free area or in insect free premises for at least the 21 days before collection of germplasm; or
- iii. be tested, within the seven days prior to germplasm collection and again 3-6 weeks after the final germplasm collection using a Simbu-group reactive cELISA (St George and Kirkland, 2004). Semen and embryos from animals that sero-convert or have rising titres between the two tests should be disqualified from entry into New Zealand. Animals that are serologically

positive at the first test or negative at both tests are suitable for use as germplasm donors.

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## 4. AUJESZKY'S DISEASE

### 4.1 Hazard Identification

*4.1.1 Aetiological Agent:* Family; Herpesviridae; suid herpesvirus1, Aujeszky's disease virus.

*4.1.2 OIE List:* Listed

*4.1.3 New Zealand Status:* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004).

#### *4.1.4 Epidemiology*

Aujeszky's disease (pseudo-rabies) is a disease of pigs that was recently eradicated from New Zealand. It occurs world-wide except in Australia, Canada, Finland, Sweden, Denmark and the UK. Some other countries are involved in eradicating the disease (Van Oirschot, 2004). The virus can be transmitted to sheep and goats, (Herweijer and de Jonge, 1977; Baker et al., 1982; Henderson et al., 1995; Van Oirschot, 2004) and other animals by close contact with infected pigs. In animals other than pigs the disease is characterized by acute pruritis and nervous signs and is invariably fatal (Herweijer and de Jonge, 1977; Baker et al., 1982; Henderson et al., 1995; Van Oirschot, 2004). Following experimental infection in sheep, the virus was excreted in nasal discharges, but infected sheep did not infect other sheep in contact with them (Mocsari et al., 1987; Mocsari et al., 1989). Animals other than pigs are not known to carry the virus or to act as sources of infection (Van Oirschot, 2004).

#### *4.1.5 Hazard identification conclusion*

The disease has been classed as an exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004). It is therefore classified as a potential hazard for the purpose of this risk analysis.

### 4.2 Risk Assessment

#### *4.2.1 Release Assessment*

Aujeszky's disease is a rare disease in sheep and goats and only occurs when they have been in close contact with pigs. When it occurs the signs are dramatic (Herweijer and de Jonge, 1977; Baker et al., 1982; Henderson et al., 1995; 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.

#### 4.2.2 Risk Estimation

Because the release assessment is negligible according to the methodology used in this analysis (Section 4.2), risk is considered to be negligible.

#### 4.3 Risk Management

Since risk is negligible risk management measures are not required.

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## 5. BLUETONGUE

### 5.1 Hazard Identification

*5.1.1 Aetiological Agent:* Family: Reoviridae; Genus: *Orbivirus*. Bluetongue virus (BTV), there are 24 known serotypes of BTV

*5.1.2 OIE List:* Listed

*5.1.3 New Zealand Status:* Exotic, notifiable (Ministry of Agriculture and Forestry, 2004).

#### *5.1.4 Epidemiology*

Bluetongue virus (BTV) can infect many ruminant species. It occurs in most tropical and sub-tropical countries. It is absent in southern hemisphere countries south of 42° south, including New Zealand, and northern hemisphere countries north of 45° north (Osburn, 2004). The virus causes disease mainly in sheep, occasionally in goats and rarely in cattle and deer. In most other species infections are asymptomatic. It is carried by *Culicoides* spp. (midges) and outbreaks of the disease usually occur in late summer to autumn when midges are most active. Outbreaks of disease cease with the advent of winter when *Culicoides* spp. become inactive. The mortality in sheep varies from 2-30% (Verwoerd and Erasmus, 2004).

#### *5.1.5 Hazard identification conclusion*

Since bluetongue virus is an exotic notifiable organism (Ministry of Agriculture and Forestry, 2004), it is classified as a potential hazard for the purposes of this risk analysis.

### 5.2 Risk Assessment

#### *5.2.1 Release Assessment*

##### 5.2.1.1 Semen (sheep and goats)

In cattle the virus is excreted in semen only while animals are viraemic (Bowen et al., 1983; Howard et al., 1985). The presence of bluetongue virus in small ruminant semen and transmission by semen has been reported by Luedke (Hare, 1985) but no supporting references were given for this statement. Excretion of the virus in sheep and goat semen can be assumed to be confined to periods of viraemia, as in cattle. Sheep usually remain viraemic for 6-8 days and rarely up to 14 days (Verwoerd and Erasmus, 2004). However, it was reported that in Lesbos sheep and goats the viraemic period lasted up to 54 days but not up to 64 days (Koumbati et al., 1999). Most older sheep in endemic areas will be immune to the serotypes of virus circulating in the area, but young animals and animals newly imported into the endemic area are likely to be susceptible. Older sheep will also

be susceptible to new serotypes of virus introduced to an area. In summer and for a period up to 60 days after *Culicoides* spp. become inactive at the onset of winter, susceptible animals may be viraemic. The likelihood of collecting infected semen in these periods is non-negligible.

#### 5.2.1.2 Embryos (sheep and goats)

Sheep were shown to be susceptible to intrauterine infection with bluetongue virus (Gilbert et al., 1987). Bluetongue virus adhered to the zona pellucida of embryos after *in vitro* exposure to the virus, but there was no evidence of penetration of the zona pellucida (Gillespie et al., 1990). The virus did penetrate blastomeres and cause embryonic death in embryos in which the zona pellucida had been damaged. In one experiment 49 embryos from bluetongue infected ewes were transplanted into 27 recipient ewes that were serologically negative to bluetongue. Eleven pregnancies and 12 lambs resulted. None of the embryo recipients or the lambs sero-converted and BTV was not isolated from any of the lambs or recipients (Hare et al., 1988). However, Gilbert found that seroconversion and viraemia occurred in 2 out of 15 recipients of embryos that had been exposed to BTV *in vitro* (Gilbert et al., 1987). These workers had only washed the embryos 4 times instead of the 10 times recommended by IETS. It was subsequently found that clean embryos exposed to BTV could not be freed from virus by washing 10 times (Singh et al., 1997). However, BTV could not be isolated from embryos derived from infected ewes and recipients of these embryos and their progeny remained free from infection (Singh et al., 1997). These reports indicate that when embryos are exposed *in vitro* to BTV, the virus will adhere strongly to the zona pellucida and cannot be removed by washing. However, embryos from viraemic ewes are not infected and do not transmit the disease to recipients.

The IETS has placed bluetongue of sheep in Category 2, a category “for which substantial evidence has accrued to show that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer but for which additional transfers are required to verify existing data” (IETS, 2002). Bluetongue in goats is only classified in Category 4 which is a category of diseases “on which only preliminary work has been done or is in progress”. Based on the IETS classification the likelihood of transmission of the virus by sheep embryos is unlikely. However, there are still areas of doubt since embryos exposed to the virus *in vitro* cannot be freed from the virus by washing and virus can penetrate into zona pellucida damaged embryos. In the case of goats the position is less clear.

Therefore, the likelihood of both sheep and goat embryos being infected with virus is considered to be non-negligible.

#### *5.2.2 Exposure Assessment*

Imported semen and embryos will be inseminated or transplanted into susceptible New Zealand sheep or goats. Therefore the likelihood of exposure is high for germplasm from both sheep and goats.

### 5.2.3 Consequence Assessment

#### 5.2.3.1 Introduction of semen and embryos from sheep and goats

Cattle inseminated with infected semen became infected and developed viraemia (Bowen et al., 1985; Schlafer et al., 1990; Bowen and Howard, 1984). As sheep and goats are more susceptible to bluetongue than cattle it can be assumed that they would also be susceptible. Inseminated recipients are likely to become infected and may become sick or die (mortality 2-30%). Those that recover may be viraemic for a period of up to 2 months, but since bluetongue is not a contagious disease they will not transmit the disease to other small ruminants in contact with them. The virus could only be transmitted to *Culicoides* vectors. A *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al., 1991) and has continued till the present time. Seroconversion has not occurred in sentinel cattle to bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses and *Culicoides* spp. have not been found. In a typical year *Culicoides* were not found amongst 15,000 insects collected from light traps (Motha et al., 1997). Reports on the surveillance programme are published regularly in the MAF Surveillance magazine. Since New Zealand is free from *Culicoides* spp. the disease cannot establish. However, the occurrence of cases of bluetongue in New Zealand, in recipients of imported germplasm, would mean that country freedom statements for bluetongue cannot be made. This in turn means that, trade in live animals semen and embryos will be temporarily suspended with all trading partners that stipulate freedom from bluetongue in their IHSs.

The risk of establishment is negligible as long as the position with regard to the occurrence of *Culicoides* spp. in New Zealand remains stable. If the disease were to establish in New Zealand ongoing losses would be experienced due to morbidity and mortality and the need for vaccination of sheep and goats. These factors would have deleterious effects on the productivity and economic performance of the sheep and goat industries. There would be no consequences for the export of meat or wool (Anonymous, 2004).

#### 5.2.3.2 Other consequences

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

It is a disease of ruminants that affects only ruminants and there is no threat to indigenous animals or birds. Some species of deer are susceptible to the disease. The effect the virus might have on them is not known.

#### 5.2.3.3 Consequence assessment conclusion

Since occurrence of bluetongue, even only to recipients of germplasm, would have deleterious effects on the trade of live animals, semen and embryos the consequences are considered to be non-negligible.

#### 5.2.4 Risk Estimation

Because release, exposure and consequence assessments for all commodities are considered to be non-negligible the risk estimate is also non-negligible.

### 5.3 Risk Management

#### 5.3.1 Risk Evaluation

Since the risk estimation for sheep and goat semen and embryos is non-negligible, risk management measures are justified to reduce the risk to an acceptable level.

#### 5.3.2 Option Evaluation

##### 5.3.2.1 Risk Management objective

The objective is to ensure that germplasm is not collected from viraemic donor animals.

##### 5.3.2.2 Options available

The options are similar for all of the commodities. Donors that have not been exposed to *Culicoides* activity for at least 100 days before collection of germplasm will not be viraemic. Therefore the objective could be achieved by accepting germplasm only from donors that have been resident in a bluetongue free zone 100 days before germplasm collection or collecting germplasm during the winter months when *Culicoides* have been inactive for 100 days (in areas that are seasonally free from *Culicoides* activity) or holding the donors in an insect free isolation facility for 100 days. The 100 day period adequately covers the incubation period, the maximum known period of viraemia and an additional safety margin. It is also the period currently recommended by OIE for the live trade in ruminants (Anonymous, 2004). However, it is likely that the OIE will soon revise the period to 60 days in the Terrestrial Animal Health Code.

A different approach would be to test animals to prove that they are not viraemic when the germplasm is collected. This can be achieved by testing aliquots of blood by virus isolation or PCR (Anonymous, 2004) or by using serological tests to demonstrate that sero-conversion did not occur during the period of germplasm collection. Alternatively donors could be tested serologically (Anonymous, 2004) before and a suitable length of time after semen collection. The latter approach would show that infection which is followed by antibody production did not occur during the time germplasm was being collected.

##### 5.3.2.3 Recommended sanitary measures

Potential importers should be offered the following range of options that closely approximate the recommendations of OIE (Anonymous, 2004):

Donor animals should:

- i. be resident for the 100 days preceding germplasm collection in a country or zone that is free from bluetongue; or
- ii. maintained free from contact with *Culicoides* spp. for the 100 days immediately before semen collection. This should be achieved by keeping them in a *Culicoides* free area, or in a seasonally free area in which *Culicoides* are inactive, or in an insect free isolation facility; or
- iii. be tested serologically with negative results for bluetongue antibodies at least every 60 days during germplasm collection and between 28 and 60 days after collection with negative results. An OIE recommended test for the detection of sero-group antibodies should be used (Eaton, 2004); or
- iv. be tested by a virus isolation procedure or PCR on the day of commencement and conclusion of collection and at least every 7 days during collection of semen and on the day of collection of embryos.

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## 6. BORNA DISEASE

### 6.1 Hazard Identification

*6.1.1 Aetiological Agent:* Family: Bornaviridae: Genus: Bornavirus. Borna disease virus is the only virus of its family.

*6.1.2 OIE List:* Not listed

*6.1.3 New Zealand Status:* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004).

#### *6.1.4 Epidemiology*

Borna disease is a disease of horses and sheep and a variety of other animals including goats, deer and rabbits (Rott et al., 2004), lynx (Desgiorgis et al., 2000) and fox (Dauphin et al., 2001). 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 etiological 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 asymptomatic (Rott et al., 2004). Antibody to Borna disease virus has been found in humans suffering from psychosomatic disorders (Rott et al., 1985; Bode et al., 1996). 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 issue remains unresolved (Staeheli et al., 2000; Carbone, 2001).

The disease occurs most commonly in Germany and Switzerland. However serologically positive animals have also been found in Poland, the Netherlands, Switzerland and Iran (Rott et al., 2004) 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 Japan (Inoue et al., 2002) and Israel (Teplitsky et al., 2003). The virus has recently been demonstrated in cats in Britain (Reeves et al., 1998).

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 intra-axonally to the brain (Carbone et al., 1987; Morales et al., 1988; Sauder and Staeheli, 2003). Experimentally the virus can be 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 (Vahlenkamp et al., 2002; Rott et al., 2004) and natural transmission is presumed to occur by direct contact, via fomites and food, by inhalation and ingestion (Rott et al., 2004). 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 asymptomatic 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 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).

Despite the fact that the disease has been known for more than 250 years (Rott et al., 2004), knowledge about the disease is still fragmentary and incomplete. The specificity and accuracy of both RT-PCR test and antibody tests has been questioned. This makes the interpretation of the results of reports 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. Since studies using RT-PCR have not generally been confirmed by viral isolation it is not known whether closely related viruses occur and what role they might play in causing disease and stimulating antibody production.

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).

#### *6.1.5 Hazard identification conclusion*

The disease is classified as an exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004) and is therefore, classified as a potential hazard for the purposes of this risk analysis.

### **6.2 Risk Assessment**

#### *6.2.1 Release Assessment*

##### 6.2.1.1 Semen and embryos (sheep and goats)

There is nothing in the literature indicating that the disease is spread venereally. Nothing is known about the potential for the virus to contaminate semen or embryos. Most 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 sheep parallels that of rats. However, peripheral mononuclear cells of sheep can remain infected with viral RNA for years.

The likelihood that semen could be contaminated with infected mononuclear cells cannot be ignored since in some infections such as *Brucella ovis* infection large numbers of cells are found in the semen. Concomitant infections with *Brucella ovis* (or other 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 contaminate semen with virus.

Until definite information is available, the likelihood of the release of virus in semen from both goats and sheep is considered to be low but non-negligible risk.

### 6.2.2 Exposure Assessment

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

### 6.2.3 Consequence Assessment

#### 6.2.3.1 Introduction of semen and embryos from sheep and goats.

Although experimental or circumstantial evidence is lacking 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 sheep 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 sheep and cases of disease in other species and possibly man.

#### 6.2.3.2 Other consequences

The association between viral infection and the occurrence of psychosomatic diseases in humans (Rott et al., 1985; Bode et al., 1996) 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 (Desgiorgis et al., 2000; Dauphin and Zientara, 2003; 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.

#### 6.2.3.3 Consequence assessment conclusion

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.

#### *6.2.4 Risk Estimation*

Because release, exposure and consequence assessments are considered to be non-negligible for germplasm from both goats and sheep the risk is estimated to be non-negligible.

### **6.3 Risk Management**

#### *6.3.1 Risk Evaluation*

Since the risk estimate is non-negligible for all commodities, management measures should be introduced to reduce the risk to a negligible level.

#### *6.3.2 Option Evaluation*

##### 6.3.2.1 Risk Management Objective

The objective is to reduce the risk of introducing the virus in semen or embryos to a negligible level.

##### 6.3.2.2 Options available

Diagnostic methods include virus isolation (Ludwig and Kao, 1990; Rott et al., 2004) and demonstration of virus proteins or RNA (Vahlenkamp et al., 2002) in tissues. However, the interpretation of the results of tests that demonstrate viral protein or RNA in tissues is often not clear. 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 (IFA) 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 asymptomatic sheep (Muller-Doblies et al., 2003). 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.

##### 6.3.2.3 Recommended sanitary measures

- i. Sheep and goat germplasm should be imported from countries in which the disease has never been reported or;

- ii. Donors should be selected from flocks with a long history of freedom from the disease in countries in which the disease is notifiable or in which reliable histories are available and;
- iii. Aliquots of semen and embryos from each collection batch of germplasm should be inoculated intracerebrally into rabbits or cultured on cell cultures derived from embryonic rabbit or rat brain with negative results.

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## 7. CAPRIPOX (SHEEP AND GOAT POX)

### 7.1 Hazard Identification

*7.1.1 Aetiological Agent:* Family Poxviridae: Genus Capripox, sheeppox virus and goatpox virus.

*7.1.2 OIE List:* Listed

*7.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

#### *7.1.4 Epidemiology*

Capripox virus causes pox in both sheep and goats. The disease is found in Africa north of the equator, the Middle East, India, Nepal and China (Kitching, 2004). Some strains of virus are more virulent for sheep than goats and some more virulent for goats. Other strains are equally virulent for both species (Kitching and Taylor, 1985; Kitching, 2004; Kitching and Carn, 2004). The disease is spread predominantly by contact. Inhalation of excreted virus or virus contained in scab material inhaled by susceptible animals is the most probable route of infection. Virus may also be transmitted through skin wounds (Merza and Mushi, 1990). *Stomoxys calcitrans* can act as a mechanical vector of the disease (Mellor et al., 1987) but there is no evidence of insect transmission in the field (Kitching, 2004). Herding sheep and goats into crowded enclosures encourages the spread of the disease. Morbidity may be as high as 70% and mortality from 5-50% (Munz and Dumbell, 1994). In lambs mortality may reach 80-100% (Kitching, 2004). Some breeds of sheep and goats that are indigenous in endemically infected countries are more resistant than exotic breeds and present with less severe forms of the disease (Kitching, 2004). The incubation period has been described as 8-13 days (Kitching and Carn, 2004) and from 4-8 days (Merza and Mushi, 1990), but for regulatory purposes a period of 21 days is used in the OIE Terrestrial Animal Health Code (Anonymous, 2004). Initial multiplication of the virus at the site of entry precedes a primary viraemic phase that is followed by multiplication of the virus in many organs and a secondary viraemic phase leading to the development of focal skin lesions. According to Likhachev et al (Jensen, 1974) the virus was detected in blood, on days 3-9 following injection of virus into the skin. Therefore, the viraemic period lasted 6 days.

The acute phase of the disease is followed by death or recovery. Recovered animals are immune and reports of long term viraemia were not found.

#### *7.1.5 Hazard identification conclusion*

Sheep and goat pox virus is an exotic, notifiable organism (Ministry of Agriculture and Forestry, 2004). Therefore it is classified as a potential hazard for the purposes of this risk analysis.

## **7.2 Risk Assessment**

### *7.2.1 Release Assessment*

#### 7.2.1.1 Semen (sheep and goats)

The disease is listed by Hare as one that is known to be excreted in semen and could be transmitted by semen (Hare, 1985). No information is available on the transmission of the virus in embryos. It is assumed that the virus would be found in semen and embryos during the viraemic period. The closely related lumpy skin disease virus of cattle was reported by Weiss (Coetzer, 2004) as being shed in semen for 22 days. However, scabs may contain virus for more than 3 months and stables and pastures may remain contaminated for 2-6 months (Liebermann, 1989). Semen or even embryos could be contaminated by scab particles and dust during collection of germplasm. Therefore, there is a long period following a disease episode during which germplasm could be contaminated. The risk is non-negligible.

#### 7.2.1.2 Embryos (sheep and goats)

No information could be found on the transmission of the virus by sheep or goat embryos. In view of this lack of information the risk is assessed as non-negligible.

### *7.2.2 Exposure Assessment*

If infected semen or embryos were to be imported they would be inseminated into or implanted into susceptible New Zealand recipients. Therefore the risk of exposure is high.

### *7.2.3 Consequence Assessment*

#### 7.2.3.1 Introduction of semen and embryos from sheep and goats.

The consequences of exposure are similar for semen and embryos from sheep and goats. Transmission of the virus in infected germplasm could lead to development of the disease in susceptible recipients and a high mortality rate could be expected. Animals that recover from the disease will have pox lesions and scabs that may take months to resolve and during this period they could potentially infect other sheep or goats they are in contact with. This could lead to the establishment of foci of infection in New Zealand. The disease is a serious disease which if left unchecked would result in high morbidity and mortality in sheep and goats. It would have significant economic effects and have adverse effects on trade in live sheep. Wool would have to be subjected to special treatment before being exported (Anonymous, 2004)

#### 7.2.3.2 Other consequences

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

Only sheep and goats are known to be infected and there would be no consequences for other species except possibly for feral goats and thar. The effect on thar is not known but it is unlikely that contact between thar and sheep would be close enough for the virus to be transmitted.

#### 7.2.3.3 Consequence assessment conclusion

Because of the significant effects the introduction would have on the economy of the sheep sector, the consequences of exposure are non-negligible.

#### *7.2.4 Risk Estimation*

Release, exposure and consequence assessments are considered to be non-negligible for all commodities. Therefore, the risk for the introduction of goat and sheep germplasm is estimated to be non-negligible.

### **7.3 Risk Management**

#### *7.3.1 Risk Evaluation*

The risk for all commodities is non-negligible and risk management measures are justified to reduce risk to an acceptable level.

#### *7.3.2 Option Evaluation*

##### 7.3.2.1 Risk Management objective

The objective is to ensure that germplasm for export to New Zealand is not contaminated with capripox virus.

##### 7.3.2.2 Options available

Donor animals could be restricted to animals from disease free countries.

Sheep and goat pox is an easily recognised disease characterized by high fever, nasal discharge and typical pox lesions on the skin and mucous membranes. In addition the incubation period is about 8-13 days (Kitching and Carn, 2004) and the period of viraemia is about 6 days. Therefore a quarantine of donor animals for 21 days before collection of germplasm could be used to prevent the collection of germplasm from viraemic animals. To increase the certainty of identifying infected sheep when the donors are resistant indigenous sheep, sentinel sheep of an exotic breed could be kept in contact with the donors during the quarantine period.

Germplasm collection centres that are situated in sheep and goat pox free zones could be identified and used for germplasm collection. These options are in line with the recommendations of the OIE Terrestrial Animal Health Code (Anonymous, 2004).

#### 7.3.2.3 Recommended sanitary measures

Donor animals (and sentinels) should:

- i. be resident in a country that is free from the disease for at least the 21 days prior to germplasm collection; or
- ii. not have been vaccinated against capripox; and
  - a) be quarantined for the 21 days before collection of germplasm on a germplasm collection centre that is free from the disease. During this period they should be regularly inspected and remain healthy. Inspection should include careful inspection and palpation of the skin and regular taking of temperature. If indigenous breeds of sheep that are of a breed that is highly resistant to sheep pox are to be donors they should be kept in close contact with sentinel sheep of a susceptible breed during the quarantine period; and
  - b) remain disease free from the disease for 21 days after the collection of germplasm is complete; and
  - c) the germplasm collection centre should be situated in a sheep and goat pox-free zone.

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## 8. CRIMEAN CONGO HAEMORRHAGIC FEVER

### 8.1 Hazard Identification

*8.1.1 Aetiological Agent:* Family Bunyaviridae; Genus: *Nairovirus*, Crimean-Congo haemorrhagic fever virus.

*8.1.2 OIE List:* Not listed.

*8.1.3 New Zealand Status:* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004).

#### *8.1.4 Epidemiology*

Crimean-Congo disease virus 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 the ELISA, can be used to detect antibody against Crimean-Congo haemorrhagic fever virus (Burt et al., 1993; Qing et al., 2003) and PCR methods and viral isolation can be used to detect virus (Schwarz et al., 1996; Burt et al., 1998). Sheep and goats have often been found to be positive in serological surveys (Wilson et al., 1990; Mariner et al., 1995; Williams et al., 2000; Qing et al., 2003). In humans the virus causes a serious disease but in animals it causes a transient inapparent infection (Swanepoel and Burt, 2004). The principle 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 principle vectors of the disease and the distribution of the disease mirrors the distribution of these ticks (Swanepoel et al., 1987). The incubation period in sheep is 3-9 days and they remain viraemic for about 7 days (Gonzalez et al., 1998; Swanepoel and Burt, 2004). There are no descriptions of long term carriers.

#### *8.1.5 Hazard identification conclusion*

Crimean-Congo haemorrhagic disease virus causes a serious disease in humans and is classified as an exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004). It is regarded as a potential hazard in this risk analysis.

## 8.2 Risk Assessment

### 8.2.1 Release Assessment

#### 8.2.1.1 Semen and embryos (sheep and goats)

No information was found on the transmission of the virus in semen or embryos. Since viraemia occurs for a period of around 7 days (Gonzalez et al., 1998) it is assumed that germplasm collected during viraemia could be infected. The likelihood of collecting germplasm during a viraemic episode is low but non-negligible.

### 8.2.2 Exposure Assessment

Any imported semen or embryos would be inseminated or implanted into susceptible New Zealand sheep and goats. Therefore the likelihood of exposure is high.

### 8.2.3 Consequence Assessment

#### 8.2.3.1 Introduction of semen and embryos from sheep and goats

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 sheep or goats 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 *Haemophysalis longicornis* is not known to be capable of carrying the virus (Heath, 2002). In addition the likelihood that a recipient of germplasm would be infested by a cattle tick while viraemic after insemination or transplantation, is very low. Establishment of the disease in New Zealand is therefore unlikely. 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 asymptomatic.

#### 8.2.3.2 Other consequences

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 asymptomatic infections in feral ruminants and small mammals.

#### 8.2.3.3 Consequence assessment conclusion

The consequences of introduction are low but because the New Zealand cattle tick has not been conclusively shown to be an incompetent vector the consequences are assessed as non-negligible. The possible effects on human health are also non-negligible.

#### *8.2.4 Risk Estimation*

The release, exposure and consequence assessments are non-negligible for all commodities. Therefore the risk estimation is considered to be non-negligible.

### **8.3 Risk Management**

#### *8.3.1 Risk Evaluation*

Since the risk estimate for all commodities is non-negligible, the implementation of risk management measures to reduce the level of risk to an acceptable level is justified.

#### *8.3.2 Option Evaluation*

##### 8.3.2.1 Risk Management objective

The objective of risk management is to ensure that semen or embryos are not collected from viraemic donors.

##### 8.3.2.2 Options available

The disease has a short incubation period and long-term carriers do not occur. Therefore, quarantine of tick free goats or sheep 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.

##### 8.3.2.3 Recommended sanitary measures

Donors should:

- i. have been resident for at least the 21 days before germplasm collection in a country that is free from the disease; or
- ii. 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 should be kept in quarantine for a minimum of 3

weeks immediately before the start of and also during semen or embryo collection and regularly inspected and maintained in a tick-free state throughout the period of quarantine; or

- iii. Donors should be serologically tested within one week 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 would be suitable for export. Germplasm from animals that are negative at both tests would be suitable for export. Germplasm from animals that sero-convert or have rising titres between the two tests should be disqualified from being exported to New Zealand. If any animal from a group of donors is disqualified due to the testing procedures, germplasm from all animals in the group should be disqualified.

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## 9. FOOT AND MOUTH DISEASE

### 9.1 Hazard Identification

*9.1.1 Aetiological Agent:* Family: Picornaviridae, Genus *Aphthovirus*, Foot and mouth disease virus. There are seven serotypes of the virus: O, A, C, SAT 1, SAT 2, SAT 3, Asia 1.

*9.1.2 OIE List:* Listed

*9.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

#### *9.1.4 Epidemiology*

Extensive reviews on foot and mouth disease are available (Sanson, 1994; Thomson and Bastos, 2004) and much of the information given below is taken from these reviews. The disease has been eradicated from or has not occurred in North America, Europe and Australasia and some Asian countries such as Japan and Korea. However devastating outbreaks of the disease have occurred in some of these countries in recent years (Thomson and Bastos, 2004). Foot and mouth disease is the most contagious and economically devastating 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. Sheep and goats are less severely affected by the virus than pigs and cattle, but because the signs of infection are less obvious the disease is harder to control in sheep. Infected animals excrete the virus in saliva, faeces, urine, milk, semen, ocular and nasal discharges (Sanson, 1994; Thomson and Bastos, 2004) and it 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 symptoms (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 most commonly from inhaling aerosolized virus (Sanson, 1994; Thomson and Bastos, 2004). Long term carriers that excrete small amounts of virus from the pharynx for long periods occur in both sheep and goats. Sheep may excrete virus in this way for up to 9 months (Thomson and Bastos, 2004).

#### *9.1.5 Hazard identification conclusion*

Foot and mouth disease is a devastating highly contagious disease and the virus is an exotic, notifiable organism (Ministry of Agriculture and Forestry, 2004). Therefore, the virus is classified as a potential hazard for the purposes of this risk analysis.

## 9.2 Risk Assessment

### 9.2.1 Release Assessment

#### 9.2.1.1 Semen (sheep and goats)

Hare listed the virus as one that can be excreted and transmitted in small ruminant semen, but provided no references or other details and made the assumption by extrapolation from bulls and boars (Hare, 1985). There is no information about the excretion of the virus in sheep or goat semen and extrapolation from what is known in cattle is necessary. The virus is excreted in the semen of bulls during the viraemic period (Callis, 1996). Transmission of the virus to susceptible females can result from insemination with infected semen. The risk of release of virus in semen is considered to be non-negligible.

#### 9.2.1.2 Embryos (sheep and goats)

Foot and mouth disease of sheep and goats is classified by IETS as a Category 3 disease “for which preliminary evidence indicates that the risk of transmission is negligible provided that the embryos are properly handled between collection and transfer, but for which additional *in vitro* and *in vivo* experimental data are required to substantiate the preliminary findings” (IETS, 2002). The likelihood that embryos from infected sheep and goats will be contaminated with foot and mouth disease virus is low, but since it is only classed as a Category 3 organism by IETS the risk is considered to be non-negligible.

### 9.2.2 Exposure Assessment

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

### 9.2.3 Consequence Assessment

#### 9.2.3.1 Introduction of semen from sheep and goats

In cattle insemination with infected semen is likely to result in infection of the recipient (Callis, 1996) and this is assumed to also be true for sheep and goats. It is not impossible that a case of disease could go unrecognized in sheep and goats. 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 even by aerosol to animals several kilometers from them. Once the infection had spread to pigs or cattle which shed larger amounts of virus than sheep and goats, the disease could spread by airborne infection over many kilometers (Gloster et al., 1982).

Animals that become infected could become the focal point for a serious outbreak of foot and mouth disease in New Zealand. An outbreak of the disease would cause serious disruption to the livestock industries, economic losses to individual farmers, considerable

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).

#### 9.2.3.2 Introduction of embryos from sheep and goats.

The likelihood that recipients of embryos would become infected is low (IETS, 2002) but non-negligible. Infected recipient animals would be highly contagious and the consequences would be the same as those described in the previous Section (12.2.3.1)

#### 9.2.3.3 Other consequences

Foot and mouth disease is “a rare human disease of medical curiosity” (Sanson, 1994) and there would be no consequences for human health.

The virus infects cloven hoofed animals and could infect feral pigs, goats and deer.

#### 9.2.3.4 Consequence assessment conclusion

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

### *9.2.4 Risk Estimation*

The likelihood of release, exposure and consequences are non-negligible for all the commodities. Therefore the risk is non-negligible.

## **9.3 Risk Management**

### *9.3.1 Risk Evaluation*

The risk for both semen and embryos is non-negligible and risk management is indicated to reduce the risk to an acceptable level.

### *9.3.2 Option Evaluation*

#### 9.3.2.1 Risk Management objective

The objective is to reduce to negligible the likelihood of introducing foot and mouth disease virus in sheep or goat germplasm.

#### 9.3.2.2 Options available

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. The OIE Terrestrial Animal Health Code gives no guidelines for the importation of embryos from sheep and goats (Anonymous, 2004). Only preliminary information is available on the ability of sheep and goat embryos to transmit the virus. In view of the paucity of information relating directly to sheep and goats, the extreme seriousness of the disease and the catastrophic consequences that could result from its introduction it is suggested that a more conservative approach is appropriate. Therefore, importation of germplasm from countries that are infected with foot and mouth disease could be prohibited.

#### 9.3.2.3 Recommended sanitary measures

Importations of semen and embryos should be restricted to importation from countries that are free from foot and mouth disease and in which vaccination is not practised.

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## 10. OVINE PULMONARY ADENOMATOSIS

### 10.1 Hazard Identification

*10.1.1 Aetiological Agent:* Family: Retroviridae; Genus *Betaretrovirus*, Jaagsiekte virus.

*10.1.2 OIE List:* Listed

*10.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

*10.1.4 Epidemiology*

Ovine pulmonary adenomatosis, also known as Jaagsiekte, has an almost world-wide distribution (Verwoerd et al., 2004) but does not occur in Australia and New Zealand. Jaagsiekte virus is an exogenous retrovirus (Palmarini et al., 1999; Verwoerd et al., 2004) that infects sheep and goats. Goats are apparently more resistant to the disease than sheep (Tustin et al., 1988) and the prevalence of the natural disease in goats is low (Verwoerd et al., 2004). An ovine lentivirus is also commonly found associated with cases of jaagsiekte but does not appear to have a role in the etiology of the disease (Querat et al., 1987; Verwoerd et al., 2004). Jaagsiekte retrovirus cannot be grown in culture but the entire genomic sequence has been elucidated from cloned sequences (York et al., 1992; York and Querat, 2003). Jaagsiekte is a contagious disease and spreads by contact. Transmission from ewe to lamb is likely since neonatal lambs are more susceptible than older sheep (Verwoerd et al., 2004).

The incubation period has been quoted as varying from 9 months to 2 or 3 years (Verwoerd et al., 1994), but more recently the same authors gave the incubation period as approximately 5-6 months (Verwoerd et al., 2004). The time lapse between introduction of affected sheep and the development of new cases varied from 5 ½ - 8 months in Iceland (Dungal et al cited by (Verwoerd et al., 1994)), to several years in South Africa (Tustin, 1969). The course of the disease varies from weeks to several months and is characterized by respiratory signs coughing and discharge of fluid from the nose particularly when the hind legs are raised (wheelbarrow test).

A PCR test done on peripheral blood leucocytes, that detects infection at an early stage, has been developed (Gonzalez et al., 2001), but it has not yet been verified to a level that would allow it to be used for export/import certification. At present a diagnosis can only be made clinically or at post mortem where typical gross and histological lesions can be identified.

#### *10.1.5 Hazard identification conclusion*

Jaagsiekte virus is an exotic, notifiable organism (Ministry of Agriculture and Forestry, 2004). Therefore, it is classified as a potential hazard for the purposes of this risk analysis.

### **10.2 Risk Assessment**

#### *10.2.1 Release Assessment*

##### 10.2.1.1 Semen (sheep and goats)

There is no information in the literature about the ability of semen to transmit the virus. However, since the virus has been demonstrated in peripheral blood leukocytes (Palmarini et al., 1996) it is possible that semen that contains peripheral blood leukocytes could be infected with jaagsiekte virus. Leakage of leukocytes into semen occurs in some conditions such as infection with *Brucella ovis* (Preziuso et al., 2003). Concomitant infections with *Brucella ovis* or other conditions that allow seepage of leukocytes into semen could result in semen becoming infected with jaagsiekte virus. Therefore the likelihood that semen could be infected is non-negligible.

##### 10.2.1.2 Embryos (sheep and goats)

There is no direct evidence that embryos can be infected with virus. It was suggested that since intrauterine infection does not occur embryo transplantation might be used to create disease-free flocks (Tustin, 1969; Parker et al., 1989). Subsequently it was shown that 38 progeny from embryos derived from infected donor ewes were not infected. In addition 11 embryos from four donor ewes that were not infected with the disease and were mated with infected rams, did not transmit the disease to the recipients or their progeny (Parker et al., 1998). IETS has classified the disease as a category 3 disease for which “preliminary evidence indicates that the risk of transmission is negligible provided the embryos are properly handled between collection and transfer, but for which additional *in vitro* and *in vivo* experimental data are required to substantiate the preliminary findings” (Anonymous, 1998; IETS, 2002). Therefore the likelihood that embryos could transmit the disease is low but non-negligible.

#### *10.2.2 Exposure Assessment*

Imported semen or embryos would be inseminated/transplanted into susceptible recipients. Therefore, the likelihood of exposure is high for all commodities.

### *10.2.3 Consequence Assessment*

#### 10.2.3.1 Introduction of semen and embryos from sheep and goats

It is assumed that insemination/transplantation of infected germplasm into susceptible sheep or goats could result in infection of the recipients. The disease has a long incubation period and could go unnoticed for some years and during this period could spread to close contacts and to the offspring of the infected animals. This could result in the establishment of a focus of infection from which the virus could be widely disseminated. Because there are no reliable tests for the disease coupled with the insidious manner in which the disease spreads, it would be hard to identify newly infected flocks and eradicate the disease. The establishment of the disease in New Zealand would cause an erosion of productivity and economic losses to individual infected farmers.

#### 10.2.3.2 Other consequences

The virus does not infect humans or other species and there would be no consequences for human. Feral goats and sheep could become infected but since contact with infected sheep or goats is not likely to occur the likelihood of infecting feral animals is low.

#### 10.2.3.3 Consequence assessment conclusion

Since the establishment of the disease in New Zealand would cause erosion of productivity for sheep farmers the consequences are assessed as non-negligible.

### *10.2.4 Risk Estimation*

Release, exposure and consequence assessments are all non-negligible. Therefore the risk is considered to be non-negligible.

## **10.3 Risk Management**

### *10.3.1 Risk Evaluation*

Since the risk for sheep and goat germplasm is non-negligible, risk management measures are justified to reduce risk to an acceptable level.

### *10.3.2 Option Evaluation*

#### 10.3.2.1 Risk management objective

The objective is to prevent the introduction of germplasm that is infected with jaagsiekte virus

#### 10.3.2.2 Options available

The OIE Terrestrial Animal Health Code does not include any recommendations relating to Jaagsiekte.

Germplasm could be introduced from countries that are free from jaagsiekte. Alternatively donor animals could be carefully selected from closed flocks with long and well substantiated histories of freedom from jaagsiekte (at least 3 years) in countries where the disease is notifiable and good records are available.

Since the risk of introducing the virus in embryos is low (IETS, 2002), and there is no information about the risk involved in the use of semen, introduction of germplasm could be restricted to embryos. In view of the long incubation period and the life-long carrier state of infected animals, pre-collection quarantine is not practical. The incubation period of the disease has not been accurately determined but has been variously given as 5-6 months ((Verwoerd et al., 1994; Verwoerd et al., 2004) and 3 years (Verwoerd et al., 1994). However, neonatal animals are most susceptible but the disease is most commonly seen in animals that are 2-4 years old and the disease may have a course of up to a year ((Verwoerd et al., 1994; Verwoerd et al., 2004). These observations seem to indicate that an incubation period of longer than 6 months is likely. Therefore, a conservative approach is appropriate and a post arrival quarantine period of three and a half years could be imposed on recipients and their offspring. This period could be altered when more definitive information about the incubation period becomes available.

Since the disease is commonly transmitted from ewe to lamb, slaughter and post mortem examination of first generation progeny before releasing second generation progeny from quarantine could be implemented.

Suitable tests for individual animals are not yet available but a promising test has been described (Gonzalez et al., 2001). In future the literature describing the use of this test should be followed and if it is well validated, its use should be incorporated into any programme to introduce genetic material from countries endemically infected with the virus.

#### 10.3.2.3 Recommended sanitary measures

It is recommended that:

- i. Germplasm should be introduced from animals that have lived their whole lives in countries that are free from jaagsiekte: or
- ii. Only embryos should be introduced: and
  - a) Donor animals should be selected from flocks with a long history of freedom from jaagsiekte. Importation of embryos from countries where reliable records are not available should not be allowed; and

- b) Recipients of imported embryos and any offspring resulting from implanted embryos should be held in post arrival quarantine in New Zealand for at least three and a half years. At the end of three and a half years recipients of germplasm and the first generation progeny of the germplasm should be slaughtered and examined for the presence of lesions of jaagsiekte. Only second generation progeny should be released from quarantine when the first generation progeny have been shown to be free from the disease.

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## 11. LOUPING ILL AND RELATED DISEASES

### 11.1 Hazard Identification

*11.1.1 Aetiological Agent:* Family: Flaviviridae; Genus *Flavivirus*, louping-ill virus. Several related viruses cause tick-borne encephalitis in Europe.

*11.1.2 OIE List:* Not listed.

*11.1.3 New Zealand Status:* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004)

#### *11.1.4 Epidemiology*

Louping ill is a tick-borne disease of sheep that occurs in Scotland and some parts of Great Britain. It is characterized by nervous symptoms and a low mortality rate in endemically infected areas. In many animals signs of infection are mild or inapparent (Swanepoel and Laurenson, 2004). Closely related diseases of the tick-borne encephalitis complex occur in parts of Europe, Russia, and Asia (Swanepoel and Laurenson, 2004).

The disease is zoonotic and occasional cases of louping ill infection occur in occupationally exposed people in Great Britain (Davidson et al., 1991). A much higher rate of infection of people is found in Russia where 11,000 cases of tick-borne encephalitis occur annually. Possibly another 3,000 cases occur in the rest of Europe. The disease is described as a dangerous infection (Gritsun et al., 2003a; Gritsun et al., 2003b). However, the literature on the occurrence of the disease in sheep in Russia and Europe is scarce. It has been stated that viruses isolated from sheep in continental Eurasia have not been well characterized but are probably identical or similar to louping ill virus (Swanepoel and Laurenson, 2004). The only natural vector for louping ill appears to be *Ixodes ricinus* but other ixodid ticks may also be capable of transmitting the disease (Swanepoel and Laurenson, 2004). *Ixodes persulcatus* is also an important vector of tick-borne encephalitis (Korenberg and Kovalevskii, 1999) and *Haemaphysalis* spp. has also been implicated as a vector (Khazova and Iastrebov, 2001). The disease can be transmitted orally since the transmission to pigs that were fed on infected lambs has been reported (Bannatyne et al., 1980). Goat kids have been infected from the ingestion of milk of infected nanny goats (Reid et al., 1984). However, lambs were not infected by ingestion of virus infected milk from infected ewes (Reid and Pow, 1985).

Louping-ill virus infects a wide variety of domestic and wild animals including goats (Reid et al., 1983; Reid et al., 1984; Hudson et al., 1995; Hudson et al., 1997; Gilbert et al., 2000) but it causes significant disease only in sheep and red grouse. The mountain hare may be a significant host for the virus and for the vector ticks (Hudson et al., 1997). The incubation period is from 2- 5 days (Swanepoel and Laurenson, 2004). In sheep the disease is usually mild but may be more severe when concomitant infections occur with *Ehrlichia phagocytophilum* (formerly *Cytoecetes phagocytophila*) which is carried by the

same vector and is the agent of tick-borne fever (Reid et al., 1986). When susceptible sheep are introduced into heavily infected areas, mortality can be as high as 60%, but sheep in endemically infected areas are usually immune and mortality is low (Swanepoel and Laurenson, 2004). The disease is characterized by a biphasic fever (Swanepoel and Laurenson, 2004). Nervous signs typically occur at the onset of the second fever when viraemia is usually no longer present but when the brain is infected. The intensity and duration of viraemia is influenced by the presence of antibody and is proportionally diminished according to the level of immunity.

#### *11.1.5 Hazard identification conclusion*

The virus is an exotic unwanted organism (Ministry of Agriculture and Forestry, 2004). It is also a zoonotic organism and it is classed as a potential hazard in this risk analysis.

### **11.2 Risk Assessment**

#### *11.2.1 Release Assessment*

##### 11.2.1.1 Semen and embryos (sheep and goats)

No information could be found on the ability of semen or embryos to transmit the virus. Viraemia of varying intensity lasts for only 3-5 days (Reid, 1983) or up to 7 days (Swanepoel and Laurenson, 2004) and it is possible that semen or embryos could be infected with virus during these periods. Most adult sheep and goats in endemically infected areas are immune (Swanepoel and Laurenson, 2004) and since long term carriers have not been described and the period of viraemia is short the likelihood that semen or embryos would be collected from a viraemic animal is low. Therefore the likelihood of introducing infected semen or embryos is low but non-negligible.

#### *11.2.2 Exposure Assessment*

Imported semen and embryos would be inseminated or transplanted into susceptible New Zealand recipients and therefore the likelihood of exposure is high.

#### *11.2.3 Consequence Assessment*

##### 11.2.3.1 Introduction of semen and embryos from sheep and goats.

There is no information in the literature about the infectivity of semen or embryos but it is assumed that infected semen or embryos could infect the recipients. Inseminated/transplanted recipients could develop signs and in extreme cases even die from the disease. However, they would not be contagious and could not infect animals they were in contact with. During the viraemic period infected recipients could infect ticks but the known vectors of the disease are not present in New Zealand. The New Zealand cattle tick *Haemophysalis longicornis* is not a known vector of louping ill (Heath, 2002) but *Haemaphysalis* spp. are vectors for tick-borne encephalitis (Khazova and Iastrebov,

2001). Although the disease is unlikely to establish in New Zealand, the occurrence of the disease in recipients of germplasm would compromise the ability to certify that New Zealand is free from the disease. This would affect the export of sheep and goats to countries requiring such a freedom statement.

#### 11.2.3.2 Other consequences

Louping ill occurs sporadically in man in occupationally exposed people. One fatal case has been described. Tick-borne fever is common in Russia and some parts of Europe. If the disease did become established it could cause rare cases of a generally non-fatal disease in humans.

The virus is not known to cause significant disease in other animals such as cattle, deer or small wild animals and birds with the exception of the red grouse. Therefore, it is unlikely to cause damage to New Zealand's wild and feral animals. Some animals such as hares might become infected but remain asymptomatic. The effects on the environment are assessed to be negligible.

#### 11.2.3.3 Consequence analysis conclusion

Since the virus is assumed to be capable of infecting recipients of germplasm, this could lead to an inability to certify New Zealand as being free from louping ill and tick-borne encephalitis. It could also lead to infections of humans with louping ill or tick borne encephalitis viruses. Therefore the consequences of introducing the virus are non-negligible.

#### *11.2.4 Risk Estimation*

Since the release, exposure and consequence assessments are all considered to be non-negligible, the risk is non-negligible.

### **11.3 Risk Management**

#### *11.3.1 Risk Evaluation*

Since the risk estimate was found to be non-negligible, risk management measures should be implemented to reduce the risk to an acceptable level.

#### *11.3.2 Option Evaluation*

##### 11.3.2.1 Risk management objective

The objective is to ensure that germplasm is not collected from animals that are in the viraemic stage of louping ill.

#### 11.3.2.2 Options available

There are no OIE recommendations regarding louping-ill for trade in germplasm. The incubation period (2-5 days) and the septicaemic period (up to 7 days) are both short and long term carriers have not been described in sheep or goats. Therefore, since the disease is a tick-borne disease, quarantine for a suitable period in tick-free premises, before germplasm collection, could ensure that donors are not septicaemic when germplasm is collected.

#### 11.3.2.3 Recommended sanitary measures

Germplasm donors should:

- i. have been resident in a country that is free from the disease, for at least 21 days immediately before and during germplasm collection; or
- ii. be scrupulously treated for ticks before being moved onto tick-free collection premises. They should be carefully inspected and maintained tick-free while on the germplasm collection centre. Germplasm collection should not begin until they have been on the tick-free premises for at least 21 days.

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## 12. MAEDI-VISNA

### 12.1 Hazard Identification

*12.1.1 Aetiological Agent:* Family: Retroviridae; Genus: *Lentivirus*, Maedi-visna virus,

*12.1.2 OIE List:* Listed

*12.1.3 New Zealand Status:* Exotic, notifiable organism (Ministry of Agriculture and Forestry, 2004)

#### *12.1.4 Epidemiology*

Maedi-visna occurs in most countries other than Australia and New Zealand (Anonymous, 2003). Maedi is characterized by a chronic pneumonia and visna is the neurological syndrome caused by the same virus. It is a disease of sheep and a closely related virus causes caprine arthritis and encephalitis (CAE) in goats. Maedi-visna does not usually occur naturally in goats and CAE is usually confined to goats. However experimental infection of sheep with CAE virus has been reported (Banks et al., 1983; Oliver et al., 1985). Recently some viruses with intermediate characteristics have been found infecting both species (Shah et al., 2004; Grego et al., 2005). However, these variants, though interesting, do not affect this risk analysis which is confined to maedi visna of sheep.

Most infected animals are asymptomatic, persistent carriers of the virus for life (Knowles and Herrmann, 2004). The disease has a long incubation period and chronic course and the maedi-visna virus is therefore, classified as a slow virus (Petursson et al., 1990). It is rarely seen in sheep less than 3-4 years old (Verwoerd and Tustin, 2004). After running a chronic course it usually ends with death 6-12 months after signs are first observed.

Transmission is usually from an infected ewe to her lamb by way of infected colostrum and milk and less commonly by lateral transmission in respiratory aerosols (Cutlip et al., 1988; Petursson et al., 1990). Transmission *in utero* is rare and is not significant in the epidemiology of the disease (Cross et al., 1975; Cutlip et al., 1988; Brodie et al., 1998). Disease free flocks can be established from lambs that were removed from their dams before they had suckled (Houwens et al., 1983; Houwers et al., 1987; Petursson et al., 1990). Diagnosis can be made serologically, infected animals develop antibody 3-4 weeks after infection (Petursson et al., 1990). The PCR test is a sensitive test for the demonstration of proviral DNA (Verwoerd and Tustin, 2004).

#### *12.1.5 Hazard identification conclusion*

The virus is an exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004) and is therefore a potential hazard for the purposes of this risk analysis.

## 12.2 Risk Assessment

### 12.2.1 Release Assessment

#### 12.2.1.1 Semen (sheep and goats)

Maedi-visna virus (Krogsrud and Udnes, 1978)) and CAE virus (Adams et al., 1983) are not transmitted in the semen. It has also been stated that “semen has never been implicated as a source of infection” (Verwoerd and Tustin, 2004). However, testicular lesions have been described in maedi-visna infected rams (Palfi et al., 1989) and this gives cause for concern that rams with lesions in their testes could be excreting virus in their semen. The virus is known to circulate in the blood in mononuclear leucocytes. Any condition in which leucocytes cross into the semen is therefore likely to result in infection of the semen. *Brucella ovis* infection is characterized by a large number of leucocytes in the semen and concurrent infection with *Brucella ovis* and maedi-visna virus has been shown to result in shedding of maedi-visna virus in semen (De La Concha et al., 1996; Preziuso et al., 2003). Since *Brucella ovis* infection is endemic in New Zealand testing for *Brucella ovis* will not be a requirement in IHSs. Therefore importation of semen contaminated with maedi-visna virus in *Brucella ovis* infected rams could occur. The likelihood of semen being infected with maedi-visna virus is low but non-negligible.

#### 12.2.1.2 Embryos (sheep and goats)

IETS classified the disease as a Category 4 disease “on which preliminary work has been conducted or is in progress” (IETS, 2002). The virus can occasionally be transmitted *in utero* (Cross et al., 1975; Cutlip et al., 1988). Many cells contain proviral DNA but no description of germ cells containing integrated proviral DNA was found. In studies on the closely related CAE virus, proviral DNA was demonstrated in the flushing media from oviducts of CAE infected goats (Fieni et al., 2002). Proviral DNA in cell lysates from the infected flushings was removed by serial dilution indicating that washing of embryos would probably remove provirus unless it was integrated into germ cell DNA. The exact mechanism by which rare cases of *in utero* transmission have occurred is not clear, but the integration of proviral DNA into germ cells and later transcription into viral RNA cannot be ruled out. Therefore, the likelihood of transmission of the virus by embryos is unlikely but non-negligible.

### 12.2.2 Exposure Assessment

Imported semen or embryos would be inseminated or implanted into susceptible recipient ewes. Therefore, the likelihood of exposure is high.

### *12.2.3 Consequence Assessment*

#### 12.2.3.1 Introduction of semen and embryos from sheep and goats.

No information has been found in the literature that describes the outcome of inseminating or transplanting infected germplasm into susceptible ewes. Therefore, it should be assumed that this action could result in infection of the recipient ewes. Since most infected sheep will remain asymptomatic lifelong carriers they could continue to infect in contact animals and transmit the virus to their progeny. Establishment of the disease in New Zealand would cause an erosion of productivity in infected flocks. It is likely that expensive eradication programmes would have to be undertaken in individual flocks or as a national eradication campaign.

#### 12.2.3.2 Other consequences

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

Natural infection only occurs in sheep and there would be no consequences for the environment.

#### 12.2.3.3 Consequence assessment conclusion

Since the disease could establish in the New Zealand sheep flock and cause erosion of productivity the consequences are considered to be non-negligible.

### *12.2.4 Risk Estimation*

Release, exposure and consequence assessments are all non-negligible and therefore the risk is considered to be non-negligible.

## **12.3 Risk Management**

### *12.3.1 Risk Evaluation*

Since the risks involved in the importation of sheep germplasm is non-negligible, risk management measures are justified to reduce risk to an acceptable level.

### *12.3.2 Option Evaluation*

#### 12.3.2.1 Risk management objective

The objective is to ensure that imported germplasm from sheep and goats is not infected with maedi-visna virus.

#### 12.3.2.2 Options available

The OIE Terrestrial Animal Health Code does not give any recommendations for trade in germplasm relating to maedi-visna virus. It recommends that for trade in live animals, animals should be taken from flocks that have remained closed and disease free for 3 years, and subjected to a serological test with negative results. Since lifelong asymptomatic carriers occur (Knowles and Herrmann, 2004), quarantine of donors is not useful for preventing entry of the disease. Donor animals could be selected from disease free flocks such as those that have been established in the Netherlands (Houwens et al., 1987). Importations could be restricted to the importation of embryos since there is preliminary work indicating that the procedure could be safe and there is evidence that washing embryos eliminates proviral DNA (Fieni et al., 2002). However the likelihood of transmission in semen is low. Serological testing is a valuable method of detecting virus carriers (Knowles and Herrmann, 2004) and serological testing of flocks that have been closed and remained disease free for at least 3 years and individual donors could be useful. Complement fixing antibodies can be detected 3-4 weeks after infection (Verwoerd and Tustin, 2004).

#### 12.3.2.3 Recommended sanitary measures

It is recommended that:

- i. donors of germplasm should have been born in and lived their entire lives in a country that is free from maedi-visna; or
- ii. donors should be selected from disease free flocks, preferably from flocks in official accreditation schemes; and
  - a) individual donors should be tested by an OIE recommended ELISA test 4-8 weeks after collection of germplasm. Germplasm from animals that are serologically positive should be disqualified from entry into New Zealand; or
- iii. flocks that are not officially accredited should have been maintained as closed flocks and remained free from clinical disease for 3 years. A sample of sheep from the flock large enough to give a 99% confidence of detecting infection at a 1% prevalence rate in the flock should be tested by an OIE recommended serological test (Knowles and Herrmann, 2004). Donors should be selected only from flocks shown to be maedi-visna free; and
  - a) individual donors should be tested by an OIE recommended ELISA 4-8 weeks after collection of germplasm. Germplasm from animals that are serologically positive should be disqualified from entry into New Zealand.

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## 13. NAIROBI SHEEP DISEASE

### 13.1 Hazard Identification

*13.1.1 Aetiological Agent:* Family: Bunyaviridae: Genus: Nairovirus, Nairobi sheep disease virus.

*13.1.2 OIE List:* Listed.

*13.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

#### *13.1.4 Epidemiology*

Nairobi sheep disease is a tick-borne viral disease that causes 30-90% mortality in outbreaks of disease in naïve sheep and goats (Davies and Terpstra, 2004; Gerdes, 2004). Contrary to expectation exotic sheep are more resistant than indigenous sheep breeds (Davies and Terpstra, 2004; Gerdes, 2004). Most sheep and goats in indigenous areas have antibody to the virus and clinical cases are rarely seen. The incubation period is 3-6 days after tick attachment (Davies and Terpstra, 2004). Signs of infection include fever, hyperventilation, anorexia and swollen lymph nodes. Diarrhoea is a prominent symptom in animals which survive the acute stage of the disease (Davies and Terpstra, 2004). Fever persists for 1-7 days and viraemia persist till 24 hours after the fever returns to normal (Terpstra, 1994). The disease is a tick-borne disease and infected animals are not contagious. *Rhipicephalus appendiculatus* is the main vector and the infection is carried transovarially in this tick. In other tick species such as *Amblyomma variegatum* and other *Rhipicephalus* spp. the disease is transmitted transstadially (Davies and Terpstra, 2004).

Reports of the disease are confined to East and Central Africa but antibody surveys indicate that its distribution in Africa may be wider than this. Ganjam virus which causes a similar disease in India is an Asian variant of Nairobi sheep disease virus (Marczinke and Nichol, 2002).

Accidental transmission to humans in the laboratory, resulting in a mild disease has been reported (Davies and Terpstra, 2004) and antibodies have been found in human sera (Morrill et al., 1991; Terpstra, 1994). Laboratory infections of people are more common with Ganjam virus (Davies and Terpstra, 2004).

#### *13.1.5 Hazard identification conclusion*

The virus is an exotic, notifiable organism and the disease is an OIE listed disease. Therefore, the organism is considered to be a potential hazard for this risk analysis.

## **13.2 Risk Assessment**

### *13.2.1 Release Assessment*

#### 13.2.1.1 Semen and embryos (sheep and goats)

There is no information about the transmission of the virus in germplasm. However it is assumed that semen and embryos collected from viraemic animals could be infected with virus. The period of viraemia is short (1-8 days) and the likelihood of collecting germplasm from viraemic animals is, therefore, low but non-negligible.

### *13.2.2 Exposure Assessment*

Imported semen and embryos would be inseminated or transplanted into susceptible recipients and therefore the likelihood of exposure is high.

### *13.2.3 Consequence Assessment*

#### 13.2.3.1 Introduction of semen and embryos from sheep and goats

There is no information on whether transmission of virus to the recipients of infected germplasm occurs. It is assumed that this is likely. Infected animals would develop signs of the disease and there would be a high mortality amongst them. However, the disease is a tick-borne disease (Davies and Terpstra, 2004) and infected recipients of germplasm would not be contagious and would not infect other susceptible animals in contact with them. Since the only New Zealand tick *Haemaphysalis longicornis* is not known to be a vector (Heath, 2002) the disease is unlikely to be able to establish. However, the occurrence of the disease in recipients of germplasm would result in the inability to certify New Zealand as free from the disease and therefore interfere with the trade of live animals.

#### 13.2.3.2 Other consequences

Cases of disease in people have only occurred due to laboratory accidents. Infected sheep would not be infectious and humans would not become infected from contact with them. Therefore the consequences for human health are negligible.

The only wild animals that might be susceptible are feral goats and thar, but since there are no competent vectors in New Zealand the likelihood of them becoming infected is negligible and the consequences for the environment are negligible.

#### 13.2.3.3 Consequence assessment conclusion

Since the infection of recipients would interfere with trade in live animals the consequences are non-negligible.

#### *13.2.4 Risk estimation*

Since the release, exposure and consequence assessments are non-negligible, risk is considered to be non-negligible.

### **13.3 Risk Management**

#### *13.3.1 Risk Evaluation*

Since the risk estimate was found to be non-negligible, risk management measures should be implemented to reduce the risk to an acceptable level.

#### *13.3.2 Option Evaluation*

##### 13.3.2.1 Risk management objective

The objective is to ensure that germplasm is not collected from animals that are in the viraemic stage of Nairobi sheep disease.

##### 13.3.2.2 Options available

There are no recommendations in the OIE Terrestrial Animal Health Code relating to Nairobi sheep disease. The incubation period (6 days) and the viraemic period (up to 8 days) are both short and long term carriers have not been described. Therefore, since the disease is a tick-borne disease, quarantine for a suitable period in tick-free premises, before germplasm collection, could ensure that donors are not septicaemic when germplasm is collected.

##### 13.3.2.3 Recommended sanitary measures

Donors of germplasm should:

- i. be resident in countries that are free from the disease for at least the 21 days prior to germplasm collection; or
- ii. be scrupulously treated for ticks before being moved onto tick-free collection premises. They should be carefully inspected and maintained tick-free while on the germplasm collection centre. Germplasm collection should not begin until they have been on the tick-free premises for at least 21 days.

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## 14. PLYAM VIRUSES

### 14.1 Hazard Identification

*14.1.1 Aetiological Agent:* Family: Reoviridae, Genus *Orbivirus*, viruses belonging to the Palyam serogroup.

*14.1.2 OIE List:* Not listed.

*14.1.3 New Zealand Status:* Exotic organism not listed as unwanted.

#### *14.1.4 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. Most of what is known about the viruses applies to cattle, but neutralizing antibody has been found in sheep and goats (Swanepoel, 2004). Because specific evidence about sheep is lacking this review focuses on cattle and it is assumed that the information is applicable to sheep and goats. 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). In one review 15 viruses were listed (Swanepoel, 2004), others have been reported (Doyle and Walton, 1992). Large numbers of isolations of arboviruses including many Palyam viruses have been made from the blood of naturally infected, asymptomatic cattle and *Culicoides* midges in South Africa and Australia (Theodoridis et al., 1979; Cybinski and St George, 1982; Gard et al., 1988a; Gard et al., 1988b; Littlejohns et al., 1988; Gard et al., 1989; Nevill et al., 1992). Although the viruses usually cause mild or asymptomatic infections they have been associated with abortions in Zimbabwe. Kasba virus was associated with congenital abnormalities such as hydranencephaly 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, Muria, Goto, Kubo and Kono reported that cattle were consistently viraemic for 2 weeks and intermittently viraemic for 8 weeks (Swanepoel, 2004). An arbovirus and *Culicoides* surveillance programme has been in operating in New Zealand since 1991 (Ryan et al., 1991). In a typical year seroconversion did not occur to bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses in samples from 10 sentinel cattle from each of 17 herds and *Culicoides* spp. were not found in 15,000 insects collected from light traps (Motha et al., 1997). The *Culicoides* monitoring programme has continued up to the present time with results of the serology programme reported regularly in the MAF Surveillance magazine. No seroconversion has been detected in sentinel cattle and no *Culicoides* have been trapped.

#### *14.1.5 Hazard identification conclusion*

The Palyam virus group does not cause economically important diseases. They are not classified as unwanted or notifiable organisms in New Zealand. However, because they are exotic and do occasionally cause abortions or foetal malformations they have been classified as potential hazards for the purposes of this analysis.

### **14.2 Risk Assessment**

#### *14.2.1 Release Assessment*

##### 14.2.1.1 Semen (sheep and goats)

There is no information on the transmission of Palyam viruses in semen or embryos. 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). It is likely that Palyam viruses will also be excreted in semen during the viraemic period that lasts for up to 8 weeks in cattle (Muria, Goto, Kubo & Kono according to Swanepoel (Swanepoel, 2004)). Therefore the likelihood of release of virus in semen is non-negligible.

##### 14.2.1.2 Embryos (sheep and goats)

No information is available about the transmission of Palyam viruses by embryos. Therefore the likelihood of release is assumed to be low but non-negligible.

#### *14.2.2 Exposure Assessment*

Semen or embryos would be inseminated or transplanted into susceptible sheep or goats. Therefore the likelihood of exposure is high.

#### *14.2.3 Consequence Assessment*

##### 14.2.3.1 Introduction of semen and embryos from sheep and goats

It is assumed that insemination or transplantation of infected germplasm would lead to infection of the recipient. Infection would be asymptomatic 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 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, and the virus then failing to become established in New Zealand are considered to be negligible.

#### 14.2.3.2 Other consequences

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.

#### 14.2.3.3 Consequence assessment conclusions

The consequences of the introduction of Palyam viruses in germplasm are considered to be negligible.

#### *14.2.4 Risk Estimation*

Since the consequences of introduction of Palyam viruses in germplasm are considered to be negligible, under the methods used in this risk analysis (Section 4.2), risk is considered to be negligible.

### **14.3 Risk Management**

#### *14.3.1 Risk Evaluation*

Since the risk estimation for sheep and goat germplasm is negligible, risk management measures are not warranted.

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## 15. PESTE DES PETITS RUMINANTS AND RINDERPEST

### 15.1 Hazard Identification

*15.1.1 Aetiological Agent:* Family: Paramyxoviridae; Genus *Morbillivirus*, peste des petits ruminants and rinderpest viruses.

*15.1.2 OIE List:* Listed.

*15.1.3 New Zealand Status:* Exotic, notifiable (Ministry of Agriculture and Forestry, 2004).

#### *15.1.4 Epidemiology*

Peste des petits ruminants (PPR) is an acute contagious disease of sheep and goats and related wild bovidae which is characterized by high morbidity and mortality (Rossiter, 2004a). Rinderpest is caused by a closely related morbillivirus and is primarily a disease of cattle (Rossiter, 2004b). The two diseases are similar in many respects although rinderpest virus may cause a less severe disease than PPR in sheep and goats. Where information on PPR is lacking extrapolation from what is known about rinderpest in cattle is justified. During 2003 the OIE handistatus database reported only 1 outbreak of rinderpest in Kenya (Anonymous, 2004a), indicating that rinderpest may soon be eradicated from the world. PPR occurred in countries in Central, West and North Africa, the Middle East and India (Anonymous, 2004a). The disease spread from sub-Saharan Africa to the Middle East and India in the late nineteen eighties and early nineteen nineties (Shaila et al., 1996; Dhar et al., 2002).

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

Infection with PPR virus most commonly occurs in the oropharynx and upper respiratory system through inhalation of aerosol particles. Primary infection establishes in the pharyngeal lymph nodes and tonsils and following a period of viraemia, in all lymphoid tissues (Rossiter and Taylor, 1994). The viraemic period usually precedes the onset of acute symptoms and high fever. During the acute phase of the disease infected animals excrete virus in ocular and nasal excretions, urine and faeces (Mushi and Wafula, 1984; Wafula et al., 1989). This stage may last for about 10 days. Viraemia begins 1-2 days before the onset of illness and begins to fall when circulating antibody first appears (Scott, 1990). Pregnant animals that recover from rinderpest may abort some weeks after recovery and the foetus and vaginal discharges are infected with virus (Wafula et al., 1989; Rossiter, 2004b). Animals that recover from peste des petits ruminants infection do not become carriers (Scott, 1990). Vaccination with attenuated rinderpest vaccine

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

#### *15.1.5 Hazard identification conclusion*

Rinderpest and PPR are highly contagious OIE listed diseases. In New Zealand they are classified as exotic, notifiable diseases (Ministry of Agriculture and Forestry, 2004) and for the purposes of this analysis are considered to be potential hazards.

### **15.2 Risk Assessment**

#### *15.2.1 Release Assessment*

##### 15.2.1.1 Semen (sheep and goats)

Peste des petits ruminants and rinderpest have both been listed as diseases in which the virus has been found in small ruminant semen and is likely to be transmitted by semen (Hare, 1985). It has also been reported that animals infected with rinderpest shed the virus in all their excretions and secretions (Scott, 1990). Therefore, semen can be assumed to be infectious during the acute stage of the disease when infected animals are viraemic. However, because rinderpest has been virtually eradicated from the world (Anonymous, 2004a) the likelihood of collecting semen from a viraemic sheep or goat infected with rinderpest virus is negligible. The likelihood of semen being infected with peste des petits ruminants virus is low but non-negligible since viraemia may occur before the onset of signs (Scott, 1990).

##### 15.2.1.2 Embryos (sheep and goats)

It is assumed that the likelihood of transmission of rinderpest virus and peste des petits ruminants virus is similar. IETS has classified rinderpest as an agent or disease in category 3 which is described as “disease or disease agent for which preliminary evidence indicates that the risk of transmission is negligible, provided that the embryos are properly handled between collection and transfer, but for which additional *in vitro* and *in vivo* experimental data are required to substantiate the preliminary findings” (IETS, 2002). This indicates that the likelihood of transmission of both viruses in embryos is low but non-negligible.

#### *15.2.2 Exposure Assessment*

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

### *15.2.3 Consequence Assessment*

#### 15.2.3.1 Introduction of semen and embryos from sheep and goats

Insemination or transplantation of infected germplasm into susceptible recipients is likely to cause infection of the recipients (Hare, 1985). This would result in the recipients developing signs of the disease and possibly dying. During the course of the illness animals would be contagious and would infect other ruminant animals they were in contact with. European breeds of pigs could become subclinically infected with rinderpest virus (Rossiter, 2004 a). If left unchecked the disease could spread rapidly through New Zealand causing high morbidity and mortality in sheep and goats. The disease could have serious effects on the economy of individual farms and the country and the productivity of the livestock industries concerned. To ensure freedom from peste des petits ruminants virus, meat derived from animals in infected areas would have to be processed by methods that would destroy the virus (Anonymous, 2004b) In the case of rinderpest additional certification concerning the origin of the meat, requirements for vaccination and deboning and removing of lymph nodes would be required (Anonymous, 2004c). This would have serious economic consequences for the meat industry.

#### 15.2.3.2 Other consequences

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

Deer are also susceptible to peste petits ruminants virus and a wide variety of ruminants and pigs are susceptible to rinderpest (Rossiter, 2004b, a). Feral sheep, goats and deer could become infected with the disease and suffer mortalities and become a source of infection. Other non ruminant animals and birds would not be affected.

#### 15.2.3.3 Consequence assessment conclusion

Since the introduction of infected germplasm could have serious effects on ruminant health and productivity the consequences of introducing the virus are non-negligible.

### *15.2.4 Risk Estimation*

Release, exposure and consequence estimates are all non-negligible. Therefore the risk is considered to be non-negligible for all commodities.

## **15.3 Risk Management**

### *15.3.1 Risk Evaluation*

Since the risk estimates for all the commodities are non-negligible, risk management measures are justified to reduce risk to an acceptable level.

### *15.3.2 Option Evaluation*

#### 15.3.2.1 Risk management objective

The objective is to ensure that germplasm for export to New Zealand is not collected from viraemic donors.

#### 15.3.2.2 Options available

Germplasm from donors that have been resident in PPR and rinderpest free countries could be safely imported. Long term carriers of virus are not known to occur and the period during which animals remain infectious is short. Therefore in infected countries, quarantine of germplasm donors could be an effective method to prevent the introduction of the virus. It is recommended in the OIE Terrestrial Animal Health Code that a 21 day quarantine period should be imposed and that donors of semen should remain free from clinical signs of disease for an additional 21 days after collection. For embryo collection OIE recommends in addition to quarantine, that unvaccinated donors are tested serologically at least 21 days after collection (Anonymous, 2004b). For rinderpest OIE recommendations are similar although not exactly the same as for PPR (Anonymous, 2004c). OIE places considerable reliance on quarantine and recognition of clinical signs of disease. Since mild strains of both diseases occur in indigenous situations (Rossiter, 2004b, a) it is recommended that testing or vaccination with modified live Rinderpest or PPR (Rossiter, 2004a; Rossiter, 2004b) vaccine could provide an additional safeguard for semen as well as for embryos.

#### 15.3.2.3 Recommended sanitary measures

The recommendations in the Terrestrial Animal Health Code for PPR should be the basis for ensuring that PPR and rinderpest viruses are not imported in germplasm. It is recommended that donors of germplasm should:

- i. kept in a country that is free from rinderpest and PPR, for at least 3 months prior to collection of germplasm; or
- ii. be kept for the 21 days prior to collection, in an establishment or germplasm collection centre where there have been no animals introduced in the 21 days prior to collection and no animal in the establishment showed signs of PPR or rinderpest at the time of collection or for the following 21 days. The germplasm collection centre should not be situated in a PPR infected zone; and
  - a) The donors have not been vaccinated against rinderpest or PPR and were tested with an OIE recommended serological test for PPR, with negative results, not less than 21 days after collection of germplasm; or

- b) The donors have been vaccinated against PPR or rinderpest at least 21 days before and not more than 4 months prior to germplasm collection.

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## 16. RABIES

### 16.1 Hazard Identification

*16.1.1 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.

*16.1.2 OIE List:* Listed

*16.1.3 New Zealand Status:* Exotic and notifiable (Ministry of Agriculture and Forestry, 2004).

#### *16.1.4 Epidemiology*

Rabies is a disease of all mammals including man. It is characterized by severe nervous symptoms 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 and 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 symptoms may vary from weeks to years. In sheep 2-17 weeks has been reported (Swanepoel, 2004). 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 may become aggressive leading to biting and transmission of the disease. 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. In sheep sexual excitability is said to occur more often than in goats which tend to become aggressive (Swanepoel, 2004). Cattle, sheep and goats are generally dead-end hosts since they are unlikely to bite other animals or man. The disease occurs less frequently in sheep and goats than in cattle. Out of 6,389 cattle, sheep and goats with a confirmed diagnosis of rabies in four Southern African countries between 1928 and 1991, only 651 (10.2%) of cases occurred in sheep and goats (Swanepoel, 1994). Assuming a population of sheep and goats of 40,000,000 in the 4 countries concerned over a period of 40 years this equates to an annual prevalence of only  $4 \times 10^{-7}$ . Therefore, even if the disease was grossly under-reported the prevalence in sheep was very low.

#### *16.1.5 Hazard identification conclusion*

Rabies virus can infect virtually all animals and man. It is an exotic, notifiable disease and is therefore classified as a potential hazard for the purposes of this risk analysis.

### **16.2 Risk Assessment**

#### *16.2.1 Release Assessment*

##### 16.2.1.1 Semen (sheep and goats)

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 some other organs may be infected (Swanepoel, 2004). It is inconceivable that anyone would collect semen from a rabid animal in the final stages of the disease and therefore the likelihood of collecting semen infected with rabies is considered to be negligible.

##### 16.2.1.2 Embryos (sheep and goats)

In pregnant females due to the immunosuppressive effects of pregnancy transplacental infection may occur in rare cases (Martell et al., 1973; Howard, 1981; Sipahioglu and Alpaut, 1985). It has been demonstrated experimentally (Swanepoel, 2004). However donor females would not be pregnant at the time of embryo collection and viraemia and infection of organs other than the central nervous system do not occur except in the terminal stages of the disease (see Section 18.2.1.1) when collection of embryos would not occur. For these reasons the likelihood of embryos being infected with rabies virus is considered to be negligible.

### 16.2.1.3 Release assessment conclusion

The likelihood release of virus in semen or embryos collected from clinically healthy sheep or goats is negligible. Therefore under the methodology chosen for this analysis (Section 4.2), the risk is considered to be negligible.

## **16.3 Risk Management**

### *16.3.1 Risk Evaluation*

Since the estimated risk is negligible risk management measures are not justified.

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## 17. RIFT VALLEY FEVER

### 17.1 Hazard Identification

*17.1.1 Aetiological Agent:* Family: Bunyaviridae; Genus *Phlebovirus*, Rift Valley fever virus.

*17.1.2 OIE List:* Listed.

*17.1.3 New Zealand Status:* Exotic, notifiable. (Ministry of Agriculture and Forestry, 2004)

#### *17.1.4 Epidemiology*

Rift valley fever is an acute disease of sheep, goats, cattle and people. The etiological 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) Goats are more resistant to the disease. In cattle the disease is less severe and abortion and morbidity rates are lower. 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. (Anonymous, 2000; Jup et al., 2002; Al-Afaleq et al., 2003; Balkhy and Memish, 2003) There is evidence that the virus was not present in the Arabian peninsular before the outbreak in 2000. (Al-Afaleq 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 *Eremaphodites*. (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 12 -36 hours. (Swanepoel and Coetzer, 2004) 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) and virus persists in visceral organs 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)

#### *17.1.5 Hazard identification conclusion*

The virus is an exotic, notifiable organism (Ministry of Agriculture and Forestry, 2004) and is therefore included as a potential hazard in this risk analysis.

### **17.2 Risk Assessment**

#### *17.2.1 Release Assessment*

##### 17.2.1.1 Semen (sheep and goats)

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 non-negligible.

##### 17.2.1.2 Embryos (sheep and goats)

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, 2002) 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 risk of release of virus is considered to be non-negligible.

#### *17.2.2 Exposure Assessment*

Imported semen or embryos would be inseminated or implanted into susceptible ewes or does. Therefore the risk of exposure is high.

#### *17.2.3 Consequence Assessment*

##### 17.2.3.1 Introduction of semen and embryos from sheep and goats

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”, (Anonymous, 2004) no evidence could be found to support or refute this contention. 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 *Ochlerotatus 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 *Ochlerotatus 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 has not been established 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 (Anonymous, 2004)

#### 17.2.3.2 Other consequences

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.

#### 17.2.3.3 Consequence assessment conclusion

Rift Valley fever is a zoonotic disease and if it were to establish it could cause serious economic consequences to the sheep industry. Therefore the consequences of introducing sheep or goat germplasm are considered to be non-negligible.

#### *17.2.4 Risk estimation*

Since release, exposure and consequence assessment for all commodities are non-negligible, the risk is non-negligible.

### **17.3 Risk Management**

#### *17.3.1 Risk Evaluation*

Since risks is estimated to be non-negligible, risk management measurements are required to reduce the risk to an acceptable level.

#### *17.3.2 Option Evaluation*

##### 17.3.2.1 Risk management objective

The objective is to avoid collecting germplasm for export to New Zealand from donor sheep or goats that are infected with Rift Valley fever virus.

##### 17.3.2.2 Options available

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). 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 are free from disease for periods of several years. During these periods mosquito activity is low and diseased animals are not found. 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. (Anonymous, 2004). These recommendations could be applied directly to donors of germplasm.

##### 17.3.2.3 Recommended sanitary measures

To prevent the importation infected germplasm the OIE recommendations for trade in live animals (Anonymous, 2004) should be applied to germplasm donors. Immediately prior to collection of germplasm donors of should have:

- i. resided for the 30 days prior to the collection of germplasm and during germplasm collection in a Rift Valley fever-free country or zone; or

- ii. 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; or
- iii. 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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## 18. VESICULAR STOMATITIS

### 18.1 Hazard Identification

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

*18.1.2 OIE List:* Listed.

*18.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004)

#### *18.1.4 Epidemiology*

The disease occurs in cattle, horses and pigs. It is considered to be a disease of horses, cattle and pigs (Schmidt, 2004), but it is also stated that “Sheep goats and many other wild species can be infected” (Schmidt, 2004). In the OIE Handistatus database data are only presented for cattle, horses and pigs. References in the scientific literature relating to infection of sheep and goats are rare, but cases were reported to occur in sheep and goats in the 1982 outbreak of the disease in the USA (Henry, 1982; Buisch, 1983; Schmidt, 2004). Experimentally infected sheep developed antibody against the virus (Ashfar et al., 1993; Rodriguez, 2002; Schmidt, 2002). Therefore the disease can be considered to be a rare disease in sheep and goats. 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 mainly of importance because it is clinically indistinguishable from foot and mouth disease (Sellers and Daggupaty, 1990; Rodriguez, 2002; Schmidt, 2002). Therefore, initial diagnosis of the disease before laboratory confirmation of the viral etiology, 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 northern locations in the USA (Schmidtman et al., 1999) and even as far as Canada (Wilks, 1994).

The disease is caused by two types of the virus, New Jersey and Indiana. Three distinct sub-types of Indiana are known (Wilks, 1994). However genomic studies have revealed further diversification amongst strains of virus (Rodriguez et al., 1996).

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; Stallknecht, 2000; Schmidtman et al., 2002) but Culicoides 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).

There is also a theory that vesicular stomatitis virus is a plant rhabdovirus that has adapted and become infectious for animals (Hanson and McMillan, 1990; Wilks, 1994). This theory has been dismissed as unlikely for the purposes of this analysis since no plant pathogens are known to infect mammals, and the evidence suggesting that it may be an arbovirus is more compelling.

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 (Wilks, 1994; Letchworth et al., 1999).

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 (Anonymous, 2004).

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). Mead states that viraemia does not occur in mammalian hosts but demonstrated transmission of the virus to non-infected blackfly when infected and non-infected blackfly fed on the same host (Mead et al., 2000). In contrast Blood and Radostits state 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 the experimental infection of deer mice (Cornish et al., 2001).

Since viraemia does not occur in domestic animals it is very unlikely that semen or embryos will be infected. The disease was classified by IETS as a category 4 disease for which “preliminary work has been conducted or is in progress” (IETS, 2002).

#### *18.1.5 Hazard identification conclusion*

Vesicular stomatitis virus is classified as an exotic notifiable disease (Ministry of Agriculture and Forestry, 2004). Therefore, it is classified as a potential hazard for the purposes of this risk analysis.

### **18.2 Risk Assessment**

#### *18.2.1 Release Assessment*

##### 18.2.1.1 Semen and embryos (sheep and goats)

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. 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. The likelihood of sheep or goats being viraemic and asymptomatic at the time of semen collection is unlikely even in the most heavily infected countries. The likelihood of release of virus in germplasm is therefore unlikely but non-negligible.

##### 18.2.1.2 Embryos (sheep and goats)

There is no evidence that embryos from infected sheep or goats can be infected with the virus. 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). 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”. However, even this limited information cannot be extrapolated directly to sheep and goats. Also if viraemia does not occur in this disease contamination of embryos with virus is unlikely. Therefore, the likelihood of embryos being infected with the virus is low but non-negligible.

#### *18.2.2 Exposure Assessment*

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

#### *18.2.3 Consequence Assessment*

##### 18.2.3.1 Introduction of semen and embryos from sheep and goats

No data relating to the use of infected semen or embryos in susceptible sheep or goats 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 it 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. It is unlikely to have any impact on the trade in animal products and germplasm (Anonymous, 2004).

#### 18.2.3.2 Other consequences

The virus is a zoonotic organism that causes disease in people. Infection is by 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 asymptomatic. Therefore the effects on the environment are likely to be negligible.

#### 18.2.3.3 Consequence assessment conclusion

Since the possibility of establishment, the economic consequences and the effects on human health are all non-negligible risk is non-negligible.

#### *18.2.4 Risk Estimation*

Since release, exposure and consequence assessments of introducing sheep and goat germplasm are all non-negligible, risk is non-negligible.

## 18.3 Risk Management

### 18.3.1 Risk Evaluation

The risk of introducing sheep and goat embryos is non-negligible and therefore risk management measures are justified to reduce risk to an acceptable level.

### 18.3.2 Option evaluation

#### 18.3.2.1 Risk management objective

The objective is to ensure that sheep and goat germplasm for export to New Zealand is not collected from viraemic donor animals.

#### 18.3.2.2 Options available

The OIE gives recommendations for trade in live animals and embryos but not for semen (Anonymous, 2004). It was concluded in the release assessment that the likelihood of sheep or goat semen being contaminated with virus is 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 (Anonymous, 2004). The serological test could be done after completion of germplasm collection instead of before germplasm collection as recommended by OIE.

#### 18.3.2.3 Recommended sanitary measures

Donors should:

- i. be resident for the 21 days prior to germplasm collection and during germplasm collection, in a country or zone that is free from vesicular stomatitis; or
- ii. be kept in an insect free quarantine station for at least the 30 days prior to and during germplasm collection; and
- a) be subjected to an OIE recommended serological test with a negative result, 3-6 weeks after germplasm collection.

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## 19. WESSELSBRON DISEASE

### 19.1 Hazard Identification

*19.1.1 Aetiological Agent:* Family: Flaviviridae: Genus *Flavivirus*, Wesselsbron disease virus.

*19.1.2 OIE List:* Not listed.

*19.1.3 New Zealand Status:* Exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004).

#### *19.1.4 Epidemiology*

Wesselsbron disease virus is a flavivirus that is carried by mosquitoes and particularly by *Aedes* spp. (Jupp and Kemp, 1998) of the subgenera *Ochlerotatus* and *Neomelanimon* but it has also been isolated from *Culex* and *Mansonia* spp. (Mushi et al., 1998; Swanepoel and Coetzer, 2004). The main vectors are *Aedes* spp. which are floodwater species that lay their eggs in mud or in grass or sedge and can survive there during dry periods to emerge after the area is again flooded (Jupp, 2004). However, the virus has also been found in *Culex uvittatus* (Swanepoel and Coetzer, 2004) which is not a floodwater species and feeds mainly on birds but also to some extent on mammals and man (Jupp, 2004).

Wesselsbron disease causes abortion and neonatal death and is zoonotic. Malformed foetuses with arthrogryposis and hydranencephaly also occur (Barnard, 1990). In many respects the disease resembles Rift Valley fever but it does not cause economic losses or zoonotic infections on the same scale as Rift Valley fever (Swanepoel and Coetzer, 2004). It is a disease of unusually wet summers when floodwater mosquitoes occur in abnormally high numbers. The disease affects sheep and goats (Mushi et al., 1998; Swanepoel and Coetzer, 2004). It has remained mainly confined to Southern Africa but demonstration of antibody or isolations of virus have occurred in other African countries (Baba, 1993; Wilson et al., 1994; Baba et al., 1995; Mushi et al., 1998), Madagascar (Morvan et al., 1990), Reunion Island (Kles et al., 1994) and even Thailand (Swanepoel and Coetzer, 2004). A Nigerian isolate of the virus was shown to be pathogenic by experimental infection of goats (Baba et al., 1988; Baba, 1993). In Zimbabwe there is a high incidence of sero-conversion in cattle (Blackburn and Swanepoel, 1980) but outbreaks of disease are rare. The prevalence of antibody positive animals in endemic areas is high and the occurrence of disease is low (Barnard, 1990), indicating that most animals have been infected and developed immunity before they become pregnant. In areas such as the South African highveld massive expansions of floodplain mosquitoes only occur in occasional seasons of high rainfall and rare disease outbreaks occur in these seasons in populations of sheep that do not have antibody against the virus. In South Africa only three outbreaks of the disease and a few sporadic cases were recorded between 1956 and 1990 (Barnard, 1990). It is rarely reported in other African countries.

The disease has an incubation period of 1-4 days and animals remain viraemic for 4 days (Coetzer et al., 1978; Theodoridis and Coetzer, 1980; Barnard, 1990). The incubation period is longer and the viraemic period shorter in adult animals than in neonates. The mortality rate in experimentally infected neonatal kids and goats was 18% and 27% respectively (Coetzer et al., 1978). The virus is hepatotropic and severe liver damage and icterus may be seen at post mortem (Swanepoel and Coetzer, 2004).

There is no evidence that the virus is contagious amongst sheep (Swanepoel and Coetzer, 1994).

#### *19.1.5 Hazard identification conclusion*

The virus is classified as an exotic, unwanted organism (Ministry of Agriculture and Forestry, 2004). Therefore, it is classified as a potential hazard for the purposes of this risk analysis.

### **19.2 Risk Assessment**

#### *19.2.1 Release Assessment*

##### 19.2.1.1 Semen (sheep and goats)

The virus is listed as a virus that is excreted in semen but in which transmission probably does not occur by artificial insemination (Hare, 1985). This listing was based on a single reference from 1981 and no confirmatory evidence has been found. However, it is reasonable to expect that the virus would be excreted in semen during the viraemic period. Viraemia lasts only 1-4 days (Coetzer et al., 1978; Theodoridis and Coetzer, 1980; Barnard, 1990) and it is unlikely that semen would be collected from a viraemic donor. The likelihood of collecting semen from viraemic donor is therefore considered to be low but non-negligible.

##### 19.2.1.2 Embryos (sheep and goats)

No information is available on the contamination of embryos by the virus. It should be assumed that virus could contaminate embryos collected from viraemic donors. Therefore, the likelihood of an infected embryo being collected for export to New Zealand is low but non-negligible.

#### *19.2.2 Exposure Assessment*

Semen or embryos from sheep and goats, imported into New Zealand would be inseminated or transplanted into susceptible recipients. Therefore the likelihood of exposure is high.

### *19.2.3 Consequence Assessment*

#### 19.2.3.1 Introduction of semen and embryos from sheep and goats

Since the virus has been reported as being present in semen (Hare, 1985) it is assumed that the virus could be transmitted to susceptible recipients by infected germplasm. The most likely outcome of infecting non-pregnant adults is a febrile asymptomatic infection (Swanepoel and Coetzer, 2004). Infected recipients would not be contagious and would not transmit the disease to other animals they were in contact with, but could transmit the virus to competent mosquito vectors during the period of viraemia. Therefore, the probability of establishment is dependant on whether suitable vectors are present in New Zealand. Since the main vectors of the disease are adapted to African conditions characterized by summer rainfall and periodic flooding it is unlikely that similar vectors species will occur in New Zealand (Pharo, 1999). However, the ability of New Zealand mosquitoes to transmit the virus has not been tested experimentally. Therefore the likelihood of transmission is non-negligible

#### 19.2.3.2 Other consequences

Wesselsbron disease is a zoonotic disease (Jupp and Kemp, 1998; Swanepoel and Coetzer, 2004). Humans are infected by handling abortion and post mortem material and by mosquito bites (Swanepoel and Coetzer, 2004). If the virus became established in New Zealand, cases of flu-like disease would occur in people during outbreaks of the disease.

The virus has a host range that includes cattle, sheep, goats, pigs, horses, donkeys, camels, guinea pigs, rabbits, wild birds, wild mammals and man (Barnard, 1990). Therefore, it is likely that feral and wild animals and birds could be infected in New Zealand. It is likely that infections in these animals would remain asymptomatic since in Africa only sheep, goats and occasionally man and cattle develop the disease. Feral goats and probably thar would be susceptible but outbreaks of disease would be likely to be rare events in these animals.

#### 19.2.3.3 Consequence assessment conclusion

Since the likelihood of establishment of the disease is non-negligible and establishment of the disease would have consequences for human health and cause some losses to sheep and goat farmers the consequences of introducing the virus are non-negligible.

### *19.2.4 Risk Estimation*

Because release, exposure and consequence assessments for all commodities are non-negligible the risk is non-negligible.

## **19.3 Risk Management**

### *19.3.1 Risk Evaluation*

Since the risk estimation for all commodities is non-negligible, risk management measures are justified to reduce risk to an acceptable level.

### *19.3.2 Option Evaluation*

#### 19.3.2.1 Risk management objective

The objective is to introduce risk management measures that will ensure that germplasm is not collected from viraemic donors.

#### 19.3.2.2 Options available

Wesselsbron disease is not an OIE listed disease and there are no OIE recommendations relating to it.

Since long term carriers have not been described and the incubation and viraemic periods are short, quarantine would be effective in ensuring animals were not viraemic at the time of germplasm collection. The quarantine could be in an area that is free from mosquito activity such as areas subject to repeated hard frosts like the South African highveld in winter or an insect free quarantine building. A quarantine period of 21 days would adequately cover the maximum reported incubation period of 4 days and a viraemic period of 4 days (Coetzer et al., 1978; Theodoridis and Coetzer, 1980; Barnard, 1990) with a substantial additional safety margin due to the limited information available about this disease. A lack of disease in a country does not necessarily indicate freedom from the virus, since the virus circulates over large parts of tropical and subtropical Africa where the disease does not occur (Swanepoel and Coetzer, 2004). The virus has also been isolated in Thailand (Swanepoel and Coetzer, 2004). Therefore the definition of virus free countries is problematical. A country can only be assumed to be free from the virus if serological surveys show that virus is not circulating.

Antibodies measured in the serum neutralisation test or the haemagglutination inhibition test developed 4 days after infection and were maximal in 2-3 weeks and remained detectable for 2 years (Swanepoel and Coetzer, 2004). Animals could be tested by a serum neutralization or haemagglutination inhibition (HAI) test, before and 3 weeks after germplasm collection to ensure that they had not become viraemic during the period of germplasm collection.

#### 19.3.2.3 Recommended sanitary measures

Prior to germplasm collection donors should have:

- i. Resided for the 21 days prior to the collection of germplasm and during the collection of germplasm in a country or zone in which Wesselsbron disease virus does not circulate (as shown by serological surveys); or
- ii. Been held in mosquito-free premises or area (e.g. frost prone areas during the winter) for at least the 21 days prior to, and during the collection of germplasm; or
- iii. Been subjected to a serological test within a week prior to germplasm collection and again 3-6 weeks after germplasm collection. Germplasm would be suitable for importation if there was a positive test prior to germplasm collection or two negative tests. Germplasm should be disqualified from importation if there is a rising titre or seroconversion between the two tests.

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## 20. ANTHRAX

### 20.1 Hazard Identification

20.1.1 *Aetiological agent: Bacillus anthracis*

20.1.2 *OIE List: Listed*

20.1.3 *New Zealand Status: Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).*

20.1.4 *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). Sheep and goats are very susceptible to experimental infection (De Vos, 1994) but in the field situation are less commonly infected than cattle (Tuchili et al., 1993; De Vos, 1994; Vaissaire et al., 1996; Liang et al., 1999; Turner et al., 1999a; Turner et al., 1999b).

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 some circumstances infection can occur by inhalation (wool sorter's disease and terrorism in humans) but this is not of importance in sheep and goats. Carriers of the disease may occur in partially immunized cattle that recover from natural infection (De Vos, 1994), and in impala (De Vos and Turnbull, 2004) but no reference was found to a carrier state in sheep or goats. The incubation period probably varies from one to 14 days and 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 of the disease 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).

#### *20.1.5 Hazard identification conclusion*

Anthrax is an exotic, notifiable (Ministry of Agriculture and Forestry, 2004) and zoonotic disease and was therefore classified as a potential hazard in this risk analysis.

### **20.2 Risk Assessment**

#### *20.2.1 Release Assessment*

##### 20.2.1.2 Semen and embryos (sheep and goats)

Sheep and goats 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” (Anonymous, 2004). 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 infected sheep or goat will only sporulate after death of the animals when the carcass is opened and the organisms are exposed to air. There is no reason to indicate that semen or embryos collected from healthy sheep and goats in facilities that meet New Zealand requirements for collection centres, and processed according to IETS recommended methods could be infected with the bacillus. In addition the vegetative form of *B 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 wash fluids. The likelihood that germplasm collected from clinically healthy sheep or goats would be infected with anthrax spores is considered to be negligible.

#### *20.2.2 Risk Estimation*

Because the likelihood of release was considered to be negligible for all commodities, under the methodology used for this analysis, (Section 4.2) the risk is assessed to be negligible.

### **20.3 Risk Management**

#### *20.3.1 Risk Evaluation*

Since the risk is negligible, risk management measures are not justified.

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## 21. BRUCELLOSIS (BRUCELLA MELITENSIS)

### 21.1 Hazard Identification

21.1.1 *Aetiological agent: Brucella melitensis*

21.1.2 *OIE List: Listed.*

21.1.3 *New Zealand Status: Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).*

21.1.4 *Epidemiology*

Brucellosis in sheep and goats is generally caused by *Brucella melitensis*, rare cases may be due to *Brucella abortus* (Garin-Bastuji and Blasco, 2004). The disease is characterized by abortion, retained placenta, orchitis, epididymitis and sometimes arthritis. Infected animals may remain long term carriers and excrete the organism in uterine discharges, semen and milk (Amin et al., 2001; Garin-Bastuji and Blasco, 2004; Godfroid et al., 2004). Uterine discharges can be copious and persist for up to 3 months after parturition (Godfroid et al., 2004). The oral route is the main route of infection after contamination of the environment by uterine discharge following abortions. Persistently infected serologically negative animals can occur, when lambs are born to infected ewes. In one investigation 62 lambs born from 42 serologically positive ewes were studied. Four of the lambs were persistently infected while remaining serologically negative (Grillo et al., 1997).

The organism causes a serious disease in man known as Malta or Mediterranean fever which can become chronic and debilitating (Blood and Radostits, 1989).

21.1.5 *Hazard identification conclusion*

The organism is exotic, notifiable (Ministry of Agriculture and Forestry, 2004) and zoonotic and is therefore classified as a potential hazard for the purposes of this risk analysis.

### 21.2 Risk Assessment

21.2.1 *Release Assessment*

21.2.1.1 Semen (sheep and goats)

Orchitis and epididymitis occurs commonly in infected male goats and sheep (Garin-Bastuji and Blasco, 2004; Godfroid et al., 2004) and the organism can be excreted in the semen (Amin et al., 2001; Godfroid et al., 2004). The risk of release of the organism in semen is therefore non-negligible

#### 21.2.1.2 Embryos (sheep and goats)

The position with regard to embryos harvested from infected ewes and does is not known. However, there is a considerable body of evidence from cattle that shows that *Brucella abortus* is not carried by properly prepared and washed embryos (Stringfellow et al., 1982; Voelkel et al., 1983; Stringfellow and Wright, 1989). 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 *B. 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, 2002).

Although it is probably correct that embryo transfer is safe some reservations remain. *Brucella* spp. are intracellular pathogens (Roop et al., 2004) that can multiply in trophoblast cells (Samartino et al., 1994) antibiotics do not always penetrate cell membranes so that experiments done with suspensions of *Brucella* organisms or organisms that are attached to the zona pellucida may not necessarily be valid. Embryos which had defective zona pellucidas or no zona pellucida could not be freed from *Brucellas* by the ordinary washing procedures (Stringfellow et al., 1984). Acceptance that embryo transplant is a safe method of introducing germplasm therefore implies a trust in the standards for preparing embryos in the exporting country and acceptance that defective zona pellucidas will be recognized in all cases. It is also known that *Brucella* can multiply within trophoblast cells in embryos or in cell culture experiments in which embryos were exposed to *Brucella abortus* *in vitro* (Samartino et al., 1994).

Experiments done in cattle with *Brucella abortus* and with cattle cells can not be extrapolated directly to infections of sheep and goats with *Brucella melitensis*. For these reasons the likelihood of release of *Brucella melitensis* in sheep and goat embryos is non-negligible.

#### 22.2.2 Exposure Assessment

Implantation or insemination of susceptible New Zealand recipients with imported germplasm would occur. Therefore the likelihood of exposure is high.

#### 22.2.3 Consequence Assessment

##### 22.2.3.1 Introduction of semen and embryos from sheep and goats.

Insemination or transplantation of infected germplasm into susceptible recipients would be likely to lead to infection. Infected recipients might abort after some months or they might not become pregnant and remain carriers of infection and abort at a subsequent pregnancy or they could give birth to a full term lamb or kid or give birth to a latently infected lamb or kid. Abortions or normal births would be followed by excretion of the

organism in vaginal discharges and/or milk and this could lead to infection of in contact sheep or goats. Ultimately this could lead to the establishment of the disease in New Zealand and an erosion of production efficiency and profitability in infected flocks. Infection of cattle with *Brucella melitensis* is unlikely but sporadic cases could occur.

#### 22.2.3.2 Other consequences

Cases of human disease are likely to occur due to eating non-pasteurised sheep and goat milk products or contact with sheep or goats at lambing and kidding. Brucellosis is a serious disease that may lead to debilitating chronic infections and serious complications.

The infection is likely to remain confined to domestic animals but could spread to feral goats and thar. Other species of animals are unlikely to be affected. The effect on the environment is likely to be negligible.

#### 22.2.3.3 Consequence assessment conclusion

Since the introduction of infected germplasm could result in the establishment of a production limiting and zoonotic disease the consequences are considered to be non-negligible.

#### *22.2.4 Risk Estimation*

Since release, exposure and consequence assessments for all commodities are non-negligible the risk is considered to be non-negligible.

### **21.3 Risk Management**

#### *21.3.1 Risk Evaluation*

Risk is assessed to be non-negligible and therefore risk management measures are justified to reduce risk to an acceptable level.

#### *21.3.2 Option Evaluation*

##### 21.3.2.1 Risk management objective

The objective is to ensure that germplasm for export to New Zealand is not collected from donors that are infected with *Brucella melitensis*

##### 21.3.2.2 Options available

The OIE makes recommendations for the trade in embryos and semen (Anonymous, 2004). The OIE Terrestrial Animal Health Code recommends selecting donors from sheep or goat flocks “officially free” or “free” from infection. The OIE Terrestrial Animal Health Code defines conditions for official freedom (without vaccination and

testing) and freedom (with vaccination and testing). Donors from officially free flocks are not required to be tested and those from free flocks are required to be tested serologically prior to germplasm collection (Anonymous, 2004). However, the recommendations do not guard against the possibility of a persistently infected serologically negative animals (Grillo et al., 1997) being used as a donor. To guard against such a possibility it is suggested that in the case of semen an aliquot of each semen collection batch could be cultured or tested by PCR before dilution and addition of antibiotics. In the case of embryos an aliquot of embryos, and the first wash fluid (without antibiotics added) from the embryos, could be cultured by OIE recommended methods (Nielsen and Ewart, 2004). Where there are substandard embryos available these could be tested. If no substandard embryos are available an aliquot of embryos could be sacrificed for testing.

#### 21.3.2.3 Recommended sanitary measures

It is recommended that:

- i. germplasm should be collected from animals that are resident in countries that are officially free from caprine and ovine brucellosis according to the OIE standards for country freedom; or
- ii. germplasm should be collected from donors resident in flocks that are officially free from brucellosis according to the OIE definition for officially free flocks; or
- iii. germplasm should be collected from donors resident in flocks that are free from brucellosis according to the OIE definition of freedom from Brucellosis; and
  - a) for semen - an aliquot of semen from each batch, before addition of antibiotics, should be cultured for isolation of *Brucella* spp (Nielsen and Ewart, 2004), with negative results.
  - b) for embryos - an aliquot of embryos made up from substandard embryos or an aliquot of available embryos, and wash fluid from the first wash without the addition of antibiotics should be cultured with negative results.
  - c) After removal of the aliquots for testing, embryos and semen should be further processed according to standard methods with the addition of antibiotics.

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## 22. CLOSTRIDIA: *C. TETANI* AND *C. BOTULINUM*

### 22.1 Hazard Identification

21.1.1 Aetiological agent: *Clostridium tetani* and *Clostridium botulinum*

21.1.2 OIE List: Not listed

21.1.3 New Zealand Status: Both *Clostridium tetani* and *Clostridium botulinum* are endemic. The position regarding the strains that occur in New Zealand is not fully known.

### 22.1.4 Epidemiology

Tetanus is a disease with a world wide distribution and it also occurs in New Zealand (Ellison, 1992). It is caused by an exotoxin (tetanospasmin) produced by *Clostridium tetani* in infected wounds. Only a single toxin type has been described (Odendaal and Kriek 2004). The organism is found in the environment and occasionally infects wounds causing disease in all domestic animals and man. The disease is not transmitted between animals.

Botulism is a disease of animals and man caused by the ingestion of a toxin produced in contaminated food by the organism *Clostridium botulinum*. The disease is not transmissible between animals but caused by environmental contamination of food or other ingested materials (Kriek and Odendaal, 2004). At least seven toxins are produced by a metabolically diverse group of Clostridia (Gardner, 1992; Kriek and Odendaal, 2004). The toxins produced are determined by the phage the organism is carrying. Curing of some strains of *Clostridium botulinum* of their phages and re-infection with another phage can result in conversion to a different toxigenic species - *Clostridium novyi* type A (Kriek and Odendaal, 2004). Several organisms presently classified as different species are therefore really phage types of the same species. *Clostridium botulinum* types C and D are the common causes of botulism in Australia and South Africa and type B is common in the USA (Kriek and Odendaal, 2004). Most outbreaks of botulism in New Zealand have occurred in waterfowl and are caused by *Clostridium botulinum* type C (Gardner, 1992).

### 22.1.5 Hazard identification conclusion

Tetanus and botulism are caused by environmental contaminants that occasionally infect wounds (tetanus) or contaminate food sources (botulism). They are not infectious diseases that can be controlled by any form of animal testing or quarantine. The organisms could be introduced in soil or other contaminated materials on animals or people or on imported products. Both tetanus and botulism are endemic in New Zealand and although not all toxigenic types of *Clostridium botulinum* occur here, no measures can be recommended to control the introduction of these organisms or indeed those that cause several other “clostridial diseases”. The position with regard to these “clostridial

diseases” has remained stable for many years while no attempt has been made to control the importation of new species of these organisms. For these reasons the organisms are not classified as potential hazards for this risk analysis.

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## 23. MYCOPLASMAS AND RELATED MOLLICUTES

### 23.1 Hazard Identification

*23.1.1 Aetiological agent:* Class: Mollicutes; Order: *Mycoplasmatales*; Family; *Mycoplasmataceae*; Genera *Mycoplasma*, *Ureaplasma* and *Acholeplasma*. A description of the relevant *Mycoplasma* organisms and diseases is given in Section 23.1.4 Epidemiology).

#### *23.1.2 OIE List:*

*Mycoplasma capricolum* subsp. *caripneumoniae* and *Mycoplasma agalactiae* are listed. *Mycoplasma mycoides* subsp. *mycoides* SC is listed but is primarily an organism that causes disease in cattle.

#### *23.1.3 New Zealand Status:*

*Mycoplasma capricolum* subsp. *caripneumoniae*, *Mycoplasma agalactiae* and *Mycoplasma mycoides mycoides* SC are exotic and notifiable (Ministry of Agriculture and Forestry, 2004).

*Mycoplasma mycoides* subsp. *mycoides* large colony (L C) occurs in New Zealand (Jackson and King, 2002).

Other *Mycoplasma* spp. are not listed as notifiable or unwanted organisms.

#### *23.1.4 Epidemiology*

There are many species of *Mycoplasmas* and other closely related organisms belonging to the class Mollicutes and the family Mycoplasmataceae which contains the genera *Mycoplasma*, *Ureaplasma* and *Acholeplasma*. *Acholeplasmas* are of no known veterinary significance (Anonymous, 2004b), and no evidence could be found that *Acholeplasmas* were significant human pathogens. The *Ureaplasmas* include a few species that may be significant pathogens but their role as pathogens is not yet well defined and understood. The *Mycoplasma* genus contains several important pathogens and some organisms that act as secondary or opportunistic pathogens. In this risk analysis only the known pathogens are discussed in detail and abbreviated information is given about the organisms of poorly defined pathogenicity .

*Mycoplasma* spp. consist of a diverse group of organisms that cause two clearly defined diseases of sheep and goats (contagious caprine pleuropneumonia and contagious agalactia) and a number of less well defined syndromes. Many of the organisms are not easily fitted into well defined species, they may appear similar when grown on culture medium in the laboratory, and in some cases have antigens that cross react with other species in the genus. This has led to the creation of several sub-species and periodic

reorganizations of the taxonomy of organisms in the group. Some organisms have been associated with disease syndromes that are similar to defined diseases and difficult to distinguish from them. It is not clear whether some organisms are primary pathogens or commensals or opportunistic pathogens.

Six species of *Mycoplasmas* are genetically and culturally closely related and belong to a single cluster (group) (Nicolet, 1994; Ruffin, 2001) this group consists of

*Mycoplasma mycoides* subsp. *mycoides* SC  
*Mycoplasma mycoides* subsp. *mycoides* LC  
*Mycoplasma mycoides* subsp. *capri*  
*Mycoplasma capricolum* subsp. *capripneumoniae*  
*Mycoplasma capricolum* subsp. *capricolum*  
*Mycoplasma* sp. Group 7 (this group is associated with pathology in cattle).

Other pathogens or potential pathogens of sheep and goats include:

*Mycoplasma agalactiae*  
*Mycoplasma putrefaciens*  
*Mycoplasma* group 11

The organisms of significance and the diseases/syndromes caused by them are summarized in Table 3.

Table 3. Diseases/syndromes of sheep and goats caused by *Mycoplasma* sp

| Organism                            | sheep | goats | cattle | signs                                                                          |
|-------------------------------------|-------|-------|--------|--------------------------------------------------------------------------------|
| <i>M capricolum capripneumoniae</i> | no    | yes   | no     | Pleuropneumonia (Rurangirwa and Kinyili, 2004)                                 |
| <i>M capricolum capricolum</i>      | yes   | yes   | no     | mastitis, arthritis, keratoconjunctivitis, pneumonia(Nicholas, 2004)           |
| <i>M mycoides mycoides</i> SC       | rare  | rare  | cattle | Pleuropneumonia (Thiaucourt et al., 2004)                                      |
| <i>M mycoides mycoides</i> LC       | yes   | yes   | rare   | pneumonia , arthritis, mastitis (Nicholas, 2004; Rurangirwa and Kinyili, 2004) |
| <i>M mycoides capri</i>             | ?     | yes   | no     | pneumonia , arthritis, mastitis (Rurangirwa and Kinyili, 2004)                 |
| <i>Mycoplasma</i> group 7           | no    | no    | cattle | Mastitis, poly arthritis and abortion (Hum et al., 2000)                       |
| <i>M agalactia</i>                  | yes   | yes   | no     | mastitis, arthritis, keratoconjunctivitis (Nicholas, 2004)                     |
| <i>M putrefaciens</i>               | rare  | yes   | no     | mastitis, arthritis, keratoconjunctivitis (Nicholas, 2004)                     |
| <i>M bovis genitalum</i> (group 11) | rare  | ?     | yes    | genital tract infections (Ayling et al., 2004)                                 |

**Contagious caprine pleuropneumonia (CCPP)** occurs in central, Western and northern Africa and in Turkey and the Middle-East (Lefevre and Thiaucourt, 2004). It is a distinct disease caused by *Mycoplasma capricolum* subsp. *capripneumoniae* (Thiaucourt and Bolske, 1996; Rurangirwa and Kinyili, 2004). This organism was formerly known as the F38 biotype (Leach et al., 1993). CCPP is a disease of goats (not sheep). It is very contagious and in a naïve flock may result in up to 100% morbidity and 60-70% mortality (Mare, 1994; Wesonga et al., 1998). The incubation period is variously given as 6 days to 4 weeks (Mare, 1994); 26days  $\pm$  15days (Lefevre and Thiaucourt, 2004) and 45 days (Anonymous, 2004a). In acute cases animals develop pneumonia and die within a few days. Animals that recover develop chronic lesions but in one investigation there was no evidence that they were carriers of the disease (Wesonga et al., 1998). However, the contention has also been made that a long term carrier state probably exists (Mare, 1994) and the OIE Terrestrial Animal Health Code states that chronic carriers occur (Anonymous, 2004a). In typical CCPP the disease is confined to the thorax and the lesions are characteristic. The disease can be diagnosed by the demonstration of typical lung lesions, isolation of the organism (Rurangirwa and Kinyili, 2004), serological tests or PCR (Bolske et al., 1996; Houshaymi et al., 2002; Rurangirwa and Kinyili, 2004). Serological tests include CF test, ELISA and latex agglutination tests. The latter is favoured because of its utility as a test that can be used with a drop of whole blood in the field (Rurangirwa and Kinyili, 2004) and its high sensitivity (Houshaymi et al., 2002). Serological tests are unreliable for individual animals and should be used on a flock basis (Rurangirwa and Kinyili, 2004).

Three other *Mycoplasmas* are sometimes associated with a similar respiratory syndrome but they may also cause mastitis and arthritis. These organisms are *Mycoplasma mycoides* subsp. *mycoides* LC, *Mycoplasma mycoides* subsp. *capri* and *Mycoplasma capricolum* subsp. *capricolum*. *Mycoplasma mycoides* subsp. *mycoides* SC has also been isolated from goats but is of doubtful significance as a pathogen in this species (see below). The diseases caused by these organisms are not as severe or as infectious as those caused by *Mycoplasma capricolum* subsp. *capripneumoniae*.

**Contagious agalactia** is mainly caused by *Mycoplasma agalactiae*. It occurs in Europe, Western Asia, the United States of America, and North Africa (Nicholas, 2004). It is a disease of both sheep and goats. Typically the disease causes mastitis, arthritis and keratoconjunctivitis and sometimes abortion (Bergonier et al., 1997; Ruffin, 2001). All of these signs are likely to be seen in the same flock but may not necessarily be seen in the same animal. Occasional cases of septicaemia also occur (Ruffin, 2001). In typical cases there is high morbidity and a mortality rate of up to 25% (Ruffin, 2001), but in some flocks asymptomatic carriers of the organism are known to occur. The disease is spread by the intranasal and intramammary routes and possibly through wound infection (Ruffin, 2001). It is highly contagious and spreads rapidly through a naïve flock. Following recovery from the acute disease the organism is excreted in the milk for up to a year (Bergonier et al., 1997) or even up to 8 years (Madanat et al., 2001). It can be diagnosed by isolation of the organism from milk (Nicholas, 2004), demonstration of the organism in milk by PCR (Tola et al., 1997; Madanat et al., 2001; Nicholas, 2004) or on a flock basis by serological tests including complement fixation, ELISA (Tola et al., 1997;

Madanat et al., 2001; Nicholas, 2004). Immunoblotting has also been used (Nicholas, 2004).

Similar syndromes are caused by *Mycoplasma mycoides subsp. mycoides* LC, *Mycoplasma capricolum subsp. capricolum* and *Mycoplasma putrefaciens* and it has been proposed that these species could also be considered to be causal agents of contagious agalactia (Nicholas, 2004). Some flocks carry the *Mycoplasma agalactia* without showing signs of mastitis.

**Other syndromes and *Mycoplasmas*** that are found in sheep and goats include the following:

*Mycoplasma putrefaciens* sometimes causes mastitis, arthritis and occasional abortions in goats and is included in the complex of organisms that cause contagious agalactiae. However outbreaks of disease are rarely reported and it has been described as an “opportunistic pathogen” and a “secondary agent” (Bergonier et al., 1997).

*Mycoplasma* group 11 has been isolated from the genital tract of sheep suffering infertility problems (Ayling et al., 2004). However it is only an occasional isolate from sheep. It has recently been found to be identical to *Mycoplasma bovis* which is more specifically associated with cattle.

*Ureaplasma* spp have been isolated from the genital tract of healthy sheep and sheep with signs of balanoposthitis and vulvovaginitis (Anonymous, 2002). There are a large number of articles in the literature relating to *Ureaplasma* infections in sheep. However, the *Ureaplasma* spp. studied are not identified to species level whereas *Ureaplasmas* of humans (*Ureaplasma urealyticum*) and cattle (*Ureaplasma diversum*) usually are. It has been suggested that each animal species is colonized by a characteristic group of *Ureaplasmas* and that they may be complicating agents in several infections (Howard, 1984). Although in some investigations they appeared to be pathogens of sheep (Livingstone and Gauer, 1982) several attempts to demonstrate a role of *Ureaplasma* spp. in experimental infections have resulted in inconclusive results (Ball et al., 1985; Ball et al., 1986; Ball and McCaughey, 1987). Natural infections were described as causing mild inflammation of the vulva but it was suggested that the signs “were not sufficiently marked to be useful in diagnosing the infection by clinical examination” (McCaughy and Ball, 1985). Sheep and goat strains cross react serologically (Howard and Pocock, 1983; Koshimizu et al., 1984). The role played by ureaplasmas in the pathogenesis of any disease syndrome of sheep and goats remains uncertain. For the purposes of this risk analysis they will be regarded as opportunistic pathogens. However, as they are not known to occur in New Zealand steps should be taken to prevent their introduction.

*Mycoplasma* spp. are also carried in the external ear (Cottew and Yeats, 1982) and in ear mites and tonsils (Bergonier et al., 1997). It is not known what role these *Mycoplasmas* and mites play as a source of diseases.

*Mycoplasma mycoides* subsp. *mycoides* SC is a serious pathogen of cattle causing contagious bovine pleuropneumonia (CBPP). It has also been isolated from goats (Cottew and Yeats, 1978; Kusiluka et al., 2000; Kusiluka et al., 2001). However, goats have limited susceptibility following experimental transmission and it is unlikely that goats play any role as reservoirs of the organism in for CBPP (Thiaucourt et al., 2004). For these reasons it is not considered to be a pathogen of sheep and goats in this risk analysis. However, since *Mycoplasma mycoides* subsp. *mycoides* SC can be isolated from sheep, steps should be taken to prevent its introduction.

*Mycoplasma mycoides mycoides* LC (Trichard et al., 1993) has been described as being associated with a specific balanoposthitis/vulvovaginitis syndrome. However the complex nature of the interaction of the various organisms associated with the syndrome (Van Vuuren and Trichard, 2004) invite some doubts about the specific and primary cause of the condition. In New Zealand the organism as been associated with polyarthritis and pneumonia in goats and calves (Jackson and King, 2002).

*Mycoplasma bovis* has been isolated from a mastitic sheep (Ayling et al., 2004) and experimental infection of the udder of sheep is has been described (Bocklisch et al., 1991). However, it is not a pathogen of sheep although it has been suggested that sheep may act as a reservoir of the organisms for cattle (Pfutzner and Sachse, 1996).

**Mycoplasma spp in semen and embryos.** There is evidence that several *Mycoplasma* spp. may infect the genital tract and germplasm. This has been discussed in Section 25.2.

**New Zealand situation:** Of the pathogens described above only *Mycoplasma mycoides mycoides* LC of the *Mycoplasma mycoides* cluster has been isolated in New Zealand (Jackson and King, 2002).

*Mycoplasma* spp. and closely related organisms that have been identified in New Zealand include *Mycoplasma ovipneumoniae* and *Acholeplasma laidlawii* that have been frequently isolated (Belton, 1990, 1996), and *Mycoplasma arginini* (Belton, 1990, 1996; Anonymous, 2002) and *Ureaplasma* spp. (Thornton and Wake, 1997). *Mycoplasma conjunctivae* has been identified in sheep and goats (Motha et al., 2003; Motha, 2003). *Mycoplasma ovipneumoniae* may play a role in some lung infections but is not a primary pathogen and the *Ureaplasma* spp. may be important but their role in sheep has not been clearly defined.

#### 23.1.5 Hazard identification conclusion

Diseases caused by *Mycoplasma* spp. are economically important and it is possible that several *Mycoplasma* spp. can be carried in semen. Three species are classified as exotic notifiable organisms. For the purposes of this analysis the following species are considered to be potential hazards:

*Mycoplasma mycoides* subsp. *mycoides* SC  
*Mycoplasma mycoides* subsp. *capri*

*Mycoplasma capricolum* subsp. *capripneumoniae*  
*Mycoplasma capricolum* subsp. *capricolum*  
*Mycoplasma agalactiae* (contagious agalactia)  
*Mycoplasma putrefaciens*  
*Mycoplasma bovis* (group 11)  
*Ureaplasma* spp.

## 23.2 Risk Assessment

### 23.2.1 Release Assessment

#### 23.2.1.1 Semen (sheep and goats)

There is little information about the excretion of *Mycoplasmas* in the semen of sheep and goats. *Mycoplasma agalactiae* has been listed as an organism that is known to be present in sheep semen and *Mycoplasma* spp. (CCPP) as an organism that is likely to be present in semen (Hare, 1985). Bergonnier reviewed the literature and quotes shedding of *Mycoplasma agalactia* in milk for up to 5 months in a goat and 7 months in ewes and in vaginal swabs for 6 to 10 weeks. He also states that “Adult males and dry and non-pregnant females can shed *Mycoplasmas* by rectal, nasal or even ocular or genital routes” (Bergonier et al., 1997). It has also been stated that the organism can be excreted from the male genitourinary tract and that vaginal swabs are suitable samples for isolation of the organism (Madanat et al., 2001). Both *Mycoplasma agalactiae* and *Mycoplasma mycoides mycoides* LC have been isolated from cervico vaginal mucous, uterine mucosa, semen and preputial swabs of sheep (Kapoor et al., 1984). *Mycoplasma* sp. were isolated from vaginal, uterine and semen samples of sheep (Livingstone and Gauer, 1983). *Mycoplasma agalactiae*, *Mycoplasma capricolum capricolum* and *Mycoplasma mycoides mycoides* LC and SC infections of the male genital tract have been described (Bergonier et al., 1997). *Mycoplasma mycoides mycoides* LC was described as the cause of a specific balanoposthitis/vulvovaginitis syndrome (Trichard et al., 1993). However, combinations of several other mollicutes and other bacteria may be involved in this syndrome. Therefore the likelihood that semen could be infected with any of the *Mycoplasma* spp. is non-negligible.

#### 23.2.1.2 Embryos (sheep and goats)

*Mycoplasma mycoides mycoides* LC has been isolated from internal organs of aborted calves and *Mycoplasma capricolum capricolum* from the swollen joints of kids and lambs born at full term (Bergonier et al., 1997). These findings indicate that infections in these animals could have taken place *in utero*. Both *Mycoplasma agalactiae* and *Mycoplasma mycoides mycoides* LC have been isolated from cervico vaginal mucous, uterine mucosa, semen and preputial swabs of sheep (Kapoor et al., 1984). *Mycoplasma* sp. were isolated from vaginal, uterine and semen samples of sheep (Livingstone and Gauer, 1983). These findings all indicate that *Mycoplasma* spp. could contaminate the female genital tract and contaminate embryos. Therefore it is reasonable to assume that *Mycoplasma* spp. could contaminate *in vivo* harvested embryos. The use of antibiotics in the preparation of

embryos is unlikely to eliminate *Mycoplasma* spp. (Riddell et al., 1989; Visser et al., 1999; Bielanski et al., 2000).

#### *23.2.2 Exposure Assessment*

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

#### *23.2.3 Consequence Assessment*

##### 23.2.3.1 Introduction of semen and embryos from sheep and goats

Insemination or transplantation of infected germplasm into susceptible recipients is assumed to be likely to result in infection of the recipients. Depending on the organism involved the animals could develop clinical signs of disease or become asymptotically infected (Section 23.1.4). These animals could infect other animals in contact with them and lead to the establishment of the organism in New Zealand and consequently the disease syndromes associated with them. This would in turn lead to losses in productivity and financial performance of affected flocks.

##### 23.2.3.2 Other consequences

Since the organisms are not zoonotic there would be no consequences for human health. The organisms have not been described as affecting wild or feral animals and probably only feral goats and that would be susceptible to infection with the organisms.

##### 23.2.3.3 Conclusions of the consequence assessment

Since the use of infected germplasm could lead to the establishment of new *Mycoplasma* spp. in New Zealand the consequences are non-negligible.

#### *23.2.4 Risk Estimation*

Release, exposure and consequence assessments for all commodities are considered to be non-negligible, therefore according to the methodology used in this risk analysis (Section 4.2) risk is non-negligible.

### **23.3 Risk Management**

#### *23.3.1 Risk Evaluation*

Risk is non-negligible and therefore risk management measures are justified to reduce the risk to an acceptable level.

#### *23.3.2 Option Evaluation*

#### 23.3.2.1 Risk management objective

The objective is to prevent the importation of germplasm contaminated with any pathogenic *Mycoplasma* spp.

#### 23.3.2.2 Options available

Since long term carriers of infection occur in both CCPP and contagious agalactia and the position with many of the other organisms of concern is not known, on its own quarantine is not considered to be a suitable measure for ensuring that donors are not infected with *Mycoplasmas* or *Ureaplasmas*. Since it is unlikely that there will be officially accredited flocks in any country, it will be necessary to undertake flock testing to locate suitable disease free flocks from which donors can be selected.

The OIE Terrestrial Animal Health Code does not contain any recommended measures relating to contagious agalactia and contagious caprine pleuropneumonia for the importation of germplasm or the establishment and maintenance of disease-free flocks. However, the OIE recommendations for live animals relating to contagious caprine pleuropneumonia, (Anonymous, 2004a) could be adapted for donors of germplasm.

Donors could be selected from closed flocks in which there is no known history of mycoplasmal infections. Potential donor flocks could be subjected to serological testing using OIE recommended tests (Nicholas, 2004; Rurangirwa and Kinyili, 2004). The number of animals sample should be sufficient to detect infection in a flock with a prevalence of 1% with a 99% confidence. The serological tests could be conducted with *Mycoplasma agalactiae*, *Mycoplasma capricolum* subsp. *capricolum*, *Mycoplasma mycoides* subsp. *mycoides* and *Mycoplasma capricolum* subsp. *capripneumoniae* antigens. Animals from selected flocks could then be transferred to disease free germplasm collection facilities in a disease-free zone and held in quarantine for 45 days before collection of germplasm. The 45 day quarantine relates to the incubation period for CCPP as stipulated by OIE (Anonymous, 2004a). Donors could be tested serologically 2-4 weeks after the collection of germplasm is completed.

The options suggested above provide safeguards primarily against CCPP and contagious agalactiae and related syndromes caused by *Mycoplasma agalactiae* and members of the *mycoides* cluster. These measures should be further supported by additional measures to prevent the introduction of the other *Mycoplasma* spp. that are more likely to be opportunistic or secondary pathogens. Therefore aliquots of semen or embryos could be submitted to cultural examination using culture methods described in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (Nicholas, 2004; Rurangirwa and Kinyili, 2004). Isolated *Mycoplasmas* could be identified and a decision made as to whether the germplasm could be exported to New Zealand.

Antibiotics normally used in semen dilution and embryo preparation are not completely effective in eliminating *Mycoplasma bovis* or *Mycoplasma bovis genitalum* from bovine semen and embryos (Riddell et al., 1989; Visser et al., 1999; Bielanski et al., 2000).

Therefore antibiotics are unlikely to be effective against the range of Mollicutes described above.

#### 23.3.2.3 Recommended sanitary measures

Either:

- i. a) Donors should be selected from countries that are free from CCPP and contagious agalactia; and
- a) Aliquots of semen and embryos from each collection batch should be cultured for *Mycoplasma* and *Ureaplasma* spp. and all isolates identified. Once isolates have been identified a decision should be made about whether to allow importation of the germplasm;

Or:

- ii. a) Flocks from which donors are selected should be subjected to serological testing using OIE recommended tests (Nicholas, 2004; Rurangirwa and Kinyili, 2004). The numbers of animals sampled for testing should be sufficient to detect infection in a flock with 99% confidence at a flock prevalence of 1%. The serological tests should be conducted with *Mycoplasma agalactiae*, *Mycoplasma capricolum* subsp. *capricolum*, *Mycoplasma mycoides* subsp. *mycoides* and *Mycoplasma capricolum* subsp. *capripneumoniae* antigens. Donors should be selected only from flocks that are shown to be free from *Mycoplasma* spp. antibodies; and
- b) Individual donors should be isolated in a collection facility situated in a CCPP-free zone, for the 45 days immediately before germplasm collection; and
- c) Donors should be tested with negative results, by OIE recommended serological tests with *Mycoplasma agalactiae*, *Mycoplasma capricolum* subsp. *capricolum*, *Mycoplasma mycoides* subsp. *mycoides* and *Mycoplasma capricolum* antigens, between 14-28 days after completion of germplasm collection; and
- d) Aliquots of semen and embryos from each collection batch should be cultured for *Mycoplasma* and *Ureaplasma* spp. and all isolates should be identified. Once isolates have been identified a decision should be made about whether to allow importation of the germplasm.

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## 24. HAEMORRHAGIC SEPTICAEMIA

### 24.1 Hazard Identification

24.1.1 *Aetiological agent: Pasteurella multocida* capsular serotypes B or E

24.1.2 *OIE List*: Listed

24.1.3 *New Zealand Status*: Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

#### 24.1.4 *Epidemiology*

Haemorrhagic septicaemia is a disease of cattle and buffaloes caused by *Pasteurella multocida* capsular serotypes B and E (Blood and Radostits, 1989; Bastianello and Henton, 2004). The B serotypes occur in Asia and both B and E are found in Africa. Other serotypes of *Pasteurella multocida* are implicated as causing respiratory disease but are not etiological agents for haemorrhagic septicaemia. The disease is described as a disease of buffalo and cattle in text books (Blood and Radostits, 1989; Bastianello and Henton, 2004), and the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (Chandrasekaran and Townsend, 2004) and in many journal articles (Anonymous. 1981). It has occasionally been reported in sheep (Bastianello and Henton, 2004). A search of two electronic databases failed to find any reference to the disease occurring naturally in sheep and goats. Rahmani sheep were resistant to experimental infection with 1/100, 5 or 10 doses of *Pasteurella multocida* that killed a calf with classic signs of the disease within 36 hours (Barakat et al., 1976). Goats kept in contact with experimentally infected buffaloes that exhibited overt disease and goats infected subcutaneously, intranasally and orally showed a high resistance to infection. 0/16 in contact animals, 2/21 subcutaneously infected animals, 2/12 intranasally infected animals and one out of 12 orally infected goats died. The conclusion was that goats are highly resistant to the disease and are unlikely to serve as a reservoir host (Wijewardana et al., 1986). De Alwis also found goats to be highly resistant to infection (De Alwis, 1992).

#### 24.1.5 *Hazard identification conclusion*

Haemorrhagic septicaemia is not a disease of sheep and goats. The importation of sheep and goat embryos and semen does not pose any threat with respect to this disease and the organism is not classified as a potential hazard in this risk analysis.

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## 25. SALMONELLOSIS

### 25.1 Hazard Identification

#### 25.1.1 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 using correct conventions the names such as *dublin* and *abortus ovis*, which do not have species status, should not be italicised. However, in this review for the sake of simplicity and convenience they are italicised as though they were species.

This analysis is concerned mainly with three important serovars: *Salmonella abortus ovis*, *Salmonella dublin* and *Salmonella typhimurium* but also refers to other serovars. Phage typing of *Salmonellas* is also commonly used to classify strains. In the case of *Salmonella typhimurium*, only the definitive phage type (DT) 104 is 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 health (Hogue et al., 1997; Jones et al., 2002). It is now widely distributed in the world

#### 25.1.2 OIE List:

*Salmonella abortus ovis* is a listed disease in the OIE Terrestrial Animal Code (Anonymous, 2004b) other species are covered in the OIE Manual of Diagnostic Tests and Vaccines under “Diseases not covered by List A and List B” (Anonymous, 2004a).

#### 25.1.3 New Zealand Status:

*Salmonella abortus ovis* is exotic and notifiable (Ministry of Agriculture and Forestry, 2004). *Salmonella dublin* is exotic and notifiable (Ministry of Agriculture and Forestry, 2004) *Salmonella typhimurium* is endemic in New Zealand but phage type 104 has only occurred rarely in humans and not in animals. It is classified in the category of “other unwanted organisms” (Ministry of Agriculture and Forestry, 2004).

#### 25.1.4 Epidemiology

*Salmonella* spp. isolated in New Zealand are identified to serovar and phage type by the Environmental Science and Research (ESR) laboratory and recorded on a database (ESR, 2003 and 2004). Isolations from medical and animal health laboratories are recorded.

*Salmonella dublin* and *Salmonella abortus ovis* have not been isolated in New Zealand. *Salmonella dublin* occurs most commonly in cattle but also occurs in sheep. *Salmonella abortus ovis* is more common in sheep.

*Salmonella typhimurium* is endemic in New Zealand in both animals and man but the definitive phage type DT 104 has only been isolated from humans, four times in 2003 and twice in 2004 (ESR, 2003 and 2004). It has also been isolated from three dogs in a household in which the owners suffered from diarrhoea after returning from an overseas visit (Julian, 2002). The sporadic occurrence of *Salmonella typhimurium* type DT 104 in a few cases in humans and once in dogs does not indicate that it has become established. There is no indication that the New Zealand animal population has become infected. Since it has not yet been isolated from production animals, strenuous attempts should be made to prevent its introduction.

*Salmonella abortus ovis* is believed to be host specific for sheep (Linklater, 1983). The organism was formerly of major importance as a pathogen in sheep in England but by 1983 it was rarely diagnosed (Linklater, 1983; Hogue et al., 1997; Davies, 2001, 2004) and in a report written in 1983 it was stated that no isolations of *Salmonella abortus ovis* had been made since 1976 while 85 incidents of *Salmonella dublin* infection were recorded from 1975-1981 (Sojka et al., 1983). Major reports on *Salmonella* isolations contain no reference to the organism and although reports in the literature are still found (Plagemann, 1989) they are comparatively rare. Reports that are available are often concerned with experimental infections or vaccines. Therefore, it seems that *Salmonella abortus ovis* is no longer a common disease of sheep. In contrast in England *Salmonella dublin* and *Salmonella typhimurium* are very common infections in animals (Hogue et al., 1997; Davies, 2001; Jones et al., 2002; Davies, 2004), particularly in cattle (*Salmonella dublin* and *Salmonella typhimurium*) and pigs (*Salmonella typhimurium* and rarely *Salmonella dublin*) but also in sheep (*Salmonella typhimurium* and sometimes *Salmonella dublin*). Infections in goats are seldom reported but when found they are most commonly due to *Salmonella dublin* (Blood and Radostits, 1989).

Infection is mainly by the oral route and factors such as infecting dose, the particular strain and species of *Salmonella* involved and various stress factors play a role in determining the outcome of the infection (Fenwick and Collett, 2004; Naser et al., 2004). The incubation period is from 1-7 days in experimental infections and 6-30 days after natural exposure (Naser et al., 2004). The intestine is initially infected and an inflammation of the gut is the primary lesion. Initial infection may be followed by penetration of the gut and mesenteric lymph node barrier followed by bacteraemia. Within about a week most lambs have developed a multifocal necrotic hepatitis and nephrosis (Naser et al., 2004). Animals that survive for a week may recover fully after 3-4 weeks (Naser et al., 2004). In the case of pregnant animals abortion is common (Naser et al., 2004). Serious illness and mortality following abortions caused by *Salmonella dublin* and *Salmonella typhimurium* is more common than in the case of *Salmonella abortus ovis* (Blood and Radostits, 1989). Animals that recover frequently become carriers for up to a year and sometimes for life. Three types of carriers have been described (Blood and Radostits, 1989):

- Active carriers excrete organisms constantly or intermittently. They may be infected in several organs, particularly in the gall bladder.

- Latent carriers carry the organism in lymph nodes and tonsils but may excrete organisms or even become clinical cases when stressed.
- Passive carriers do not become infected but constantly pick up organisms from the environment and re-excrete them. If removed from an infected environment, passive carriers cease to excrete organisms.

Excreted organisms contaminate the environment and become a source of infection (Blood and Radostits, 1989). Young animals are more often affected by the disease than adults and very young animals may die after a short period of bacteraemia. Serious disease and mortality also occurs in some adults particularly following abortion (Blood and Radostits, 1989). Ewes that abort excrete large numbers of organisms in their uterine discharges.

Carriers of infections can be detected by culturing faeces samples but because excretion is intermittent repeated sampling and culture is necessary (Davies, 2004). Serology can also be used but is best applied on a flock basis (Davies, 2004). However, it has been claimed that *Salmonella dublin* infections can be detected in individual cattle by the ELISA (Nielsen and Ersboll, 2004; Nielsen et al., 2004) but no comparable studies are available for sheep and goats.

#### 25.1.5 Hazard identification conclusion

*Salmonella dublin*, and *Salmonella abortus ovis* are exotic, notifiable, zoonotic organisms and *Salmonella typhimurium* type DT104 is an unwanted and zoonotic organism. Therefore these organisms are classified as potential hazards for this analysis.

## 25.2 Risk Assessment

### 25.2.1 Release Assessment

#### 25.2.1.1 Semen (sheep and goats)

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 of eggs is common. However, extrapolation should not be made from birds to sheep. 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). Rams infected intrapreputially with *Salmonella abortus ovis* excreted the organism in their semen for up to 13 days (Sanchis and Pardon, 1986). It was also concluded that *Salmonella abortus ovis* was transmitted by mating (Vodas and Marinov, 1986). In addition sheep infected with *Salmonella* spp. may be septicaemic and therefore, excretion in semen is possible. Semen could also become contaminated by faeces particularly in animals that have diarrhea and have soiled skin and hair or wool with infected faeces.

Because of the common occurrence of antibiotic resistance in *Salmonella* spp. (Wray et al., 1991; Jones et al., 2002) the use of antibiotics in semen diluents is not a reliable method of eliminating *Salmonella* spp. from germplasm. The likelihood of release of *Salmonella* spp. in semen is therefore non-negligible.

#### 25.2.1.2 Embryos (sheep and goats)

In ewes *Salmonella* spp. are excreted in vaginal discharges following abortions. Furthermore since *Salmonella* are frequently excreted in faeces and contamination of semen or embryos with faeces is possible. IETS does not list *Salmonella* in any risk category, thereby indicating that work on the transfer of the organism by embryo transfer has not been done (IETS, 2002). Because of the common occurrence of antibiotic resistance in *Salmonella* spp. (Wray et al., 1991; Jones et al., 2002) the use of antibiotics in embryo preparation cannot be regarded as a reliable method of eliminating *Salmonella* spp. from germplasm. The likelihood of release of the organism in embryos is therefore non-negligible.

#### 25.2.2 Exposure Assessment

Imported germplasm would be inseminated or implanted into susceptible sheep or goats and the likelihood of exposure is high.

#### 25.2.3 Consequence Assessment

##### 25.2.3.1 Introduction of semen and embryos from sheep and goats

The introduction of infected germplasm would result in infection of germplasm recipients and these animals could become carriers and excretors of organisms that could infect other in contact animals and people. The introduction and establishment of any of the species covered in this section could result in gradual spread of the organisms in New Zealand and the establishment of production limiting diseases of livestock and human disease.

##### 25.2.3.2 Other consequences

Because of its resistance to antibiotics establishment of *Salmonella typhimurium* DT 104 in animal populations would establish a source of infection for people and be of particular concern to human health (Hogue et al., 1997; Davies, 2001). *Salmonella dublin* and *Salmonella abortus ovis* are also zoonotic organisms 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.

#### 25.2.3.3 Consequence assessment conclusion

Introduction of infected germplasm could lead to the establishment of new *Salmonella* spp. that have the potential to cause human disease and production limiting disease of animals. Therefore the consequences are non-negligible.

#### *25.2.4 Risk Estimation*

Release, exposure and consequence assessments are all non-negligible and therefore the risk is non-negligible.

### **25.3 Risk Management**

#### *25.3.1 Risk Evaluation*

The risk of introducing a *Salmonella* spp. is non-negligible and therefore risk management measures are justified to reduce the risk to an acceptable level.

#### *25.3.2 Option Evaluation*

##### 25.3.2.1 Risk management objective

The objective is to ensure that semen and embryos imported into New Zealand are not contaminated with *Salmonella* spp.

##### 25.3.2.2 Options available

Many strains of *Salmonella* spp. are resistant to a wide range of commonly used antibiotics (Wray et al., 1991; Jones et al., 2002) 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 that the semen was free from *Salmonella* spp. Aliquots for culturing could be taken before antibiotics are added to semen or used in embryo processing.

##### 25.3.2.3 Recommended sanitary measures

It is recommended that:

Aliquots of semen and embryos and wash fluid from embryo processing should be cultured according to OIE recommended culture methods (Davies, 2004). All isolated strains of *Salmonella* spp. should be fully identified before final clearance is given to

import germplasm. Aliquots of semen for culturing should be collected in pre-enrichment media before the addition of extender containing antibiotics to the semen. Embryos that are substandard for use as embryos for transplantation should be used for culturing. If no substandard embryos are available then an aliquot of embryos should be used for culturing. In addition the first washing of embryos should be carried out in medium that does not contain antibiotics and this medium should be centrifuged and the deposit cultured. Entry of germplasm that is contaminated with any *Salmonella* of a species that is exotic to or unwanted in New Zealand should be prohibited.

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## 26. TUBERCULOSIS

### 26.1 Hazard Identification

26.1.1 *Aetiological agent: Mycobacterium bovis and Mycobacterium caprae.*

26.1.2 *OIE List: Listed*

26.1.3 *New Zealand Status:* Endemic, and the subject of an official eradication campaign by a Pest Management Strategy under the Biosecurity Act of 1993.

#### 26.1.4 *Epidemiology*

Bovine tuberculosis is primarily a disease of cattle but it affects many other species of animals. It is a rare disease of goats in most countries (Cousins et al., 2004), but in some countries, particularly in Spain, it is significant problem (Gutierrez et al., 1998; Acosta et al., 2000; Seva et al., 2002) Tuberculosis also occurs in sheep but is a rare disease. Two significant outbreaks of disease have been described in sheep in New Zealand (Cordes et al., 1981; Davidson et al., 1981). However, the prevalence was found to be only 0.00002% in nearly 11 million sheep slaughtered in New Zealand abattoirs (Allen, 1988). The disease has been found in feral goats. In an area with a high prevalence of tuberculosis in possums the prevalence was found to be 7.2% (Sanson, 1988).

The advance in molecular biology has enabled detailed study of the DNA of various isolates of *Mycobacteria* and has caused a number of name changes for the species most commonly isolated from goats. The name has been changed from *Mycobacterium bovis* to *Mycobacterium tuberculosis* subsp. *caprae* then to *Mycobacterium bovis* subsp. *caprae* and it is now proposed to call it *Mycobacterium caprae* (Aranaz et al., 1999; Aranaz et al., 2003). From an epidemiological point of view the changes in name are of minor importance, the disease of goats is similar to that of cattle in all aspects of the clinical disease, gross and histological lesions, transmission and pathogenesis of the disease. *Mycobacterium caprae* infections have been described in Spain, France, Germany, the Czech Republic and Austria (Erler et al., 2004). *Mycobacterium caprae* was not found in Italy, Ireland, Cameroon, Argentina, Australia, Canada or Iran (Prodinger et al., 2002). Isolations of *Mycobacterium caprae* have been made from goats, cattle, humans, red deer, wild boar and camels (Erler et al., 2004). In Germany from 176 human isolates that were presumed to be *Myycobacterium bovis*, made between 1999 and 2001, 55 were found to be *Mycobacterium caprae* (Kubica et al., 2003). *Mycobacterium caprae* has not been described in New Zealand (De Lisle, 2005), but it has not been specifically searched for.

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 become generalized or occasionally cause miliary tuberculosis (Cousins et al., 2004). The symptoms 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 genital tract is rare but endometritis, salpingitis and oophoritis have been described (Muscarella et al., 1974; Biolatti et al., 1989; Cousins et al., 2004).

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.

Tests used for the diagnosis of the disease have been investigated disease caused by *Mycobacterium caprae* in goats. The tuberculin test in combination with the gamma interferon test was found to give the highest sensitivity (95.8%) and specificity (96%) (Gutierrez et al., 1998). The intradermal tuberculin test has been used in sheep in New Zealand. In one study the sensitivity was found to be 81.6% and the specificity 99.6% but the sample tested was small (Cordes et al., 1981). In another study the test identified infected sheep but no estimates of sensitivity and specificity were given (Davidson et al., 1981). In goats the tuberculin test was calculated to have a specificity of 99.1% (Sanson, 1988) but the sample was small.

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

#### *26.1.5 Hazard identification conclusion*

*Mycobacterium bovis* is an organism that is the subject of a national eradication campaign run by the Animal Health Board and supported by the Ministry of Agriculture and Forestry. Therefore, introduction of organisms and establishment of new foci of infection is undesirable and both *Mycobacterium bovis* and *Mycobacterium caprae* have been classified as a potential hazard in this analysis.

### **26.2 Risk Assessment**

#### *26.2.1 Release Assessment*

##### 26.2.1.1 Semen (sheep and goats)

*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). It is assumed that the position is similar in sheep and goats. However, the disease is rare in goats and sheep and the excretion of the organism in small ruminant semen has apparently not been described. 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 release of the organism in

semen of sheep and goats is very low but non-negligible. The position with regard to *Mycobacterium caprae* is assumed to be similar.

#### 26.2.1.2 Embryos (sheep and goats)

The infection of embryos with *Mycobacterium bovis* and *Mycobacterium caprae* has not been described. However, the uterus and genital tract of cattle can be infected by *Mycobacterium bovis* (Muscarella et al., 1974; Biolatti et al., 1989; Cousins et al., 2004). *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 sheep and goats 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 and no reference to them could be found for sheep and goats. The likelihood of release of the organism in embryos is therefore, low but non-negligible.

#### 26.2.2 Exposure Assessment

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

#### 26.2.3 Consequence Assessment

##### 26.2.3.1 Introduction of semen and embryos from sheep and goats.

Insemination of cattle with infected semen led to the infection of recipients (Roumy, 1966). Insemination or transplantation of infected germplasm into susceptible sheep or goats has not been described but is likely to lead to infection of the recipients. Infected sheep or goats could develop a chronic disease and become infectious to in-contact cattle, deer, possums and other susceptible animals. Establishment of infection in cattle or deer herds and possum 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. It is unlikely that the consequences of the introduction of introduction of *Mycobacterium caprae* would differ significantly from those of introducing *Mycobacterium bovis*.

##### 26.2.3.2 Other consequences

*Mycobacterium bovis* and *Mycobacterium caprae* are zoonotic organisms and any increase in the prevalence of the disease in cattle increases the risk to humans. However, *Mycobacterium bovis* infections in humans are rare and the increase in the number of cases caused by introducing infected small ruminant 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.

#### 26.2.3.3 Consequence assessment conclusion

Since the introduction of infected germplasm could lead to new outbreaks of bovine tuberculosis and the introduction of a new closely related species or organism the consequences are non-negligible.

#### *26.2.4 Risk Estimation*

Because release, exposure and consequence assessments are all non-negligible risk is considered to be non-negligible.

### **26.3 Risk Management**

#### *26.3.1 Risk Evaluation*

Since the risk for all commodities is non-negligible, risk management measures are required to reduce risk to an acceptable level.

#### *26.3.2 Option Evaluation*

##### 26.3.2.1 Risk management objective

The objective is to prevent the collection of germplasm for export to New Zealand from any donors that are infected with *Mycobacterium bovis*.

##### 26.3.2.2 Options available

The OIE Terrestrial Animal Health Code gives no recommendations, relating to tuberculosis, in sheep and goat germplasm. However, it recommends that goats should be subjected to a tuberculin test before being introduced onto semen collection centres OIE Code, Appendix 3.2.2.2. Recommendations are made for semen and embryos from cattle and pigs.

Worldwide the prevalence of tuberculosis in sheep is very low but it is more common in goats. All OIE recommendations apply only to goats, not to sheep. In countries such as Spain tuberculosis of goats particularly due to *Mycobacterium caprae* is a problem.

Donors of germplasm could be restricted to accredited flocks in infected countries (Spain) and to animals that have been subjected to tuberculin and gamma interferon tests (Gutierrez et al., 1998).

### 26.3.2.3 Recommended sanitary measures

It is recommended that:

- i. germplasm donors should originate from countries in which tuberculosis does not occur in goats; or
- ii. if imported from countries where tuberculosis is endemic in goats donors should be sourced from accredited flocks; and
  - a) donors should be tested, with negative results, by the tuberculin test and the gamma interferon test within 30 days after the collection of germplasm.

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## 27. LYME DISEASE

### 27.1 Hazard Identification

27.1.1 *Aetiological agent: Borrellia burgdorferi*

27.1.2 *OIE List: Not listed*

27.1.3 *New Zealand Status: Unwanted, classified as “other exotic organism” (Ministry of Agriculture and Forestry, 2004).*

27.1.4 *Epidemiology*

Lyme disease is a tick-borne disease (Anonymous, 2004) of humans and other animals. The vectors for the disease are ticks of the genus *Ixodes*, particularly *Ixodes scapularis* and *Ixodes pacificus* (Anonymous, 2004) in the United States and *Ixodes ricinus* and *Ixodes persulcatis* in Europe (Ogden et al., 1997; Alekseev and Dubinina, 2000; Hubalek et al., 2004; Utenkova et al., 2004). The main reservoir of the organism in the United States is the white footed mouse but many other rodents and deer are also hosts and play a role in the complex epidemiology of the disease (LoGiudice et al., 2003). It is not known to be transmitted by other means. Specifically there are no reports of sexual transmission of the disease and attempts to transmit the disease sexually in rats and hamsters failed (Moody and Barthold, 1991; Woodrum and Oliver, 1999).

Sheep and goats have been shown to have antibody to the organism (Fridriksdottir et al., 1992a; Ciceroni et al., 1997; Travnicek et al., 2002), but the clinical disease has not been described except in a case where the disease was suspected in two lambs (Fridriksdottir et al., 1992b). However, the cause of the disease in the lambs could not be confirmed as the organism could not be isolated from them. In one investigation it was found that sheep did not support systemic infections of the organism but the organism was transmitted amongst ticks when they co-fed on sheep (Ogden et al., 1997).

27.1.5 *Hazard identification conclusion*

*Borrelia burgdorferi* is exotic (Ministry of Agriculture and Forestry, 2004) and the cause of a significant human disease therefore it is considered to be a potential hazard in this analysis.

## 27.2 Risk Assessment

### 27.2.1 Release Assessment

#### 27.2.1.1 Semen and embryos

There is no evidence that *Borrelia burgdorferi* can be excreted in germplasm. Transmission of the disease by any means other than by tick bite has not been described. Therefore the likelihood that the disease would be transmitted by germplasm is considered to be negligible.

### 27.2.4 Risk Estimation

Since the release assessment is negligible, according to the methods used in this assessment (Section 4.2), the risk is considered to be negligible.

## 27.3 Risk Management

### 27.3.1 Risk Evaluation

Since the risk is negligible, risk management measures are not justified.

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## 28. LEPTOSPIROSIS

### 28.1 Hazard Identification

#### 28.1.1 Aetiological agent:

*Leptospira* spp. There are over 200 *Leptospira* serovars classified into 23 serogroups (Bolin, 2004). A newer and alternative scheme based on genomic considerations classifies the pathogenic organisms into several species. For the purposes of this risk analysis serovars are written as if they were single species e.g. *Leptospira hardjo*, *Leptospira pomona* etc.

#### 28.1.2 OIE List: Listed.

#### 28.1.3 New Zealand Status:

*Leptospira hardjo*, *Leptospira pomona*, *Leptospira balcanica*, *Leptospira copehageni*, *Leptospira ballum* and *Leptospira tarrasovi* have been isolated from animals in New Zealand (Worthington, 1982). 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, 2004a). Other *Leptospira* spp. are classified by MAF as “other exotic organisms” (Ministry of Agriculture and Forestry, 2004).

#### 28.1.4 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 (maintenance host) 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). *Leptospiras* seldom cause economically important diseases in their maintenance hosts. In maintenance hosts the *Leptospira* may localise in the kidneys and the animals may continue to excrete the organism in their urine for years. Sheep can remain carriers of *Leptospira hardjo* for at least 8 months (Farina et al., 1996). *Leptospira hardjo* infection in sheep may have led to an increase in cases of leptospirosis in meat plant operators in New Zealand (Hill, 2003). Descriptions of disease in sheep and goats are rare and reports are usually confined to reporting on the incidence of serological titres. 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).

The disease is 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 of infection (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 ELISA tests are also available, the ELISA tests lack serovar specificity (Bolin, 2004). Leptospirosis is seldom the cause of economically serious disease in animals. It is mainly of concern because it is a zoonotic disease that occasionally causes serious disease in humans (Thornley et al., 2002). At the 2004 general session of OIE members voted to remove the leptospirosis chapter from the OIE Terrestrial Animal Health Code because of the ubiquity of the organism and the absence of meaningful control programmes and effective treatments in live animals (Pharo, 2005)

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

#### *28.1.5 Hazard identification conclusion*

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

### **28.2 Risk Assessment**

#### *28.2.1 Release Assessment*

##### 28.2.1.1 Semen (sheep and goats)

*Leptospira* spp. are commonly excreted in the semen of bulls (Kiktenko et al., 1976; Masri et al., 1997; Heinemann et al., 1999; Heinemann et al., 2000). The position is likely to be similar in sheep and goats. 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 (Anonymous, 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 release is therefore, low but non-negligible.

#### 28.2.1.2 Embryos (sheep and goats)

*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 release is therefore considered to be low but non-negligible.

#### 28.2.2 Exposure Assessment

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

#### 28.2.3 Consequence Assessment

##### 28.2.3.1 Introduction of semen and embryos from sheep and goats.

According to Blaha “the genital excretions of animals can function as primary infection sources” for leptospirosis (Blaha, 1989). Therefore insemination or transplantation of infected, imported germplasm that has not been treated with antibiotics, could lead to infection of the recipients. Infection of a recipient would be dependant on the particular *Leptospira* serovar being one to which sheep and/or goats are susceptible. If an infected recipient is able transmit the organism to a suitable maintenance host during the period it is excreting the organisms in its urine, the organism could become established.

##### 28.2.3.2 Other consequences

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, 2004a).

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

#### 28.2.3.3 Consequence analysis conclusion

The likelihood of establishment of new *Leptospira* serovars is low but non-negligible. Establishment of new serovars could cause sporadic cases of disease in humans. Therefore the consequences of establishment are non-negligible.

#### *28.2.4 Risk Estimation*

Since release, exposure and consequence assessments are non-negligible, risk is considered to be non-negligible.

### **28.3 Risk Management**

#### *28.3.1 Risk Evaluation*

Since risk is non-negligible, risk management measures are justified to reduce the risk to an acceptable level.

#### *28.3.2 Option Evaluation*

##### 28.3.2.1 Risk management objective

The object is to ensure that imported germplasm does not contain viable *Leptospiras*.

##### 28.3.2.2 Options available

Because of the occurrence of long term carriers of infection, quarantine is not a suitable option. Diagnosis by means of serology 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. Testing of aliquots of semen or embryos by culture or PCR is problematic because isolation of organisms is difficult and selection of primers for PCR that will recognize all serovars has not yet been achieved. The remaining option is to rely on the use of antibiotics in the preparation of semen and embryos or for the treatment of donors.

##### 28.3.2.3 Recommended sanitary measures

Germplasm should be prepared according to the recommendations of OIE Terrestrial Animal Health OIE Terrestrial Animal Health Code (Anonymous, 2003, 2004b) and IETS (IETS, 2002) including the use of penicillin and streptomycin in semen diluents and

embryo washing media as recommended in the OIE Terrestrial Animal Disease Code (2004) for bovine semen (Article 3.2.1.9) and embryos ( Code Section 3.3.2.4)

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## 29. BABESIOSIS

### 29.1 Hazard Identification

29.1.1 *Aetiological agent: Babesia ovis and Babesia motasi*

29.1.2 *OIE List:* Not listed.

29.1.3 *New Zealand Status:* *Babesia* spp. are listed as notifiable organisms (Ministry of Agriculture and Forestry, 2004).

29.1.4 *Epidemiology*

*Babesia motasi* and *Babesia ovis* occur in sheep and goats. *Babesia ovis* occurs widely in southern Europe (Yeruham et al., 1995; Friedhoff, 1997; Ferrer et al., 1998; Yeruham et al., 1998b; Caeiro, 1999; Papadopoulos, 1999), Central Asia and North Africa (Friedhoff, 1997). Both *Babesia motasi* and *Babesia ovis* are absent in southern Africa (Anonymous, 2004). In northern Europe *Babesia motasi* is a benign parasite (Lewis et al., 1981; Alani et al., 1987; Alani and Herbert, 1988; Friedhoff, 1997). *Babesia ovis* is generally a more pathogenic parasite but in India and Northern Africa *Babesia motasi* is more pathogenic than *Babesia ovis* (Friedhoff, 1997). Malignant babesiosis of sheep and goats is an important disease in Iran, Iraq and India (Friedhoff, 1997) and Israel and the Mediterranean basin and parts of Asia (Yeruham et al., 1995; Yeruham et al., 1998a).

Recovered sheep can carry the organism for at least 2 years (Habela et al., 1990).

Babesiosis is exclusively a tick-borne disease and the types of ticks involved include:

*Babesia ovis:* *Rhipicephalus bursa*, *Rhipicephalus tiranicus*, *Hyalomma anatolicum* and possibly *Rhipicephalus evertsi* (Yeruham et al., 1995; Friedhoff, 1997; Yeruham et al., 1998b)

*Babesia motasi:* *Haemaphysalis punctata*, *Rhipicephalus bursa* (Lewis et al., 1981; Alani and Herbert, 1988; Yeruham et al., 1995; Friedhoff, 1997; Yeruham et al., 1998b)

29.1.5 *Hazard identification conclusion*

*Babesia motasi* and *Babesia ovis* are exotic, notifiable organisms (Ministry of Agriculture and Forestry, 2004) and are therefore potential hazards for this analysis.

## 29.2 Risk Assessment

### 29.2.1 Release Assessment

#### 29.2.1.1 Semen (sheep and goats)

There is no evidence in the literature that *Babesia* spp. are transmitted in semen. The only method of transmission of the organism described in the literature is by vector ticks or by transfer of infected red blood cells. In ticks *Babesia* spp have a complex life cycle which may include sexual reproduction and transovarial infection. Except in horses infected with *Babesia equi*, which may be reclassified as *Theileria equi*, the parasites develop only within erythrocytes in their mammalian hosts and are not found in other cells (De Vos et al., 2004). Therefore, unless semen is contaminated by erythrocytes the likelihood that semen will contain parasites is negligible. It can be assumed that the parasite is not transmitted in semen. The likelihood of release in semen is therefore, considered to be negligible.

#### 29.2.1.2 Embryos (sheep and goats)

There is no evidence in the literature that *Babesia* spp. are transmitted by embryos. Since *Babesias* in their mammalian hosts are found only in erythrocytes (De Vos et al., 1994) they will not be found in oocytes. Even if embryos were to be contaminated with blood the erythrocytes and their intracellular parasites would be removed during the washing steps in the preparation of the embryos. Therefore the likelihood of release of the parasite in embryos is considered to be negligible.

#### 29.2.1.3 Release assessment conclusion

The release assessment for all commodities is negligible and therefore, according to the methodology used in this analysis (Section 4.2) the risk is considered to be negligible.

## 29.3 Risk Management

### 29.3.1 Risk Evaluation

Since the estimate of risk is negligible, risk management measures are not justified.

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## 30. THEILERIOSIS

### 30.1 Hazard Identification

#### 30.1.1 Aetiological agent:

*Theileria lestoquardi* (*hirci*), *Theileria ovis* (*recondita*), *Theileria seperata*, *Theileria* sp. (China 1) (Schnittger et al., 2003; Yin et al., 2004), and *Theileria* sp. (China 2) (Schnittger et al., 2003; Yin et al., 2004)

#### 30.1.2 OIE List :Not listed

30.1.3 New Zealand Status: *Theileria* spp. (pathogenic species) are classified as exotic notifiable organisms (Ministry of Agriculture and Forestry, 2004).

#### 30.1.4 Epidemiology

The *Theileria* species *Theileria ovis* (*recondita*) and *Theileria seperata* are benign species that usually cause asymptomatic or mild infections in non-splenectomised animals (Alani and Herbert, 1988a, b; Sayin et al., 1997; Papadopoulos, 1999; Mazyad and Khalaf, 2002; Anonymous, 2004; Lawrence, 2004) Experimental infection with these species is often done in splenectomised animals that are more susceptible to infection (Alani and Herbert, 1988a, b). *Theileria lestoquardi* (formerly *hirci*) (Hashemi-Fesharki, 1997; Leemans et al., 1999a; Leemans et al., 1999b; Razmi et al., 2003; Salih et al., 2003; Lawrence, 2004) and the two newly described Chinese strains (Guo et al., 2002; Schnittger et al., 2003; Yin et al., 2004) are pathogenic for sheep and goats and may cause high mortality in young susceptible animals (Luo and Yin, 1997; Guo et al., 2002).

A high number of animals in endemic areas are carriers of infection (Guo et al., 2002) or serologically positive (Salih et al., 2003).

Natural transmission of all *Theileria* spp. is only by ticks. The parasite can be experimentally transmitted by inoculation of blood. Tick species that are probably involved in natural transmission include:

*Theileria ovis* (*recondita*): *Haemaphysalis punctata* (Alani and Herbert, 1988a)

*Theileria lestoquardi*: *Hyalomma anatolicum* (Alani and Herbert, 1988a; Friedhoff, 1997; Brown et al., 1998; Kirvar et al., 1998; Razmi et al., 2003), *Hyalomma impeltatum* (Hashemi-Fesharki, 1997; El-Azazy et al., 2001).

*Theileria* sp (China1): *Haemaphysalis qinghaiensis* (Luo and Yin, 1997; Yin et al., 2002).

This list of vectors is not complete and transmission by other species of ticks probably occurs.

A literature search did not reveal any evidence that the New Zealand cattle tick (*Haemaphysalis longicornis*) is a vector of any *Theileria* species of sheep or goats. This finding is confirmed by the investigations done by Heath (Heath, 2002). However ticks of the *Haemaphysalis* genus are competent vectors of the pathogenic *Theilerias of sheep*, and *Haemaphysalis longicornis* is a competent vector of three species of *Theilerias* that occur in cattle (Heath, 2002).

#### *30.1.5 Hazard identification conclusion*

Pathogenic species of *Theileria* are classified by MAF as exotic unwanted organisms (Ministry of Agriculture and Forestry, 2004). The three pathogenic organisms described above (*Theileria lesquardi*, China 1 and China 2) are therefore considered to be potential hazards for this analysis. *Theileria ovis (recondita)* and *Theileria seperata* are not regarded as potential hazards in this risk analysis.

### **30.2 Risk Assessment**

#### *30.2.1 Release Assessment*

##### 30.2.1.1 Semen and embryos (sheep and goats)

There is no evidence that any *Theileria* spp. can be transmitted by semen or embryos, all evidence points to the fact that the parasites are transmitted only by ticks (Lawrence, 2004). Therefore the likelihood that the disease could be introduced with imported germplasm is negligible.

#### *30.2.2 Risk Estimation*

The likelihood of release was found to be negligible for all commodities and according to the methodology used in this analysis (Section 4.2) this means that the risk is considered to be negligible.

### **30.3 Risk Management**

#### *30.3.1 Risk Evaluation*

Since risk is estimated to be negligible, there is no justification for implementation of risk management measures.

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## 31. TRYPANOSOMES (TSETSE FLY TRANSMITTED)

### 31.1 Hazard Identification

31.1.1 *Aetiological agent: Trypanosoma brucei brucei, Trypanosoma congolense and Trypanosoma vivax*

31.1.2 *OIE List:* Listed as a cattle disease, but not as a sheep and goat disease.

31.1.3 *New Zealand Status:* Exotic and notifiable (Ministry of Agriculture and Forestry, 2004).

#### 31.1.4 *Epidemiology*

Tsetse fly transmitted trypanosomiasis is caused by three species of extracellular blood parasites. *Trypanosoma brucei brucei* and *Trypanosoma congolense* are transmitted only by or predominantly by tsetse flies and do not occur outside of tsetse fly infested areas of Africa. *Trypanosoma vivax* is also carried by tsetse flies but it has become established in South America where tsetse flies are not present (Connor and Van den Bossche, 2004). The vector in South America is not known but it is assumed to be one or more species of biting flies. Mechanical transmission of *Trypanosoma vivax* by the African tabanid *Aylotus fuscipes* has been demonstrated (Desquesnes and Dia, 2004) and *Trypanosoma vivax* is the predominant species found in areas of Africa where tsetse fly control is carried out (Magona et al., 2000). However, in areas bordering infested areas where mechanical transmission was thought to be the only means of transmission, improved trapping methods have revealed low densities of tsetse flies (Connor and Van den Bossche, 2004)

Sheep and goats are susceptible to infection but are more resistant than cattle and often survive well in areas in which cattle farming is severely compromised by the occurrence of the disease (Connor and Van den Bossche, 2004).

#### 31.1.5 *Hazard identification conclusion*

*Trypanosoma congolense, Trypanosoma brucei brucei and Trypanosoma vivax* are exotic notifiable organisms (Ministry of Agriculture and Forestry, 2004) and are therefore classed as potential hazards in this analysis

## 31.2 Risk Assessment

### 31.2.1 Release Assessment

#### 31.2.1.1 Semen (sheep and goats)

Many studies have demonstrated that infection with *Trypanosoma* spp. causes a rapid deterioration of semen quality in infested sheep, goats and cattle and they may become sterile (Sekoni et al., 1988; Ngeranwa et al., 1991; Sekoni, 1992; Sekoni et al., 2004b, a); (Akpavie et al., 1987). Semen quality improves on treatment of the parasitaemia (Akpavie et al., 1987). However, despite careful examination of semen in these and many other studies, excretion of trypanosomes in semen has not been described. Since the parasite is not excreted in the semen of cattle, goats and sheep the likelihood of transmission in semen is considered to be negligible.

#### 31.2.1.2 Embryos (sheep and goats)

No evidence was found that the parasites are transmitted by embryos. The parasites are typically extracellular blood parasites and would be seen when semen is examined microscopically. They are not found attached to cells and would therefore be removed from embryo preparations by the washing process. For these reasons it is concluded that the likelihood of trypanosomes infecting embryos is negligible.

#### 31.2.1.3 Risk Estimation

Since the release assessment was found to be negligible for all commodities it is considered that the likelihood of introducing trypanosomes in germplasm is negligible. Therefore, according to the methods used in this analysis (Section 4.2) risk is considered to be negligible.

## 31.3 Risk Management

### 31.3.1 Risk Evaluation

Since risk is negligible, risk management measures are not justified.

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## 32. ANAPLASMOSIS

### 32.1 Hazard Identification

32.1.1 *Aetiological agent: Anaplasma ovis and Anaplasma mesaeterum.*

32.1.2 *OIE List:* Not listed.

32.1.3 *New Zealand Status:* *Anaplasma* spp are classified as exotic, notifiable organisms (Ministry of Agriculture and Forestry, 2004).

32.1.4 *Epidemiology*

*Anaplasmas* are intracellular parasites of red blood cells.

*Anaplasma mesaeterum* has been described as occurring only on offshore Dutch Islands in the North Sea (Uilenberg et al., 1979; Uilenberg et al., 1980). It does not generally cause serious disease. It is transmitted by ticks and it is most unlikely that it could be transmitted by semen or embryos.

*Anaplasma ovis* has a world-wide distribution and is probably found wherever suitable tick vectors are found. The organism generally causes sub-clinical disease (Yeruham et al., 1992; Stoltz, 1994; Hashemi-Fesharki, 1997) in sheep and does not cause economically significant losses. The disease is transmitted by ticks and although iatrogenic transmission has been postulated mechanical transmission by biting arthropods has not been demonstrated (Stoltz, 2004). Ticks from the genera *Rhipicephalus*, *Dermacentor*, *Hyalomma*, *Haemophysalis*, *Ornithodoros* and *Ixodes* have been implicated as carriers of the organism (Kocan and Stiller, 1992; Stoltz, 1994; Friedhoff, 1997; Hashemi-Fesharki, 1997).

Clinical signs are more likely to be seen in goats than in sheep (Stoltz, 2004). Overt disease, abortion and mortality may occur, probably when there are concurrent infections with other haemoparasites (Stoltz, 2004). The incubation period varies from 5 days in experimentally infected animals up to 6 weeks with tick transmission. Infected animals remain life-long carriers of infection (Potgieter and Stoltz, 1994; Stoltz, 2004)

32.1.5 *Hazard identification conclusion*

*Anaplasma mesaeterum* has only been described as occurring on off shore islands in the North Sea and does not cause serious disease (Uilenberg et al., 1979; Uilenberg et al., 1980). Therefore, it has been excluded from further consideration in this analysis. *Anaplasma* spp. are exotic unwanted organisms (Ministry of Agriculture and Forestry, 2004) and *Anaplasma ovis* is considered to be a potential hazard for this risk analysis.

## 32.2 Risk Assessment

### 32.2.1 Release Assessment

#### 32.2.1.1 Semen (sheep and goats)

Experimental infection of sheep with *Anaplasma ovis* led to a transient illness and a more severe illness in splenectomised sheep and a marked degeneration in sperm quality. However, excretion of *Anaplasmas* in the semen was not described (Kumi-Diaka et al., 1988). Similarly infection of bulls with *Anaplasma marginale* also led to testicular degeneration and a marked decline in sperm quality but *Anaplasma marginale* was not excreted in the semen. No information was found that indicates that the organism may be excreted in semen and it is generally accepted that sheep and goat anaplasmosis is a tick-borne disease. Therefore, the likelihood that the organism can be transmitted in semen is negligible.

#### 32.2.1.2 Embryos (sheep and goats)

Bovine anaplasmosis is classified IETS as a category 4 disease, which is one “on which preliminary work has been conducted or is in progress”. No evidence could be found that *Anaplasma ovis* is transmitted by embryos. The disease is widely accepted as being a tick-borne disease. Therefore the likelihood that embryos could transmit *Anaplasma ovis* is negligible.

### 32.2.2 Risk estimation

Since the release assessment was considered to be negligible, according to the methodology adopted for this analysis (Section 4.2), risk is negligible.

## 32.3 Risk Management

### 32.3.1 Risk Evaluation

Because risk is considered to be negligible there is no justification for the implementation of risk management measures.

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## 33. ENZOOTIC ABORTION (CHLAMYDIOSIS)

### 33.1 Hazard Identification

33.1.1 *Aetiological agent: Chlamydophila abortus.*

33.1.2 *OIE List: Listed.*

33.1.3 *New Zealand Status:* Exotic, notifiable (Ministry of Agriculture and Forestry, 2004) and zoonotic organism (Aitken and Longbottom, 2004).

#### 33.1.4 *Epidemiology*

Enzootic abortion is an economically important disease of sheep and goats in countries where the disease is endemic. Ewes that abort excrete large numbers of *Chlamydophila abortus* organisms in their uterine discharges and placentas (Aitken, 1983). They may also harbour the organism in their intestinal tracts and excrete organisms in faeces. The main method of transmission of the disease is by the oral route after consuming organisms on pasture or in water contaminated by uterine discharges (Aitken, 1983). Ewes that become infected early in pregnancy may abort in the same gestation but ewes that become infected later may carry the infection until the next pregnancy and abort in the late stages of pregnancy (Aitken, 1983; Andersen, 2004). The highest number of abortions may occur in the second year after a flock becomes infected. Ewes that have aborted remain long term intestinal carriers (Aitken, 1983) and may also be chronically infected in their reproductive tracts intestinal tracts (Papp et al., 1994; Papp et al., 1998; Andersen, 2004). Bulls may remain carriers for at least 18 months (Domeika et al., 1994).

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 (Dagnall and Wilshire, 1990; Thomas et al., 1990; Domeika et al., 1994; Szeredi and Bacsadi, 2002; Aitken and Longbottom, 2004). Serological tests include the complement fixation test and ELISA, but some cross reactions occur with antibodies to *Chlamydophila pecorum* and some gram negative (Aitken and Longbottom, 2004) (Aitken and Longbottom, 2004).

#### 33.1.5 *Hazard identification conclusion*

*Chlamydophila abortus* is an exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004) and for the purposes of this analysis is considered to be a potential hazard.

## **33.2 Risk Assessment**

### *33.2.1 Release Assessment*

#### 33.2.1.1 Semen (sheep and goats)

Bulls and rams may excrete the organism in their semen and venereal transmission has been demonstrated (Storz et al., 1976; Appleyard et al., 1985; Suri et al., 1986; Domeika et al., 1994; Amin, 2003). Insemination with infected semen resulted in sero-conversion and a recovery of the organism from three out of ten ewes. Infection by natural service and by intravaginal infection resulted in sero-conversion without demonstrable infection and abortion did not occur (Appleyard et al., 1985). Although it does occur, venereal transmission is not regarded as being an important method of spread of the disease (Aitken, 1983; Appleyard et al., 1985). Since transmission by natural service and insemination is possible the likelihood of release is non-negligible.

#### 33.2.1.2 Embryos (sheep and goats)

It was shown that embryos collected from ewes that were excreting the organism in their uterine discharges were not infected and did not infect recipients of the embryos or the progeny derived from them (Williams et al., 1998). 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, 2002). The safety of embryo transfer remains to be conclusively proved. Therefore, the likelihood of introducing infection with embryos is low but non-negligible.

### *33.2.2 Exposure Assessment*

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

### *33.2.3 Consequence Assessment*

#### 33.2.3.1 Introduction of semen and embryos from sheep and goats

The organism can be transmitted venereally and could therefore, be transmitted with semen or embryos. Transmission of the disease with semen or embryos may result in abortion and it is likely that the recipient of germplasm would remain a carrier of the organism and could transmit the organism to other in contact animals by excretion of the organism in contaminated uterine discharges or faeces. Establishment of the disease would result in decreased productivity and financial performance in infected flocks where abortions would initially occur in up to 30% of ewes but in subsequent seasons abortions are likely to occur mainly in younger ewes and the prevalence of abortions may continue at around 5-10% (Aitken, 1983).

#### 33.2.3.2 Other consequences

*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). Therefore, introduction of the disease would have consequences for human health.

The organism infects sheep and goats, and more rarely cattle and deer. For this reason feral goats, deer and thar could be infected but the consequences for the environment are likely to be minor since it is a disease that is closely associated with intensive farming and is unlikely to become a problem in free ranging wildlife. The consequences for the environment are therefore likely to be minor.

#### 33.2.3.3 Consequence assessment 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.

#### *33.2.4 Risk Estimation*

Release, exposure and consequence assessments are all non-negligible and therefore risk is non-negligible.

### **33.3 Risk Management**

#### *33.3.1 Risk Evaluation*

The risk is non-negligible and risk management procedures are justified to reduce the risk to an acceptable level.

#### *33.3.1 Option Evaluation*

##### 33.3.1.1 Risk management objective

The objective is to ensure that donors of germplasm for export to New Zealand are not infected with *Chlamydophila abortus*.

##### 33.3.1.2 Options available

The OIE Terrestrial Animal Health Code provides guidelines for trade in semen but not for trade in embryos. IETS has classified *Chlamydophila abortus* in sheep in category 4 which is a Category 4 organism for which “Preliminary information has been conducted or is in progress” (IETS, 2002). No other information on embryo transfer, relating to this organism, could be found. No information was found about transmission by goat

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 a viable option

Donors could be selected from flocks that are disease free as defined in the OIE Terrestrial Animal Health Code, or from animals that have been resident on disease-free germplasm collection centres for at least 2 years. Criteria for flock and collection centre freedom include keeping a closed flock and regular testing to demonstrate freedom from the organism for at least 2 years.

Individual donors could be tested serologically using an OIE recommended test, 3 weeks after germplasm collection.

Aliquots of semen and embryos/washing fluid could be tested for *Chlamydia* by culture, PCR or antigen detection ELISA (Aitken and Longbottom, 2004).

#### 33.3.1.3 Recommended sanitary measures

It is recommended that:

- i. donors should be selected from animals that have been resident since birth or for the previous 2 years in a country that is free from the infection; or
- ii. donors should be selected from flocks or from animals kept on germplasm collection centres that are infection-free as defined in the OIE Terrestrial Animal Health Code; and
  - a) Individual donors should be tested serologically using an OIE recommended test, 2-3 weeks after germplasm collection; and
  - b) Aliquots of semen and embryos should be tested for *Chlamydia* by culture, PCR or antigen detection ELISA. In the case of embryos, wash fluid and embryos that are substandard and not suitable for export, could be used for testing; or
- iii. a sample of the flock should be tested with negative results by an OIE recommended serological test, the sample being large enough to give a 99% confidence of detecting infection at a prevalence of 1%. Donors should only be selected from flocks that are shown to be free from the infection; and
  - a) Individual donors should be tested serologically using an OIE recommended test, 2-3 weeks after germplasm collection; and

- b) Aliquots of semen and embryos should be tested for *Chlamydophila* by culture, PCR or antigen detection ELISA. 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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## 34. COXIELLA BURNETII (Q FEVER)

### 34.1 Hazard Identification

34.1.1 *Aetiological agent: Coxiella burnetii*

34.1.2 *OIE List: Listed*

34.1.3 *New Zealand Status: Exotic notifiable disease (Ministry of Agriculture and Forestry, 2004)*

34.1.4 *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 (Marrie, 1990; Marin and Raoult, 1999). 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. 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 sporadically causes abortions in both humans and animals (Raoult et al., 2002; Hatchette et al., 2003).

Transmission frequently occurs from contacts with infected uterine discharges and placentas and probably by inhalation of dust contaminated by animals and their birth products (Behymer and Riemann, 1989; Marrie, 1990; Selvaggi et al., 1996; Hawker et al., 1998; Marin and Raoult, 1999; 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 animal's coats. Sheep shed the organism in vaginal secretions for up to 2 months after parturition and may shed organisms at subsequent pregnancies (Kelly, 2004). Infection in goats is also reported to probably be limited to two seasons (Hatchette et al., 2003).

Infected animals generally remain asymptomatic thus making the determination of the incubation period and the interval to the development of antibodies problematic. Data are available for humans and the incubation period is given as 1-3 weeks and the development of detectable antibody titers takes 2-3 weeks after the onset of symptoms (Marin and Raoult, 1999). Extrapolating from this information it is assumed that infected sheep or goats will develop antibody within 6 weeks of infection.

The infection is diagnosed by serological tests or by identification or isolation of the organism (Pepin et al., 2000).

#### 34.1.5 Hazard identification conclusion

*Coxiella burnetii* is an exotic, notifiable (Ministry of Agriculture and Forestry, 2004) and zoonotic organism. Therefore, for the purposes of this analysis it is considered to be a potential hazard.

### 34.2 Risk Assessment

#### 34.2.1 Release Assessment

##### 34.2.1.1 Semen (sheep and goats)

*Coxiella burnetii* is excreted in semen of bulls and mice (Kruszewska and Tylewska-Wierzbanska, 1993; Kruszewska and Tylewska-Wierzbanska, 1997). It is likely that it would also be excreted in the semen of sheep and goats. Therefore the likelihood of release is non-negligible.

##### 34.2.1.2 Embryos (sheep and goats)

No reports were found about Q fever transmission by embryo transfer. Since *Coxiella burnetii* is frequently isolated from placentas and foetuses (Marrie, 1990; Marin and Raoult, 1999; Hatchette et al., 2003), 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 low but non-negligible.

#### 34.2.2 Exposure Assessment

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

#### 34.2.3 Consequence Assessment

##### 34.2.3.1 Introduction of semen and embryos from sheep and goats

*Coxiella burnetii* can be transmitted venereally in mice (Kruszewska and Tylewska-Wierzbanska, 1993) and probably in humans and cattle (Tylewska-Wierzbanska et al., 1991; Kruszewska et al., 1996; Milazzo et al., 2001). Therefore, it is probable that sexual transmission can occur in sheep and goats and insemination or transplantation of infected germplasm could result in infection of the recipients. Infected recipients would remain carriers for long periods and infected sheep have been shown to excrete large numbers of organisms in their birth products at parturition (Welsh, HH, Lenette EH, Albatini FR, Winn JF cited by (Marrie, 1990).

Establishment of the infection in New Zealand would be likely to have a negligible effect on the livestock industries as infected animals are usually asymptomatic. However, there is a small likelihood that the introduction into a naïve population might initially cause

some abortions. The New Zealand cattle tick could also become infected (Heath, 2002) and play an important role in the disease becoming endemic.

#### 34.2.3.2 Other consequences

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.

#### 34.2.3.3 Consequence assessment conclusion

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

#### *34.2.4 Risk Estimation*

Release, exposure and consequence assessments for all commodities are non-negligible and therefore the risk is considered to be non-negligible.

### **34.3 Risk Management**

#### *34.3.1 Risk Evaluation*

The risk of introduction is non-negligible and the implementation of risk management measures is justified to reduce risk to an acceptable level.

#### *34.3.2 Option Evaluation*

##### 34.3.2.1 Risk management objective

The objective is to ensure that *Coxiella burnetii* is not introduced in imported sheep or goat germplasm.

##### 34.3.2.2 Options available

There are no recommendations relating to Q fever in the OIE Terrestrial Animal Health Code. Infected sheep and goats would be asymptomatic, long term carriers of infection and quarantine would not prevent the entry of the organism. However, quarantine in tick free premises would 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.

Donor animals could be tested serologically 10-30 days after collection of the germplasm to ensure that they have not become infected shortly before or during germplasm collection.

It is hoped that a verified PCR test for testing germplasm for the presence of *Coxiella burnetii* DNA will become available in the future. This will allow direct testing of germplasm.

#### 34.3.2.3 Recommended sanitary measures

It is recommended that:

- i. donors should 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 should be kept in quarantine for a minimum of 4 weeks days immediately before the start of semen or embryo collection and regularly inspected and maintained in a tick-free state throughout the period of quarantine and germplasm collection; and
  - a) donors should be tested by a complement fixation test or ELISA, with negative results 14-30 days after the final collection of the germplasm. A positive test should result in prohibition of importation of the germplasm.

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## 35. HEARTWATER

### 35.1 Hazard Identification

*35.1.1 Aetiological agent:* Ehrlichia ruminantium formerly Cowdria ruminantium.

*35.1.2 OIE List:* Listed.

*35.1.3 New Zealand Status:* Exotic, notifiable disease (Ministry of Agriculture and Forestry, 2004).

*35.1.4 Epidemiology*

Heartwater is a tick-borne disease of cattle sheep, goats and some wild ruminants that is carried only by ticks of the genus *Amblyomma* (Allsopp et al., 2004).

*35.1.5 Hazard identification conclusion*

*Ehrlichia ruminantium* is an exotic notifiable disease (Ministry of Agriculture and Forestry, 2004), and therefore it is a potential hazard for the purposes of this analysis.

### 35.2 Risk Assessment

*35.2.1 Release Assessment*

35.2.1.1 Semen and embryos (sheep and goats)

Heartwater is a tick-borne disease and transmission by insemination or transplantation of embryos has not been described for it. .

*35.2.2 Risk Estimation*

Since the release assessment for all commodities was considered to be negligible, under the methods used in this risk analysis (Section 4.2) risk is considered to be negligible.

### 35.3 Risk Management

*35.3.1 Risk Evaluation*

Since risk has been estimated as negligible, there is no justification for implementing risk management measures.

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## 36. EHRLICHIOSIS

### 36.1 Hazard Identification

36.1.1 Aetiological agent: *Ehrlichia chaffeensis*, *Ehrlichia ovina* and *Anaplasma (Ehrlichia) phagocytophilum*

36.1.2 OIE List: Not listed

36.1.3 New Zealand Status: *Ehrlichia* spp. are classified as exotic, unwanted organisms (Ministry of Agriculture and Forestry, 2004).

#### 36.1.4 Epidemiology

The term ehrlichiosis is used in this risk analysis to cover a group of diseases caused by organisms belonging to the genus *Ehrlichia* which parasitise the white blood cells of humans and animals.

There has been considerable recent confusion regarding the taxonomy of the organisms in the genera *Ehrlichia*, *Anaplasma* and *Cowdria* due to the re-classification of a number of species (Uilenberg et al., 2004). The advent of DNA sequencing technology has resulted in the re-organisation of several genera. Some recently adopted or recommended nomenclature changes to organisms of interest include:

*Ehrlichia ruminantium* (formerly *Cowdria ruminantium*) the cause of heartwater in animals.

*Anaplasma phagocytophilum* (formerly *Ehrlichia phagocytophila*, *Ehrlichia equi*, *Cytoecetes phagocytophila* and *Anaplasma phagocytophila*) the cause of human granulocytic ehrlichiosis (now anaplasmosis) and tick-borne fever of ruminants.

It should be noted that *Ehrlichia chaffeensis*, *Ehrlichia canis*, *Anaplasma marginale* and *Anaplasma centrale* remain unaltered. These changes result in difficulty in following the literature relating to the species.

For the purposes of this review *Ehrlichia ruminantium* (heartwater) has been considered under a separate section, since it is the cause of a distinct and economically important disease. *Ehrlichia canis*, *Anaplasma marginale* and *Anaplasma centrale* are not parasites of sheep and goats and are not considered. The organisms discussed in this section are *Ehrlichia chaffeensis*, *Anaplasma phagocytophilum* and *Ehrlichia ovina*.

*Ehrlichia chaffeensis* is a zoonotic organism that is the etiological agent of monocytic ehrlichiosis in humans. It is carried by ticks and in the United States its main animal host is the white tailed deer (Ahrens et al., 2003; Paddock and Childs, 2003; Varela et al., 2003). In Europe deer, sheep and goats act as hosts (Dugan et al., 2000; Dugan et al.,

2004) and other animals can also be infected. It does not cause an economically significant disease in any of the animal hosts. It is a tick-borne disease and ticks from the genera *Ixodes*, *Amblyomma* and *Dermacentor* have been confirmed or suspected of carrying the organism (Holden et al., 2003; Kim et al., 2003; De Shields et al., 2004; Inayoshi et al., 2004; Varela et al., 2004). *Amblyomma americanum* is probably the major vector in the United States.

*Anaplasma phagocytophilum* is the agent of human granulocytic ehrlichiosis and tick borne fever of cattle, sheep and goats. In ruminants it causes predominantly benign infections. It also infects a wide range of animal species including deer, hares, bank voles, mice, foxes, boars, cows, and horses (Hulinska et al., 2004) and horses, sheep, cattle, dogs, cats and foxes (Sreter et al., 2004). It causes tick-borne fever, a mild disease of sheep, goats and cattle (Brun-Hansen et al., 1998; Gokce and Woldehiwet, 1999a). It may cause the exacerbation of other infections where concurrent infections occur (Scott, 1994; Gokce and Woldehiwet, 1999b). Long term carriers occur (Woldehiwet, 1983). It is a tick-borne disease with the main vectors being *Ixodes* spp. (Alberdi et al., 1998; Telford et al., 2002). A recent report from Korea indicates that *Ehrlichia phagocytophilum* DNA was identified in *Haemaphysalis longicornis*, (Kim et al., 2003) but the report does not confirm that the tick can transmit the parasite. In an extensive investigation in Russia *Anaplasma phagocytophilum* DNA was not found in *Haemaphysalis concinna*.

*Ehrlichia ovina* is a parasite of sheep but the literature on this organism is scarce and dated. Reports less than 30 years old are generally about antigens that cross react with heartwater (van Vliet et al., 1995, 1996) or of serological findings or observations of *Ehrlichias* in blood smears (Gueye et al., 1990; Gueye et al., 1993). Older reports of disease caused by the organism refer to cases in sheep that were concurrently heavily parasitised by helminths and suffering from malnutrition (Sumption and Scott, 2004). Neitz was able to transmit the organism with adult *Rhipicephalus evertsi* ticks that had been infected by feeding the nymph stages on infected sheep. The infection caused a febrile reaction which lasted for 3-10 days (Sumption and Scott, 2004). It can therefore be assumed that *Ehrlichia ovina* is a benign parasite that seldom causes a significant disease and is mainly of interest because it causes cross reactions in serological tests for heartwater. Other *Rhipicephalus* spp. may also be vectors.

#### 36.1.5 Hazard identification conclusion

Since *Ehrlichia* spp. are classified as exotic unwanted organisms, they are included as potential hazards in this analysis.

## 36.2 Risk Assessment

### 36.2.1 Release Assessment

#### 36.2.1.1 Semen and embryos (sheep and goats)

There is no evidence in the literature to suggest that any of the *Ehrlichia* or *Anaplasma* spp. can be transmitted in semen or embryos. Since all the organisms are intracellular parasites of leucocytes it is possible that the infected leucocytes could be excreted in semen if the donor animals were concurrently infected with a bacterial infection that results in leucocytes being excreted in semen. However, the parasites have not been described as occurring in other cells and it is unlikely that they would occur in oocytes and infect embryos. The likelihood of transmission of this tick-borne disease in semen or embryos is considered to be negligible.

### 36.2.2 Risk Estimation

Since the organisms included in this section are all tick-borne infections the likelihood of release in germplasm was considered to be negligible. Therefore according to the methods used in this risk analysis the risk is considered to be negligible

## 36.3 Risk Management

### 36.3.1 Risk Evaluation

Since risk is negligible, risk management measures are not justified.

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