Import risk analysis:
Pig semen

FINAL

December 2012

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Ministry for Primary Industries
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand

Tel: 64 4 894 0100
Fax: 64 4 894 0731

Science and Risk Assessment
Ministry for Primary Industries

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Lisa Oakley
Manager, Risk Analysis (Animals and Aquatic)
Ministry for Primary Industries

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CONTENTS

Contributors to this risk analysis

1. Author

Bob Worthington	Contractor, Risk Analysis, Ministry for Primary Industries, Wellington
Stephen Cobb	Principal Adviser, Risk Analysis, Ministry for Primary Industries, Wellington

2. Internal Peer Review

Stuart MacDiarmid	Principal International Adviser Risk Analysis, Ministry for Primary Industries, Wellington
Lincoln Broad	Senior Adviser, Risk Analysis, Ministry for Primary Industries, Wellington
Cathy Dyer	Senior Adviser, Animal Imports, Ministry for Primary Industries, Wellington
Richard Soons	Senior Adviser, Animal Imports, Ministry for Primary Industries, Wellington
José Derraik	Senior Adviser (Human Health), Ministry for Primary Industries, Wellington
Sandy Toy	Senior Adviser (Indigenous Fauna), Ministry for Primary Industries, Wellington
Howard Pharo	Manager, Import and Export (Animals), Ministry for Primary Industries, Wellington

3. External Scientific Review

Colin Wilks	Professor, School of Veterinary Science, Parkville, Melbourne, Australia
Maurice Pensaert	Professor, Laboratory of Virology, Faculty of Veterinary Medicine, University of Gent

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1. Executive Summary

In March 2012, the Ministry for Primary Industries (MPI) released a draft import health standard (IHS) for the importation of pig semen into New Zealand from all countries[1]. This was developed from the 2011 draft import risk analysis (IRA) for pig semen from Australia, the USA, Canada, the European Union, and Norway. The 2011 draft IRA identified six organisms of potential concern in porcine semen (blue eye disease virus, foot and mouth disease virus, Nipah virus, Teschovirus serotype 1, Venezuelan encephalitis virus, and vesicular exanthema virus) but did not assess the risk due to these since Australia, the USA, Canada, the European Union and Norway claim freedom from these diseases.

Following stakeholder consultation on the draft IHS for the importation of porcine semen into New Zealand, this final IRA has been amended to assess the risks associated with porcine semen imported from all countries.

Twelve disease agents have been assessed to be risks in the commodity. Options for risk management are presented for each of these:

- Aujeszky's disease virus
- Blue eye disease virus
- Classical swine fever virus
- Bovine viral diarrhoea virus
- Foot and mouth disease virus
- Japanese encephalitis virus
- Porcine reproductive and respiratory syndrome virus
- Porcine myocarditis virus
- Swine vesicular disease virus
- *Brucella suis*
- *Leptospira* spp.
- *Salmonella* spp.

[1] http://www.biosecurity.govt.nz/biosec/consult/draft-ihs-pigsemic.gen

2. Introduction

This risk analysis has been undertaken at the request of the Animal Imports team, MPI in order to address matters of consistency between existing IHSs and to consider new scientific information published since the existing IHSs were issued.

An initial draft *Import risk analysis: Pig semen from Australia, the USA, Canada, the European Union, and Norway* was released in August 2009 for public consultation. Two stakeholder submissions were received in response to this draft publication, from NZPork and PIC. Following clarification that stakeholder consultation sought to elicit views on the risk management options presented in an import risk analysis, a further submission from NZPork was received in February 2010. MPI considered these submissions and made significant amendments to the August 2009 document to address stakeholder concerns in the 2011 draft *Import risk analysis: Pig semen from Australia, the USA, Canada, the European Union, and Norway*.

In March 2012, MPI released a draft IHS for the importation of pig semen into New Zealand from all countries[2]. Although the conclusions of the 2011 draft import risk analysis were valid for semen imported from Australia, the USA, Canada, the European Union, and Norway, this final import risk analysis has been updated to assess the risks associated with porcine semen imported from all countries.

3. Scope

This risk analysis is limited to the risks posed by infectious agents in imported porcine semen. Genetic diseases and other risks that may be of commercial importance to importers are not assessed.

This document does not consider speculative events that could occur in the future, such as the possible establishment of disease vectors due to climate change. MPI has the flexibility to modify any IHS based on this risk analysis if future events make this appropriate.

4. Commodity definition

The commodity is defined as fresh porcine semen from Australia or frozen porcine semen from approved countries collected and processed in accordance with international guidelines set out in the *Terrestrial Animal Health Code* (OIE 2008a).

It is generally accepted that if any testing of animals or semen is necessary to support certification of the health status of donors, it is carried out at a laboratory approved by the veterinary administration of the exporting country.

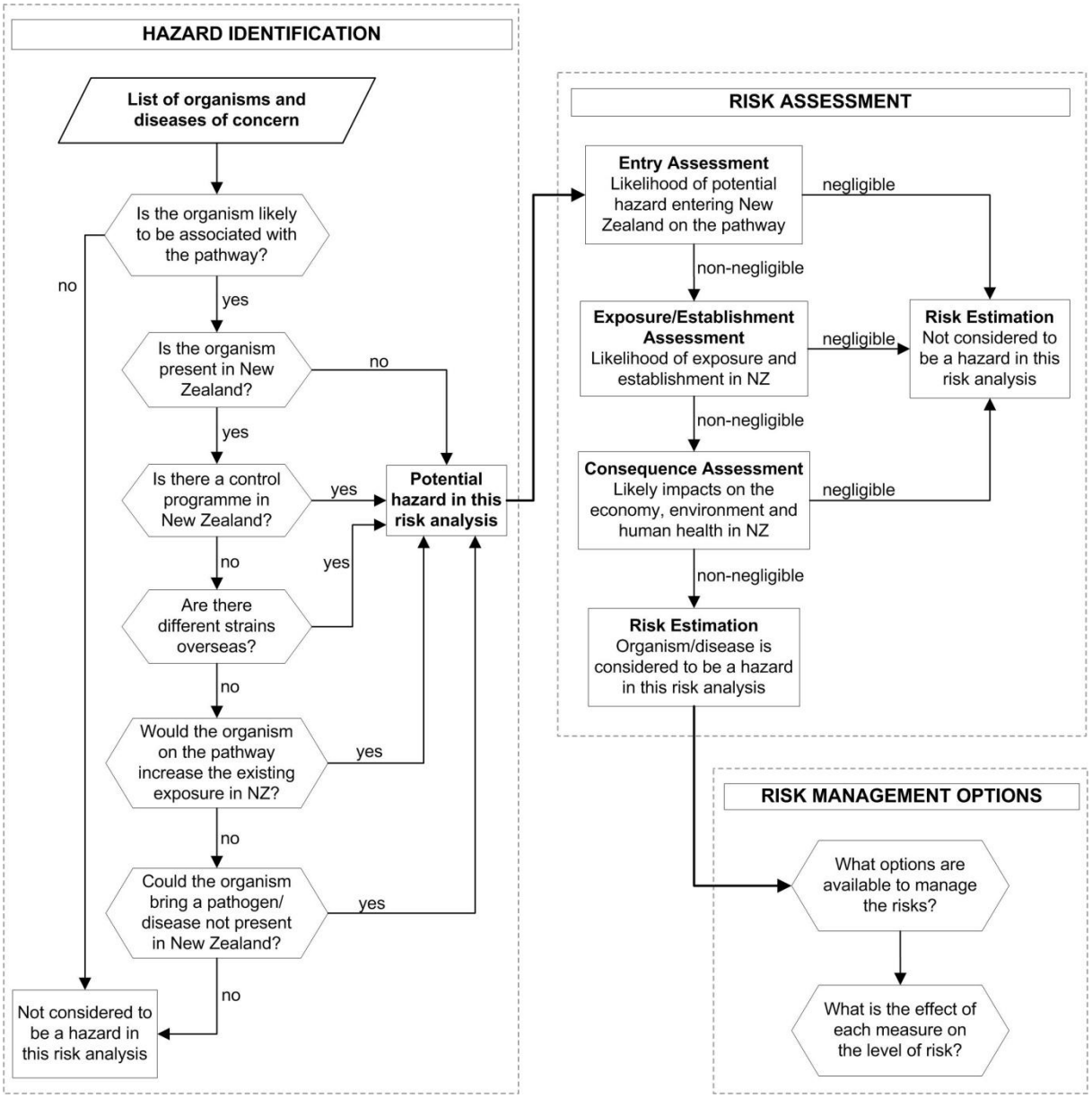
5. Risk analysis methodology

The methodology used in this risk analysis is described in MPI's *Risk Analysis Procedures Version 1* (MAF 2006) and is consistent with the guidelines in section 2 of the *Code* (OIE 2008a).

The risk analysis process is summarised in Figure 1.

[2] http://www.biosecurity.govt.nz/biosec/consult/draft-ihs-pigsemic.gen

Figure 1. The risk analysis process.



5.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms likely to be associated with the commodity. The basis for the preliminary hazard list is all the diseases/disease agents of pigs that were listed by the OIE in 2008 as well as other diseases mentioned in the following sources:

- MPI's IHSs and Overseas Market Access Requirements (OMARS) for pigs and pig products.
- Diseases of Swine *(2006). Edited by BE Straw, JJ Zimmerman, S D'Allaire, DJ Taylor, 9th edition. Blackwell Publishing, Ames, Iowa. ISBN-13: 978-0-8138-1703-3.*
- Virus Infections of Porcines *(1989). Edited by MB Pensaert, Elsevier Science Publishers, Amsterdam. ISBN 0-444-42909-3.*
- Infectious Diseases of Livestock *(2004). Edited by JAW Coetzer and RC Tustin, 2nd edition. Oxford University Press, Cape Town. ISBN 0-19-578202 X.*

Organisms listed in the above sources that were not considered to be of concern were:

Porcine retroviruses. The only retroviruses known in pigs are endogenous retroviruses which are integrated as part of the genome. They are present in the DNA of all pigs and do not cause disease. Therefore they are not regarded as pathogens and are of no relevance.

Porcine lymphotrophic herpesviruses occur widely in all countries and in all types of pigs. They have not been associated with naturally occurring diseases. They were unknown prior to 1999 and since pigs were widely traded before this date they could be presumed to be universally distributed. They are presently of some concern in relation to transplantation experiments since they may be associated with post-transplant lymphoproliferative disease. Since they are not associated with naturally occurring diseases and are universally distributed, they have been excluded from consideration in this risk analysis.

Additional diseases or disease agents were included as a result of suggestions by experts reviewing the draft risk analysis or by interested parties that were consulted on the subject.

Organisms/diseases identified in the above sources that were considered to be organisms of potential concern are listed in Table 1. The table indicates whether the organisms are zoonotic, and whether they are present in New Zealand and in the relevant exporting countries that were the focus of the first two drafts of this risk analysis. References are given to substantiate the entries in the table.

For agents that are known to be present in New Zealand, information about occurrence in other countries is generally irrelevant and references relating to the exporting countries have not always been given.

Table 1. Organisms of potential concern

Disease agent	OIE List	Zoonotic	Present in NZ	Present in Australia	Present in USA and Canada	Present in EU or Norway	Prelim Hazard
Viruses/diseases							
Adenovirus	No (OIE 2008a)	No	?	?	Yes (Kleiboeker 2006)	Yes (Kleiboeker 2006)	Yes
African swine fever virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008c)	Yes
Aujeszky's disease virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Blue eye disease virus	No (OIE 2008a)	No	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No
Bovine viral diarrhoea 1 virus	Yes (OIE 2008a)	No	Yes (Vilcek et al 1998; Horner 2000)	Yes (Mahony et al 2005)	Yes (Potgieter 2004)	Yes (Barkema et al 2001; Drew et al 2002; Vilcek et al 2003)	No
Bovine viral diarrhoea 2 virus	Yes (OIE 2008a)	No	No (Vilcek et al 1998; Horner 2000)	No (Mahony et al 2005)	Yes (Potgieter 2004)	Yes (Barkema et al 2001; Drew et al 2002; Vilcek et al 2003)	Yes
Classical swine fever virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Congenital tremors virus	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No
Cytomegalovirus	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No
Eastern equine encephalitis virus	Yes (OIE 2008a)	Yes	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes
Encephalomyocarditis virus	No (OIE 2008a)	No	Yes (Sutherland et al 1977)	Yes (Acland and Littlejohns 1975; Seaman et al 1986)	Yes (Koenen 2006)	Yes (Koenen 2006)	No
Foot and mouth disease virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No
Hepatitis E virus	No (OIE 2008a)	Yes	Yes (Garkavenko et al 2001)	Yes	Yes	Yes	No
Japanese encephalitis virus	Yes (OIE 2008a)	Yes	No (CDC 2006)	Yes (Hanna et al 1999)	No (CDC 2006)	No (CDC 2006)	Yes
Kunjin virus	No (OIE 2008a)	Yes	No	Yes (Broom et al 2003)	No	No	Yes
Menangle virus	No (OIE 2008a)	Yes (Kirkland and Stephano 2006)	No	Yes (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	Yes
Murray Valley encephalitis virus	No (OIE 2008a)	Yes	No	Yes (Mackenzie et al 1994)	No	No	Yes
Nipah virus	Yes (OIE 2008a)	Yes	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No (Kirkland and Stephano 2006)	No
Porcine circovirus and PMWS	No (OIE 2008a)	No	Yes (Rawdon et al	Yes (Raye et al 2005;	Yes (Segales et al 2006)	Yes (Segales et al	No

Disease agent	OIE List	Zoonotic	Present in NZ	Present in Australia	Present in USA and Canada	Present in EU or Norway	Prelim Hazard
			2004)	Muhling et al 2006)**		2006)	
Porcine epidemic diarrhoea virus	No (OIE 2008a)	No	No (MAF 2007)	No#	No (Pensaert and Yeo 2006)	Yes (Pensaert and Yeo 2006)	Yes
Porcine haemagglutinating encephalomyelitis virus	No (OIE 2008a)	No (Pensaert 2006)	No#	Yes (Forman et al 1979)	Yes (Pensaert 2006)	Yes (Pensaert 2006)	Yes
Porcine myocarditis virus	No (OIE 2008a)	unknown	No	Yes (Biddle 2007)	No	No	Yes
Porcine parvovirus	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No
Porcine reproductive and respiratory syndrome virus	Yes (OIE 2008a)	No	No (MAF 2007)	No (Animal Health Australia 2007)	Yes (Drew and Paton 2004)	Yes(Drew and Paton 2004)	Yes
Porcine respiratory coronavirus	No (OIE 2008a)	No	No (MAF 2007)	No#	Yes (Saif and Sestak 2006)	Yes (Van Reeth and Pensaert 2004)	Yes
Rabies virus	Yes (OIE 2008a)	Yes	No (OIE 2008b)	No (OIE 2008b)	Yes (OIE 2008b)	Yes (OIE 2008b)	Yes
Ross River virus	No (OIE 2008a)	Yes	No (McFadden et al 2009)	Yes (Hu et al 2007)	No	No	Yes
Rotavirus and Reovirus	No (OIE 2008a)	No (Yuan et al 2006)&	Yes (Fu and Hampson 1987; Fu et al 1989)	Yes (Nagesha and Holmes 1988; Nagesha et al 1988; Nagesha et al 1992)	Yes (Yuan et al 2006)	Yes (Yuan et al 2006)	Yes***
Rinderpest virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No
Swine influenza virus	No (OIE 2008a)	No (Thompson and Easterday 2004)&	Yes (Stanislawek 2001)	Yes (Thompson and Easterday 2004)	Yes (Thompson and Easterday 2004)	Yes (Thompson and Easterday 2004)	Yes***
Swine pox virus	No (OIE 2008a)	No (Delhon et al 2006)	No (MAF 2007)	Yes (Delhon et al 2006)	Yes (Delhon et al 2006)	Yes (Delhon et al 2006)	Yes
Swine vesicular disease virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Some (OIE 2008b; OIE 2008c)	Yes
Teschovirus serotype 1	No (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	No
Torovirus	No (OIE 2008a)	No (Jamieson et al 1998; Wilhelm et al 2003; Lodha et al 2005)	Yes (Horner 1994)	Yes	Yes	Yes	No
Torque teno virus	No (OIE 2008a)	No	?	?	Yes (Sibila et al 2009)	Yes (Sibila et al 2009)	Yes
Transmissible gastroenteritis virus	Yes (OIE 2008a)	No	No (OIE 2008b; OIE 2008c)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Venezuelan encephalitis virus	Yes (OIE 2008a)	Yes	No (OIE 2008c)	No (OIE 2008c)	No (OIE 2008c)	No (OIE 2008c)	No

Disease agent	OIE List	Zoonotic	Present in NZ	Present in Australia	Present in USA and Canada	Present in EU or Norway	Prelim Hazard
Vesicular stomatitis virus	Yes (OIE 2008a)	Yes (Mare and Mead 2004)	No (OIE 2008b)	No (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	No (OIE 2008b)	Yes
Vesicular exanthema virus	No (OIE 2008a)	No (House and House 1999)	No (House and House 1999)	No (House and House 1999)	No (House and House 1999)	No (House and House 1999)	No
West Nile virus	Yes (OIE 2008a)	Yes	No (OIE 2008c)	No (OIE 2008c)	Yes (OIE 2008c)	Yes (OIE 2008c)	Yes
Western equine encephalitis virus	Yes (OIE 2008a)	Yes	No (OIE 2008c)	No (OIE 2008c)	Yes (OIE 2008c)	No (OIE 2008c)	Yes
Bacterial agents/ diseases							
Actinobacillus pleuropneumoniae	No (OIE 2008a)	No	Yes (Hilbink et al 1992)	Yes (Gottschalk and Taylor 2006)	Yes (Gottschalk and Taylor 2006)	Yes (Gottschalk and Taylor 2006)	No
Actinobacillus suis	No (OIE 2008a)	No	Yes (Carman and Hodges 1982)	Yes (Wilson and McOrist 2000)	Yes (Taylor 2006b)	Yes (Taylor 2006b; Taylor 2006c)	No
Actinobaculum suis (Eubacterium suis)	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes (Taylor 2006c)	Yes (Taylor 2006c)	Yes (Taylor 2006c)	No
Arcanobacterium (Actinomyces or Corynebacterium) pyogenes	No (OIE 2008a)	No	Yes (Quinn et al 2002)	Yes	Yes	Yes	No
Bacillus anthracis	Yes (OIE 2008a)	Yes (Taylor 2006a)	No (Gill 1992)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Brucella suis	Yes (OIE 2008a)	Yes (Macmillan et al 2006)	No (MAF 2007)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Burkholderia pseudomallei	No (OIE 2008a)	Yes (Thomas 1981; Inglis 2004)	No (MAF 2007)	Yes (Thomas 1981; Inglis 2004)	No (Van der Lugt 2004)	No (Van der Lugt 2004)	Yes
Chlamydia trachomatis *Chlamydophila pecorum* *Chlamydophila psittaci*	No (OIE 2008a)	Yes (some species)	Yes	Yes	Yes	Yes	No
Clostridium tetani	No (OIE 2008a)	Yes (Odendaal and Kriek 2004)	Yes (Ellison 1992)	Yes	Yes	Yes	No
Clostridium botulinum	No (OIE 2008a)	Yes (Kriek and Odendaal 2004)	Some strains	Yes	Yes	Yes	Yes***
Other clostridial species	No (OIE 2008a)	Some species/ strains	Some species/ strains	Yes	Yes	Yes	Yes***
Erysipelothrix rhusiopathiae	No (OIE 2008a)	Yes (Wood and Henderson 2006)	Yes (Fairley 1997)	Yes	Yes	Yes	No
Escherichia coli	No (OIE 2008a)	Yes	Yes	Yes	Yes	Yes	No
Lawsonia intracellularis	No (OIE 2008a)	No (McOrist and Gebhart 2006)	Yes (Fairley 1997)	Yes	Yes	Yes	No
Leptospira serovars	Yes (OIE 2008a)	Yes (Ellis 2006)	Yes (Midwinter 1999)	Yes	Yes	Yes	Yes***

Disease agent	OIE List	Zoonotic	Present in NZ	Present in Australia	Present in USA and Canada	Present in EU or Norway	Prelim Hazard
Mycobacterium spp.	Yes (OIE 2008a)	Yes (Cousins et al 2004)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	No
Mycoplasma hyopneumoniae	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No
Mycoplasma hyorhinis	No (OIE 2008a)	No	Yes (MacPherson and Hodges 1985)	Yes	Yes	Yes	No
Mycoplasma hyosynoviae	No (OIE 2008a)	No	No#	Yes (Thacker 2006)	Yes (Thacker 2006)	Yes (Thacker 2006)	Yes
Other *Mycoplasma* spp.	No (OIE 2008a)	No	No#	?	?	?	Yes
Pasteurella multocida	Yes (OIE 2008a)	No (De Jong 2006)	No (OIE 2008b)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes (OIE 2008b; OIE 2008c)	Yes
Pasteurella spp.	No (OIE 2008a)	No	Yes##	Yes	Yes	Yes	No
Rhodococcus equi	No (OIE 2008a)	No	Yes (Carman and Hodges 1987)	Yes (Taylor 2006d)	Yes	Yes	No
Exotic *Salmonella* spp.	No (OIE 2008a)	Yes	No	Yes	Yes	Yes	Yes
Serpulina pilosicoli	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No
Brachyspira (Serpulina) hyodysenteriae	No (OIE 2008a)	No	Yes (Fairley 1997)	Yes	Yes	Yes	No

Table footnotes:

** Porcine circovirus 2 is present in Australia but PMWS has not been reported.
*** Serotype/strain variation occurs.
& Possibly zoonotic following reassortment with human viruses
# An extensive search of the literature revealed no evidence for the presence of the disease in the countries concerned.
Commonly isolated and included in quarterly reports in the MAF publication *Surveillance*.

The final version of this risk analysis also contains chapters assessing the risks associated with blue eye disease virus, foot and mouth disease virus, Nipah virus, Teschovirus serotype 1, Venezuelan encephalitis virus, and vesicular exanthema virus in porcine semen imported from countries other than Australia, the USA, Canada, the European Union or Norway. These agents were not assessed to be risks with semen from those countries. However, the development of an IHS for porcine semen from all countries requires an assessment of the risk posed by these additional pathogens.

Rinderpest virus is not subject to risk assessment as this disease was formally declared to be globally eradicated in June 2011[3].

5.2. HAZARD IDENTIFICATION

For each organism, the epidemiology is discussed, including a consideration of the following questions:

[3] See: http://www.fao.org/ag/againfo/programmes/documents/grep/A_RESO_18_FMD_Eradication.pdf

1. Could the imported commodity act as a vehicle for the introduction of the organism?
2. If the organism requires a vector, could competent vectors be present in New Zealand?
3. Is the organism exotic to New Zealand?
4. If it is present in New Zealand,
    i. is it "under official control", which could be by government departments, by national or regional pest management strategies or by a small-scale programme, or
    ii. are more virulent strains known to exist in other countries?

For any organism, if the answer to question one is “yes” (and the answer to question two is “yes” in the cases of organisms requiring a vector), and the answers to either questions three or four are “yes”, it is classified as a potential hazard requiring risk assessment.

Under this framework, organisms that are present in New Zealand cannot be considered as potential hazards unless there is evidence that strains with higher pathogenicity are likely to be present in the commodity to be imported. Therefore, although there may be potential for organisms to be present in the imported commodity, the risks to human or animal health are no different from risks resulting from the presence of the organism in this country already.

If importation of the commodity is considered likely to result in an increased exposure of people to a potentially zoonotic organism already present in New Zealand, then that organism is also considered to be a potential hazard.

5.3. RISK ASSESSMENT

In line with the MPI and the OIE risk analysis methodologies, the following analysis is carried out for each potential hazard requiring risk assessment:

Risk Assessment

a)	Entry assessment -	the likelihood of the organism being imported in the commodity.
b)	Exposure assessment -	the likelihood of animals or humans in New Zealand being exposed to the potential hazard.
c)	Consequence assessment -	the consequences of entry, 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 note that all of the above steps may not be necessary in all risk assessments. The MPI and the OIE risk analysis methodologies make it clear that if the likelihood of release is negligible for a 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 likelihood of exposure to susceptible species is negligible, or where both release and exposure are non-negligible but the consequences of introduction are concluded to be negligible.

5.4. RISK MANAGEMENT

For each organism classified as a hazard in the commodity, the risk management section identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a hazard, these are described alongside options of similar, lesser, or greater stringency where available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all hazards. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an import health standard (IHS) is drafted. As obliged under Article 3.1 of the WTO Agreement on Sanitary and Phytosanitary Measures (the SPS Agreement), the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

5.5. RISK COMMUNICATION

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

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6. Porcine adenovirus

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: *Adenoviridae*, Genus; *Mastadenovirus*, Species: *Porcine adenovirus* (Benko et al 2005). There are at least six serotypes of the virus (Kleiboeker 2006).

6.1.2. OIE list

Not listed.

6.1.3. New Zealand status

Porcine adenovirus is widely distributed in the world and could be considered likely to be present in New Zealand. However, since there is no evidence to confirm its presence here, for the purposes of this risk analysis porcine adenovirus is assumed to be exotic.

6.1.4. Epidemiology

Swine are the only species known to be susceptible to porcine adenoviruses (Kleiboeker 2006). While the role of the virus in causing diarrhoea is uncertain (Derbyshire et al 1966), infections of caesarean-derived, colostrum-deprived piglets have resulted in gastroenteric disease. The incubation period following faecal-oral transmission is 3-4 days, and virus is found in enterocytes for up to 45 days post infection (Kleiboeker 2006). Infection by the respiratory route may also be possible.

While antibodies are very commonly found in sows, they rarely excrete virus and there is a negative correlation between antibody levels and excretion of virus. Most virus is shed during the post-weaning period (Derbyshire et al 1966).

There is no indication that porcine adenovirus is excreted in semen and the available information suggests that it is an enteric infection with virus found in enterocytes. Most of the literature on the epidemiology of the virus is 20-30 years old, suggesting that this virus is no longer considered to be important.

6.1.5. Hazard identification conclusion

In view of the above, porcine adenovirus is not identified as a hazard in the commodity.

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7. African swine fever virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: *Asfviridae*, Genus; *Asfvirus*, Species: African swine fever virus (ASFV) (Sánchez-Vizcaíno 2006).

7.1.2. OIE list

Listed (OIE 2009a).

7.1.3. New Zealand status

Exotic notifiable organism (MAF 2009).

7.1.4. Epidemiology

The United States, Canada, Australia, and Norway are free from African swine fever (ASF). With the exception of Sardinia (Italy), all EU member states are free of ASF (OIE 2009b). The European Commission decision 2005/362/EC has approved an eradication plan for ASF in Sardinia. This complies with the Directive 2002/60/EC which (amongst other measures) prohibits the movement of porcine semen from any holding within 10km of a premise infected with ASF. The Agreement between the European Community and New Zealand on sanitary measures applicable to trade in live animals and animal products (97/132/EC) prevents the importation of any animal products to New Zealand that are unable to move freely in the EU.

ASF is not recognised in any of the official EU candidate countries (Croatia, the Former Yugoslav Republic of Macedonia, and Turkey) or in any of the recognised potential candidate countries (Albania, Bosnia and Herzegovina, Montenegro, Serbia, and Iceland) (OIE 2009b). However, ASF is now spreading through Russia, the Ukraine, and neighbouring countries in the Caucasus (Callaway 2012) and is recognised as a potential source of possible incursions into the EU (Anonymous 2009). ASF is considered to be endemic in most countries in sub-Saharan Africa (FAO 2012).

When initially described, ASF infection of domestic pigs was associated with morbidity rates up to 100% and mortality rates often over 90%. However, where the disease has become enzootic, a decrease in virulence may be seen resulting in fatality rates as low as 2-3% (Mebus 1988).

In African countries, ASF is maintained in a subclinically infected wild pig population and transmitted to domestic pigs via the argasid tick *Ornithodoros moubata*, although other tick vectors (*O. savignyi* and *O. porcinus porcinus*) have also been described (Kleiboeker et al 1998; Mebus 1988; Kleiboeker et al 1999; Burrage et al 2004). In Europe, the soft tick *O. erraticus* is recognised as the vector of ASFV (Wilkinson 1984) and it is thought that most *Ornithodoros* spp. of ticks that will feed on pigs are capable of acting as vectors for ASFV (Radostits et al 2007).

Once infection is established in domestic pigs, it can spread rapidly between pigs by direct or indirect contact. ASFV is present in a high titre in nasopharyngeal secretions at the onset of clinical signs, and can be recovered from all organs of acutely sick pigs (Radostits et al 2007).

Viraemia develops within 48-72 hours of infection, and infectivity then persists for at least 7 days. The effect of the virus on haemostasis and on the vascular endothelium usually results in

death due to oedema and haemorrhage (Rodríguez et al 1996; Takamatsu et al 1999; Vallée et al 2001).

Some authors have suggested that ASFV can be found in boar semen and even transmitted to recipient sows (Thacker et al 1984; Wittmann 1989; Guérin and Pozzi 2005), although the only evidence for this provided in any of these sources appears to be a personal communication by DH Schlafer in 1984. More recently, Maes et al 2008 stated that there is no published evidence to support this hypothesis.

7.1.5. Hazard identification conclusion

Of the countries initially considered in this risk analysis, ASF is only found in Sardinia and legislative controls described above prohibit the movement of semen from any premises within 10Km of a property infected with this disease. ASF is endemic in sub-Saharan Africa and is an emerging disease throughout Russia, the Ukraine, and neighbouring countries in the Caucasus.

However, as there is no published evidence to show that ASFV can be found in the semen of infected boars, ASFV is not identified as a hazard in the commodity.

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8. Aujeszky's disease virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family: *Herpesviridae*, Subfamily: *Alphaherpesvirinae*, Genus: *Varicellovirus*, Species: *Suid herpesvirus* 1, Aujeszky's disease virus (Mocsari et al 1989; Davison et al 2005).

8.1.2. OIE list

Listed (OIE 2008a).

8.1.3. New Zealand status

Exotic, notifiable organism (MAF 2009).

8.1.4. Epidemiology

Aujeszky's disease (AD) virus can infect most mammals except primates, and is fatal in all animals except pigs. Therefore the pig is the only reservoir host, and all other animals are considered to be dead-end hosts (Pejsak and Truszczynski 2006).

Canada, Norway, and Australia are free from AD. In the EU only Italy, Poland, Spain, Northern Ireland, and Portugal are reported to be infected or possibly infected. The virus is present in the USA (OIE 2008b). Elsewhere, Aujeszky's disease is found in parts of Southeast Asia, and Central and South America including Mexico. The virus has also been reported from Cuba, Samoa, and Rwanda (Center for Food Security and Public Health 2006).

The disease has an incubation period ranging from 1-11 days (Pejsak and Truszczynski 2006). Respiratory and nervous signs may occur, but many pigs are subclinically infected. Mortality may be high in young pigs. The virus is found in nasal secretions and is transmitted by direct nose-to-nose contact or over distances of a few meters by aerosols (Van Oirschot 2004). It is also found in vaginal secretions and semen, and therefore may be transmitted by natural breeding and by artificial insemination (Pejsak and Truszczynski 2006; Van Oirschot 2004; Wittmann 1986).

Pigs become long-term latent carriers after recovery from infection, and reactivation and excretion of the virus can occur due to stress or as a result of treatment with corticosteroids. However, reactivation is rare and it is considered insignificant in the epidemiology of the disease (Van Oirschot 2004). The virus can be eradicated from infected herds by conventional test and slaughter procedures. In the case of the New Zealand eradication programme, vaccination with deletion marker vaccines was used as an additional aid (Motha et al 1997).

Infection can be diagnosed by isolation of the virus, identification of viral DNA by PCR, or by serological methods, particularly the ELISA (Pejsak and Truszczynski 2006; Toma et al 2008; Van Oirschot 2004). Since tissue culture is not an option for diagnosis of the virus in semen due to toxic effects of semen on cell lines, PCR is preferred (van Rijn et al 2004).

8.1.5. Hazard identification conclusion

Aujeszky's disease virus (ADV) is an exotic, notifiable organism that is readily transmitted in pig semen. Therefore it is identified as a potential hazard in the commodity.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

Pigs may be sub-clinically infected, long-term latent carriers of ADV. Therefore the likelihood of entry of the organism in the commodity is non-negligible for semen imported from any country where the virus is present.

8.2.2. Exposure assessment

Imported semen will be used to inseminate sows and the disease can be transmitted by infected semen. Therefore the likelihood of exposure of naïve New Zealand sows to virus in imported semen is non-negligible.

8.2.3. Consequence assessment

When previously introduced into New Zealand the virus spread through much of the North Island. Therefore, if reintroduced and left uncontrolled, it would likely become endemic over large areas. Production losses would be likely, particularly through mortality of young pigs. Introduction of the virus might interfere with New Zealand's limited pig exports. The consequences for domestic pigs are assessed to be non-negligible.

AD is not zoonotic and if introduced there would be no consequences for human health. ADV can infect a wide range of non-primate animals, and since infections of animals other than pigs are almost always fatal, they can be considered dead-end hosts. Moreover, infection of animals other than pigs is rare, occurring only where there is close contact with infected pigs (Pejsak and Truszczynski 2006). Spread from inseminated pigs to other animals is therefore unlikely, but could occur under certain circumstances. Spread to other farmed pigs would be possible if inseminated pigs were moved between herds. Spread to wild pigs is theoretically possible if there is contact with farmed pigs, and virus might circulate in wild pig populations if they are of sufficient density. However, feral pigs seldom have contact with domestic pigs and when AD was endemic in the North Island, feral pigs remained free from infection. In the unlikely event that animals other than pigs did become infected, there would be no further spread as they are considered to be dead-end hosts. The consequences for the environment are therefore assessed to be negligible.

8.2.4. Risk estimation

Entry, exposure and consequence are all assessed to be non-negligible. As a result the risk estimate for ADV is non-negligible and it is assessed to be a risk in the commodity. Therefore risk management measures can be justified.

8.3. RISK MANAGEMENT

8.3.1. Options

The *Code* chapter on AD (OIE 2008a) defines AD free countries, zones and establishments and provisionally free countries or zones.

The *Code* chapter on AD contains the following recommendations relating to importation of pig semen:

Article 8.2.12. Recommendations for importation from AD free countries or zones

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

1. the donor animals:
    a. showed no clinical sign of AD on the day of collection of the semen;
    b. were kept in an establishment or artificial insemination centre located in an AD free country or zone at the time of semen collection;
2. the semen was collected, processed and stored in conformity with the provisions of Chapters 4.5 and 4.6.

Article 8.2.13. Recommendations for importation from AD provisionally free countries or zones

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

1. the donor animals:
    a. have been kept for at least 4 months prior to semen collection in an artificial insemination centre which has the status of AD free establishment, and where all boars are subjected to a serological test to the whole AD virus, with negative results, every 4 months;
    b. showed no clinical sign of AD on the day of collection;
2. the semen was collected, processed and stored in conformity with the provisions of Chapters 4.5 and 4.6.

Article 8.2.14. Recommendations for importation from AD infected countries or zones

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

1. the donor animals:
    a. were kept in an AD free establishment for at least 6 months prior to entering the artificial insemination centre;
    b. have been kept for at least 4 months prior to semen collection in the artificial insemination centre which has the status of AD free establishment, and where all boars are subjected to a serological test to the whole AD virus, with negative results, every 4 months;
    c. were subjected to a serological test to the whole AD virus, with negative results, within 10 days prior to or 21 days after semen collection;
    d. showed no clinical sign of AD on the day of collection;
2. the semen was collected, processed and stored in conformity with the provisions of Chapters 4.5 and 4.6.

Chapters 4.5 and 4.6 of the *Code* (OIE 2009a) contain requirements for AD for boars standing at semen collection centres. Under these requirements, boars from a country or zone that is not free of AD must be clinically healthy, physiologically normal, not vaccinated against AD, kept exclusively in an AD-free establishment since birth, and subject to serological testing for whole AD virus with negative results within 30 days prior to entry into isolation at the quarantine station of the semen collection facility. Boars must remain in this quarantine station for at least 28 days and, at least 21 days after entering the quarantine station, must be tested negative for AD

as per Articles 8.2.12, 8.2.13, or 8.2.14 of the *Code*. In countries, zones or compartments which are not free of AD, any boars resident in the semen collection facility must also be tested at least annually as per Articles 8.2.12, 8.2.13 or 8.2.14 of the *Code*.

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

Option 1

Aliquots of each batch of semen to be imported could be tested by PCR for the presence of virus (van Rijn et al 2004). Importation of semen giving positive results could be prohibited.

Option 2

Semen originating from collection centres certified as compliant with OIE Code Chapters 4.5 and 4.6 could be considered suitable for importation.

Option 3

Semen for export to New Zealand could come from donor boars that were kept in an establishment or artificial insemination centre located in an AD-free country or zone at the time of semen collection.

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9. Blue eye disease virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family: *Paramyxoviridae*, Genus: *Rubulavirus*. Porcine rubulavirus (PoRV), also known as La-Piedad-Michoacan-Mexico virus (LPMV) (King et al 2012).

9.1.2. OIE list

Not listed.

9.1.3. New Zealand status

There are no reports of porcine rubulavirus or related paramyxoviruses of pigs in New Zealand.

9.1.4. Epidemiology

Blue eye pig disease was described in 1980 in a commercial herd in La Piedad, Central Mexico (Stephao et al 1981; 1982, cited by Stephan et al 1988). Although this disease has only been reported in Mexico (Center for Food Security and Public Health 2006), closely related paramyxoviruses of pigs have been found in Australia (Menangle virus, see Chapter 16), Canada (Salles et al 2011), Japan (Sasahara et al 1954), Israel (Lipkind et al 1986), and the United States (Janke et al 2001).

Infection of young pigs results in blue eye disease which is characterised by encephalitis, pneumonia, and corneal opacity. The disease presents clinically with sudden onset pyrexia followed by progressive neurologic signs such as weakness, ataxia, rigidity, or hyperexcitability (Center for Food Security and Public Health 2006). Morbidity in affected litters is usually between 20 and 50% and mortality around 90%. Pigs older than 30 days show moderate and transient clinical signs with fewer than 2% of this age group affected with low mortality. Reproductive failure is described in pregnant sows (Kirkland and Stephano 2006).

Pigs are the only species clinically affected by PoRV with virus most commonly transmitted by nose-to-nose contact between infected and susceptible pigs (Kirkland and Stephano 2006). Experimental infection (intranasal and intramuscular inoculation) of boars results in swelling of the epididymis, reduced spermatozoan motility and concentration with viral antigen detectable in the head of the epididymis. Viral antigen remains detectable in the epididymis for up to 10 weeks and detection of viral antigen in the lumina of the epididymal ductuli suggests the possibility of venereal transmission (Ramirez-Mendoza et al 1997). A subsequent study demonstrated that PoRV could be detected in the semen of infected pigs between 2 and 49 days after infection, leading the authors to propose that semen from PoRV-infected boars could be an important route of virus transmission among and within farms (Solís et al 2007).

9.1.5. Hazard identification conclusion

Although transmission in semen has not been proven experimentally, PoRV can be recovered from the semen of infected boars. Two recent reviews of diseases in swine likely to be transmitted by artificial insemination have both included PoRV (Guérin and Pozzi 2005; Maes et al 2008). PoRV is identified as a potential hazard in porcine semen.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Unilateral or bilateral swelling in the testicular and epididymal regions of infected boars is described from 15 days after infection which may subside within the following 10 days although subsequent testicular atrophy has been recorded (Ramirez-Mendoza et al 1997). However, overt clinical signs are not seen in all infected individuals so physical examination cannot be relied upon to detect boars infected with PoRV and virus can be recovered from the semen of infected boars up to 49 days post infection (Solís et al 2007). The likelihood of entry is assessed to be high.

9.2.2. Exposure assessment

Mumps virus is another member of the Rubulavirus genus and, although epididymo-orchitis is associated with human infections with the mumps virus and virus has been recovered from the semen of infected individuals (Jalal et al 2004), no reports of venereal transmission of mumps virus could be found.

Hernández-Jáuregui et al (2004) intranasally inoculated pregnant gilts at six weeks gestation with PoRV and were able to recover virus from the ovary, placenta, and uterus between two and four weeks later. Although transmission via infected semen has not been demonstrated, this study demonstrates that PoRV has a tropism for the tissues of the reproductive tract so the likelihood of exposure is assessed to be non-negligible.

9.2.3. Consequence assessment

Pigs are the only species clinically affected by PoRV. Infection may cause progressive neurological disease in younger pigs with high mortality although less marked signs are described in older animals. The consequences of infection are assessed to be non-negligible.

9.2.4. Risk estimation

Since the entry, exposure, and consequence assessments are all non-negligible, the risk is estimated to be non-negligible and PoRV is assessed to be a risk in porcine semen. Risk management measures are justified.

9.3. RISK MANAGEMENT

9.3.1. Options

PoRV has only been described in Mexico so sanitary measures are not required for imports of porcine semen originating from pigs in any country other than Mexico.

A number of serological tests have been described to detect exposure to PoRV including haemagglutination inhibition (Escobar-López et al 2012), virus neutralisation (Sánchez-Betancourt et al 2012), indirect immunofluorescence, and enzyme-linked immunosorbent assays (Nordengrahn et al 1999). All of these serological tests detect seroconversion by the 8th day after infection (Center for Food Security and Public Health 2006).

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

Option 1

Semen for export to New Zealand could come from donor boars that were kept in an establishment or artificial insemination centre located in a PoRV-free country or zone at the time of semen collection.

Option 2

Donor boars could be subject to serological testing with negative results.

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10. Classical swine fever virus and other pestiviruses

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Flaviviridae*, Genus: *Pestivirus*, Species: *Classical swine fever virus* (CSFV), *Bovine viral diarrhoea virus* 1 and 2 (BVDV-1 and 2), and *Border disease virus* (BDV) (Barkema et al 2001; Choi and Chae 2003; Thiel et al 2005). See also Chapter 21 (porcine myocarditis virus).

10.1.2. OIE list

Classical swine fever and bovine viral diarrhoea are listed diseases (OIE 2008a).

10.1.3. New Zealand status

BDV and BVDV-1 are endemic in New Zealand, while BVDV-2 and CSFV are both exotic (Horner 2000; Vilcek et al 1998; OIE 2008b).

10.1.4. Epidemiology

CSFV is the causative agent of classical swine fever (CSF), a highly contagious disease of pigs. CSFV is exotic to Australia, Norway, the USA, and Canada. Since 2007, outbreaks of CSF have been reported in the EU in Bulgaria, France, Germany, Hungary, Romania, and Slovakia (OIE 2008b), possibly as a result of infected wild boars transmitting the virus to farmed pigs. CSFV is found in much of Asia, some Caribbean islands, the African countries of Madagascar and Mauritius, and much of South and Central America (Center for Food Security and Public Health 2009).

BVDV causes bovine viral diarrhoea in cattle, and BDV causes border disease in sheep. Both BVDV-1 and BDV are endemic in New Zealand and, with the exception of Norway (OIE 2008b), in all the countries considered in this risk analysis. Some strains of BVDV-2 cause a more severe disease in cattle than BVDV-1. BVDV-2 is present in the EU (Barkema et al 2001; Cranwell et al 2005; Drew et al 2002; Nettleton and Gunn 2002; Vilcek et al 2002) and North America (Nettleton and Willoughby 2008; Potgieter 2004), but not in Norway or Australia (Horner 2000; Vilcek et al 1998).

Infection of pregnant sows with CSFV strains of low virulence can result in the birth of immunotolerant, persistently infected pigs, particularly when infection occurs in late gestation (Van Oirschot 2004). In pigs infected after birth the incubation period is 7-10 days. They are usually infective between days 5 and 14 post-infection, but can remain infective up to 3 months in cases of chronic infections (OIE 2008a). Pigs that recover from mild infections can carry the virus for long periods of time (Van Oirschot 2004). Some pigs persistently infected with CSFV develop late-onset disease with typical signs of acute or sub-acute infection after 4-6 months (Van Oirschot 2004). Infected pigs shed the virus in saliva, nasal and ocular secretions, faeces and urine. Boars may shed the virus in semen (Choi and Chae 2003; De Smit et al 1999; Floegel et al 2000) and sows can be infected by artificial insemination (Floegel et al 2000).

CSFV can be detected in semen by virus isolation in tissue culture, but reverse transcription PCR is a more sensitive method of detection which allows the detection of viral RNA for months after infection. For diagnosis of the disease a number of virus detection and serological tests are available. Serological testing is not suitable for detecting immunotolerant animals and cross reactions may occur with antibodies generated in response to BVDV infections. The virus can

also be demonstrated in tissues by immunological staining methods (Drew 2008b). Gross and histological lesions may be typical in cases of acute swine fever and are valuable diagnostic aids. Sensitive PCR methods are available that use group specific probes to detect all pestiviruses (BVDV-1, BVDV-2, BDV and CSFV) (Vilcek et al 1994) and PCR methods are available for genotyping pestiviruses (Sandvik et al 1997).

BVDV and BDV can infect pigs (Nettleton 2004), but the infection is usually subclinical. However, when these viruses infect naïve pregnant sows, infection of foetuses occurs and results in foetal mortality or birth of persistently-infected immunotolerant piglets. Piglets that have been infected *in utero* excrete large quantities of virus, but when they are infected at birth they excrete little or no virus (Le Potier et al 2006). It is assumed that persistently-infected pigs can excrete BVDV in their semen since this occurs with CSFV in pigs and in cattle that are persistently infected with BVDV (Kirkland et al 1991; Potgieter 2004).

Concerns have been raised by domestic stakeholders regarding the emergence of porcine neurologic and reproductive syndrome virus (novel Pesti-like virus in the US) and the occurrence of untypable or cross-reactive Pestivirus infections of swine in North America and elsewhere in the world (Clement 2009). However, at a subsequent meeting with domestic stakeholders to discuss these concerns (8th December 2009), no further information regarding this specific matter was provided. A literature search has identified one publication regarding the isolation of a novel viral agent (termed "Virus X" by the authors) associated with porcine reproductive and neurological syndrome and reproduction of the disease (Pogranichniy et al 2008). This publication concluded that the possibility that Virus X is a pestivirus cannot be completely ruled out. However, there has been no further published confirmation of the identity of this agent or any studies which indicate that it is likely to be transmitted in semen.

10.1.5. Hazard identification conclusion

Therefore CSFV is identified as a potential hazard. BVDV-1 and BDV are endemic in New Zealand and therefore are not potential hazards. BVDV-2 could infect pigs and be excreted in their semen and it is therefore also identified as a potential hazard.

There is insufficient evidence to conclude that other (novel) pestiviruses should be identified as a hazard in porcine semen.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Blood, secretions and excretions (including oronasal and lacrimal secretions, urine, faeces, and semen) and tissues from infected pigs contain CSFV. Virus shedding can begin before the onset of clinical signs, and occurs throughout the course of acute or subclinical disease. Chronically or persistently infected pigs can shed virus continuously or intermittently for months (Center for Food Security and Public Health 2009). The likelihood of CSFV entry in the semen of pigs from infected countries is assessed to be high.

In Germany and France the occurrence of CSF is associated with infection of wild boars that transmit the virus to farmed pigs. In most areas of Germany and France the likelihood of infection of pig semen is low. However, if donor pigs originate from areas where contact with infected wild pigs is possible, the likelihood of infection is assessed to be non-negligible.

Pigs originating from countries where BVDV-2 is found could be subclinically infected with BVDV-2 and could excrete the virus in their semen. The likelihood for entry of BVDV-2 virus in pig semen from these countries is therefore non-negligible.

10.2.2. Exposure assessment

Imported semen will be inseminated into New Zealand pigs. As it has been demonstrated that sows can be infected with CSFV by artificial insemination with infected semen (Floegel et al 2000), it is assumed that this is also likely for pigs that are infected with BVDV-2, the likelihood of exposure of New Zealand pigs is assessed to be non-negligible.

10.2.3. Consequence assessment

Insemination of pigs would result in the transmission of the pestiviruses concerned to the recipients of the semen. Contact between infected pigs and other New Zealand pigs or ruminants could result in spread of imported pestiviruses. CSFV could cause disease that could spread widely in pigs due to movement of animals and contact transmission. BVDV could be transmitted from pigs to cattle and establishment of virulent strains of BVDV-2 could result in outbreaks of a disease that would have production-limiting and economic implications for the New Zealand cattle industries.

CSF is an OIE listed disease. Introduction of virulent strains of CSFV could cause catastrophic mortality in a naïve pig population and bans on trade in New Zealand pig meat. Low virulence strains would cause less dramatic economic losses but would lead to difficulties in diagnosis and ongoing economic losses to pig farmers. Introduction of CSF would result in the need for an expensive eradication campaign involving stamping out of the disease or vaccination of the pig population.

BVDV and CSFV are not zoonotic and there would be no consequence for human health. BVD is not described as a disease of any significance in wild ruminants and therefore would be of no consequence to feral ruminants. CSF could infect wild pigs that could then act as a reservoir of infection for domestic pigs. However, under New Zealand conditions the likelihood of contact between wild pigs and domestic pigs in commercial units that might be users of imported semen is assessed to be low, but non-negligible.

Since BVDV-2 could be transmitted from infected pigs to cattle with subsequent spread of the virus between cattle the consequences are assessed to be non-negligible. Introduction of CSFV could lead to high mortalities in pigs and limited trade bans.

10.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for CSFV and BVDV-2 is non-negligible and they are classified as a risk in the commodity. Therefore risk management measures can be justified.

10.3. RISK MANAGEMENT

10.3.1. Options

Within the EU, CSF occurs only in Germany and France. EU directive 2008/855/EC prohibits movement of semen from areas infected with CSF and the Agreement between the European Community and New Zealand on sanitary measures applicable to trade in live animals and

animal products (97/132/EC) prevents the importation of any animal products to New Zealand that are unable to move freely in the EU.

The *Code* defines CSF-free countries, zones and compartments and also countries free from CSF in domestic pigs but with a wild pig population. The *Code* chapter on CSF contains the following recommendations relating to importation of pig semen:

> **Article 15.3.8.** Recommendations for importation from countries, zones or compartments free of CSF
>
> Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:
>
> 1. the donor animals:
>   a. were kept in a country, zone or compartment free of CSF since birth or for at least 3 months prior to collection;
>   b. showed no clinical sign of CSF on the day of collection of the semen;
> 2. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.
>
> **Article 15.3.9.** Recommendations for importation from CSF infected countries or zones
>
> Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:
>
> 1. the donor animals:
>   a. were kept in a compartment free of CSF since birth or for at least 3 months prior to collection;
>   b. showed no clinical sign of CSF on the day of collection of the semen and for the following 40 days;
>   c. met one of the following conditions:
>   i. have not been vaccinated against CSF and were subjected to a serological test performed at least 21 days after collection, with negative results; or
>   ii. have been vaccinated against CSF and were subjected to a serological test in accordance with the Terrestrial Manual performed at least 21 days after collection and it has been conclusively demonstrated that any antibody is due to the vaccine; or
>   iii. have been vaccinated against CSF and were subjected to a virological test performed in accordance with the Terrestrial Manual on a sample taken on the day of collection and it has been conclusively demonstrated that the boar is negative for virus genome;
> 2. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.

Chapters 4.5 and 4.6 of the *Code* (OIE 2009a) contain requirements for CSF for boars standing at semen collection centres. Under these requirements, boars must be clinically healthy, physiologically normal, kept in a CSF-free zone or compartment since birth or for the past three months prior to entry into isolation at the quarantine station of the semen collection facility, and neither be vaccinated against CSF nor the progeny of vaccinated sows (unless there are OIE validated means of distinguishing vaccinated and infected pigs). Boars must remain in this

quarantine station for at least 28 days and, at least 21 days after entering the quarantine station must be tested negative for CSF as per Articles 15.3.8 of 15.3.9 of the *Code*. In countries, zones or compartments which are not free of CSF, any boars resident in the semen collection facility must also be tested at least annually as per Articles 15.3.8 or 15.3.9 of the *Code*.

Available options, for exclusion of both CSFV and BVDV-2 are:

Option 1

Semen originating from collection centres certified as compliant with OIE Code Chapters 4.5 and 4.6 could be considered suitable for importation.

Option 2

Every batch of semen to be imported could be tested by a group-specific reverse transcriptase PCR test that detects all relevant pestiviruses (Drew 2008a; Nettleton and Willoughby 2008; Vilcek et al 1994). A positive test on any batch of semen could result in disqualification of that semen.

Option 3

Donor boars could have lived their entire lives in countries that are free from both CSFV and BVDV-2.

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11. Eastern equine encephalomyelitis virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Family: *Togaviridae*, Genus: *Alphavirus*, Species: *Eastern equine encephalomyelitis virus* (EEEV) (Weaver et al 2005).

11.1.2. OIE list

Listed (OIE 2008).

11.1.3. New Zealand status

Notifiable disease (MAF 2008).

11.1.4. Epidemiology

EEEV is endemic in the eastern USA. It is transmitted between birds by the mosquito *Culiseta (Climacura) melanura*, which frequents wetlands in the southern and southeastern USA, particularly those with peat-muck soil dominated with hardwood trees. These wet and murky habitats and hardwood root systems favour oviposition by *C. (C.) melanura*, as its larvae require water with a high content of organic matter and protection from sunlight for development. Female *C. (C.) melanura* feed almost exclusively on birds, especially passerines, which maintain the cycle of infection by the development of high viraemias. Because *C. (C.) melanura* is highly ornithophilic, other vectors are probably responsible for affecting the escape of the virus from the bird/mosquito cycle. *Coquillettidia perturbans* is suspected of fulfilling this role, as it feeds on mammals and birds equally. Cases of encephalitis caused by EEEV tend to occur within 8 km of swampy habitats, which is within the flight range of *C. perturbans* (Gibbs 2004).

Although EEEV has been isolated from over 20 different mosquito species in six genera, its establishment has been shown to require specific ecological conditions such as those found in hardwood wetlands of southeastern USA, particularly the presence of the highly ornithophilic mosquito *C. (C.) melanura*, and the specific passerine birds on which it feeds. This mosquito vector is not present in New Zealand (Belkin 1968) and neither are the specific passerines nor the forest ecosystems that it inhabits.

EEEV is mainly responsible for disease outbreaks in humans and horses, but has also been involved in outbreaks of disease in pigs (Elvinger et al 1994; Elvinger et al 1996a; Elvinger et al 1996b). In general, mammals are considered dead-end hosts because the very low virus titres that develop are insufficient to infect vectors. However, nursing pigs that were infected experimentally developed a high-titre viraemia that lasted up to 168 hours after infection, and virus could be isolated from oropharyngeal and rectal swabs up to 96 hours after infection (Elvinger and Baldwin 2006). Experimental infection has been achieved by intracranial, intradermal, intravenous, and oral routes of virus administration (Elvinger and Baldwin 2006). However, there are no reports suggesting virus can be transmitted in porcine semen.

11.1.5. Hazard identification conclusion

There is no evidence to suggest that transmission of EEV by semen should be considered likely, so the virus is not identified as a hazard in pig semen.

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12. Porcine haemagglutinating encephalomyelitis virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*, Species: *Porcine haemagglutinating encephalomyelitis virus* (Spaan et al 2005).

12.1.2. OIE list

Not listed.

12.1.3. New Zealand status

It is classified as an "other exotic virus" (MAF 2008).

12.1.4. Epidemiology

The virus occurs commonly in pigs and is probably distributed world-wide. It has not been described in New Zealand, but this may reflect a lack of investigation rather than true absence of the virus.

The disease syndrome caused by this virus has been well described in several reviews (Andries 1989; Pensaert 2004; Pensaert 2006). Outbreaks of disease are rare and are usually confined to neonatal pigs which are susceptible to the virus unless protected by colostral antibody. Older pigs are resistant and infections are subclinical, but they develop antibody to the virus. If the virus is introduced into an immunologically naïve pig herd, young pigs develop signs which may include vomiting and wasting followed by severe encephalomyelitis and high mortality. Such outbreaks are generally self-limiting, lasting only a few weeks until sows have developed immunity. The incubation period under experimental conditions was 4 days. In natural outbreaks piglets as young as 2 days old may develop signs of infection, but the incubation period is usually considered to be 4-7 days. Pigs older than 4-5 weeks develop an age-related resistance to the disease and 43-98 per cent of pigs are serologically positive in infected regions. Pigs excrete the virus in oronasal secretions for 8-10 days and transmission is from exposure to nasal secretions. Long-term carriers are not described. Spread to the central nervous system is along nerve tracts and viraemia is believed to be of little importance in the pathogenesis of the disease. There is no evidence that the virus is excreted in semen.

12.1.5. Hazard identification conclusion

The disease is predominantly one of young pigs; animals older than 4-5 weeks are resistant to the disease. Adult semen donors are therefore unlikely to be infected with the virus and there is no evidence that the virus is transmitted in semen. Therefore the virus is not identified as a hazard in the commodity.

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13. Foot and mouth disease virus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Family: Picornaviridae; Genus: Apthovirus, foot and mouth disease virus (FMDV). There are seven serotypes of the virus: O, A, C, SAT 1, SAT 2, SAT 3 and Asia 1 (OIE 2009).

13.1.2. OIE list

Foot and mouth disease (FMD) is listed within the category of multiple species diseases.

13.1.3. New Zealand status

FMD is an exotic notifiable disease that has never occurred here.

13.1.4. Epidemiology

The disease has been eradicated from or has not occurred in North America, Australia and many European countries. FMD is a highly contagious viral disease that causes high fever, vesicular lesions and ulcerations. FMD is considered the most contagious and economically devastating animal disease. 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.

The disease is widespread occurring endemically in areas of South America, Africa and Asia. Currently, there are many unresolved disease events, including outbreaks in Europe (Bulgaria), China, North and South Korea, and South Africa (WAHID 2011).

Host species include cattle, zebus, domestic buffaloes, yaks, sheep, goats, swine, all wild ruminants, wild *Suidae* and members of the *Camelidae* family. Although all cloven hoofed animals are susceptible, expression of disease is variable from severe clinical signs to inapparent infections (OIE 2009). Sheep may show no clinical signs whilst infectious, and pigs are an important amplifying host.

The incubation period ranges from 2-14 days. However, for the purposes of the *Code* the incubation period is considered to be 14 days. Morbidity in domestic species is near 100% but is variable in wildlife. About 15-50% of cattle become carriers following infection. The virus may persist in the pharyngeal region for up to 3.5 years. The virus type influences the duration of the carrier state. However, carriers are not epidemiologically important since evidence suggests that they do not act as a source of infection (USAHA 2008). In pigs, a carrier state does not occur (Farez and Morley 1997).

The levels of virus present in animals peak at around the time of onset of clinical signs, but significant levels of virus may be present before this time. FMDV infected animals may excrete virus 4 days prior to clinical signs appearing (Geering et al 1995).

Seven immunologically distinct types of FMDV have been identified. For each virus type, immunologically related subtypes also exist, creating 60 known type-subtype combinations. During an infection, virus recombinations, mutations, and host selection result in the constant generation of new FMD variants, creating challenges in vaccine strain selection (USAHA 2008).

Vaccination reduces virus shedding and prevents clinical signs but does not necessarily prevent infection. Potent and highly purified vaccines protect animals from disease within 4-6 days post vaccination. Vaccinated or un-vaccinated animals that are infected with FMDV produce antibodies to both non-structural and structural proteins. Vaccinated animals that are not infected with FMDV only produce antibodies to the structural proteins (OIE 2009). This feature permits serological tests to differentiate non-infected vaccinated animals from infected vaccinated animals.

Transmission occurs by direct contact with infected animals that excrete the virus in saliva, faeces, urine, milk, semen, ocular, and nasal discharges. Infected animal products, contaminated objects, and transmission by aerosol for distances up to 60 km overland and 300 km by sea have been reported (Gloster et al 1982).

13.1.5. Hazard identification conclusion

FMDV is identified as a potential hazard in porcine semen.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

Following infection, FMDV spreads from the original site of infection to the testes and accessory glands via the lymphatics and blood stream, and the virus may also multiply in the skin around the preputial orifice (Guérin and Pozzi 2005).

FMDV has been isolated from porcine semen (Thacker et al 1984). More recently, quantitative real-time polymerase chain reaction tests have shown that FMDV can be detected in the semen of an infected boar as soon as 1 day after infection (van Rijn et al 2004).

The likelihood of entry is assessed to be high.

13.2.2. Exposure assessment

Although artificial insemination using contaminated semen did not transmit infection to inseminated sows (McVicar et al 1977), these transmission studies have not been repeated. However, pigs are recognised to be important amplifying hosts for FMDV and are much more susceptible to infection by the oral route than are ruminants (AQIS 2000).

The likelihood exposure is assessed to be low.

13.2.3. Consequence assessment

Animals that become infected could become the focal point for an outbreak of foot and mouth disease. Where large numbers of pigs are infected, airborne spread over considerable distances has been described. For example, the virus was transmitted by favourable winds from Brittany in France to the Isle of Wight in England (Gloster et al 1982). Infected individuals may infect fomites and the movement of infected pigs could result in widespread exposure of other susceptible cloven-hoofed animals.

An outbreak of FMDV would cause serious disruption to the livestock industries, economic losses to individual farmers, very large expenses for an eradication campaign, and significant disruption to export markets for both animals and animal products. The overall effects could be catastrophic as dramatically demonstrated by the losses that resulted from an outbreak of the

disease in Britain where the costs to government were estimated at 3.1 billion pounds (Thompson et al 2002).

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

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

The consequences are assessed to be high.

13.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for FMDV is non-negligible and it is assessed to be a risk in the commodity. Therefore risk management measures can be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

Article 8.5.15. of the OIE *Code* recommends that, for fresh porcine semen imported from FMD free countries or zones where vaccination is not practised or FMD free compartments, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

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

Article 8.5.16. of the OIE *Code* recommends that, for frozen porcine semen imported from FMD free countries or zones where vaccination is not practised or FMD free compartments, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

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

Article 8.5.17. of the OIE Code recommends that, for porcine semen imported from FMD free countries or zones where vaccination is practised, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the donor animals:

a. showed no clinical sign of FMD on the day of collection of the semen and for the following 30 days;
b. were kept for at least three months prior to collection in a FMD free country or zone;
c. if destined to an FMD free country or zone where vaccination is not practised:
    i. have not been vaccinated and were subjected, not less than 21 days after collection of the semen, to tests for antibodies against FMD virus, with negative results; or
    ii. had been vaccinated at least twice, with the last vaccination not more than 12 and not less than one month prior to collection;

2. no other animal present in the artificial insemination centre has been vaccinated within the month prior to collection;
3. the semen:

    a. was collected, processed and stored in conformity with the provisions of Chapters 4.5. and 4.6.;
    b. was stored in the country of origin for a period of at least one month following collection, and during this period no animal on the establishment where the donor animals were kept showed any sign of FMD.

Article 8.5.18. of the OIE Code recommends that, for porcine semen imported from FMD infected countries or zones, Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

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

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

Because of the extreme seriousness of the disease and the catastrophic consequences that would result from its introduction, it may be concluded that importation of porcine semen should be limited to countries or zones that are free of FMDV in accordance with the OIE *Code*, or countries or zones in which compliance with measures in accordance with the recommendations

of the OIE *Code* for import of germplasm from FMD infected countries or zones has been reviewed and accepted by MPI.

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

Option 1

The international standards described in articles 8.5.15 to 8.5.18. of the OIE *Code* could be adopted to effectively manage the risk associated with FMDV in porcine semen.

Option 2

In addition to the measures described in Option 1, MPI may also require approval of each semen collection and processing and storage facility/ies in the exporting country intended to be used during the preparation of an export consignment to New Zealand. This approval may be dependent on the facility, its location and operating standards and that the verification systems of the Veterinary Authority achieve a very high level of risk management for FMD. The process for MPI approval may include site inspection. MPI may also reserve the right to supervise collection, require the use of New Zealand approved semen collection personnel, or require any other measures deemed necessary to ensure compliance with facility and operating standards upon which the approval is based.

Option 3

Imports of porcine semen could be limited to countries recognised to be free of FMDV without vaccination.

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14. Japanese encephalitis virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Flaviviridae*, Genus: *Flavivirus*, Species: *Japanese encephalitis virus* (JEV) (Thiel et al 2005).

14.1.2. OIE list

Listed (OIE 2008).

14.1.3. New Zealand status

Exotic notifiable organism (MAF 2008).

14.1.4. Epidemiology

Japanese encephalitis is a zoonosis that results in 30-50,000 human disease cases per year in the parts of Asia where it is endemic (CDC 2006). The causative virus is maintained in a bird (particularly egrets and herons) to mosquito cycle. Pigs are the primary amplifying host (Geering et al 1995). Infection of humans and horses may cause a severe and often fatal encephalitis. Although clinical signs are not common in pigs in endemic areas, there can be significant rates of abortion in immunologically naive sows (Hoke and Gingrich 1994; Platt and Joo 2006). Inapparent infections also occur in goats, sheep, cattle, and dogs, and have been reported in cats, rodents, bats, snakes, and frogs (Geering et al 1995).

Flaviviruses exhibit a high degree of specificity in their ability to infect and be transmitted by individual insect species, or even strains of individual species. Vector competence is under genetic control, with the susceptibility of the midgut epithelium being the primary determinant. In a susceptible insect, a sufficient concentration of the virus must be ingested to exceed the midgut infection threshold (Monath and Heinz 1996).

Although 28 mosquito species have exhibited vector competence for JEV in field and laboratory studies, only a few species found in endemic areas develop sufficient abundance, have long enough flight ranges, exhibit sufficient longevity, and have the breadth of host feeding preferences to become natural vectors (Hoke and Gringrich 1994). In mainland Japan and Okinawa, the Philippines, the Korean peninsula, China, Taiwan, the Indochina peninsula (except Malaysia), Indonesia, Sri Lanka, India, and Nepal, the primary vectors belong to the *Culex vishnui* group, mainly *Cx. tritaeniorhynchus*. However, other mosquitoes are important vectors locally. These include *Cx. vishnui* in India, Thailand, and Taiwan, *Cx. fuscocephala* in Malaysia, Thailand and Taiwan, *Cx. gelidus* in Indonesia, Thailand, and Vietnam, and *Cx. annulus* in Taiwan. The type of available larval habitat determines which species will predominate. Both *Cx. tritaeniorhynchus* and *Cx. vishnui* breed predominantly in rice paddies and are therefore the most important rural vectors (Hoke and Gingrich 1994).

None of the above mosquito species are present in New Zealand (Belkin 1968). Nevertheless, the introduced mosquito *Cx. quinquefasciatus* has been shown to be a competent vector of the virus under laboratory conditions, and the virus has also been isolated from naturally infected mosquitoes of this species (Van den Hurk et al 2003).

JEV occurs throughout the temperate and tropical regions of Asia and has also spread to Indonesia, northern Australia, Papua New Guinea and possibly Pakistan (Center for Food Security and Public Health 2007). Despite rare appearances in northern Australia, the virus remains largely confined to the endemic areas of Asia and it is highly doubtful whether it could establish in New Zealand if introduced. The occurrence of JEV in Australia is sporadic and confined to the far northern regions and the Torres Strait islands (Johansen et al 2003; Liu et al 2005; Mackenzie et al 1994; Ritchie and Rochester 2001).

JEV has been isolated from the testicles of boars with orchitis and from the semen of infected boars for up to 5 weeks following infection (Ogasa et al 1977; Guérin 1995).

14.1.5. Hazard identification conclusion

Transmission by semen has been described. Therefore, JEV is identified as a potential hazard in pig semen from infected areas.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Mosquitoes blown from Papua New Guinea seem to have been responsible for outbreaks of JE in the Torres Strait islands since 1995 and in Northern Australia in 1998, but there is no evidence that the virus may become established on the Australian mainland (Russell 1998). The last report of the virus on Australian territory involved a seropositive pig on a northern island in the Torres Strait, and it remains exotic to the Australian mainland (Anonymous 2007).

Therefore the likelihood of entry in pig semen from countries where JEV is endemic or from northern Australia is assessed to be non-negligible.

14.2.2. Exposure assessment

Exposure in the first instance would be limited to inseminated animals. In view of the highly complex natural history of this disease, which involves species of birds and high densities of pigs and insects in climates and ecosystems which are not found in New Zealand, even if the virus were introduced into New Zealand in this way it is extremely unlikely that any transmission would occur. Climatographs, plotted on a monthly basis (Gerlach 1974), show that the hottest part of New Zealand is the far North in the month of February. The mean daily temperature in that month is 20ºC (the mean daily minimum and the mean daily maximum during that month is 17ºC and 29ºC respectively). However, summer is also the driest time of the year in New Zealand; the mean February rainfall in Northland is less than 100 mm. Thus, even if a suitable vector did exist in New Zealand, the time of the year with the most suitable temperature for the development of the virus in vectors would be least suitable for the build-up of high vector densities. In addition, amplification of the virus requires a high density of seronegative pigs, which would probably only be found in areas where intensive pig production is practised, and this is limited in most parts of New Zealand, especially in the far north.

Considering that the virus has never established outside the endemic areas of Asia, the absence of major mosquito vectors, the unsuitable climate and the absence of high densities of pigs, it is considered highly unlikely that JEV could establish in New Zealand. However, in view of the presence of a potential mosquito vector in this country, the likelihood of exposure is assessed to be non-negligible.

14.2.3. Consequence assessment

In the unlikely event that any consequences were to result from the insemination of sows with imported semen that might harbour JEV, these would in the first instance be restricted to the animals that were inseminated. The primary disease manifestation of infection in sows and gilts is abortion and abnormal farrowings. Sexually mature swine do not show any significant clinical signs of infection, but transient anorexia and a mild febrile response have been observed (Platt and Joo 2006). As there are no reports of disease in non-pregnant sows, it is concluded that the immediate consequences of insemination with infected semen would be negligible.

For any secondary consequences to arise beyond the inseminated sows, there would need to be viraemia in the inseminated animal and transmission by a competent vector mosquito. While the likelihood of this occurring in New Zealand is very low, for the purposes of this risk analysis it is assessed to be non-negligible.

Infection of humans and horses may result in a severe and often fatal encephalitis. Further, if the virus were transmissible via mosquitoes in this country, there could be some level of abortions in immunologically naïve sows (Hoke and Gingrich 1994).

Therefore the consequences for infected humans, pigs and horses would be non-negligible. As infections in other species are asymptomatic, the environmental consequences would be negligible.

14.2.4. Risk estimation

Entry, exposure and consequence assessments are non-negligible. Therefore, JEV is assessed to be a risk in semen from countries where the virus is endemic or northern Australia.

14.3. RISK MANAGEMENT

14.3.1. Options

The OIE *Code* does not include recommendations for managing the risk of JEV in semen of any species.

The options available for managing the risk of the virus in the commodity are:

Option 1

Semen donors could be tested for presence of antibody at the time of collection and again 30 days after collection of semen. Importation of semen from donors that were positive at the time of collection could be permitted on the basis that seropositive animals are not viraemic. Semen from boars that were negative at the time of collection and negative 30 days later could be permitted while semen from donors that were negative at collection and positive 30 days later could be prohibited on the basis that they may have been viraemic at the time of semen collection.

Option 2

Importation could be restricted to semen collected from donors resident in regions that are free from JEV.

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15. Kunjin virus

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Family: *Flaviviridae,* Genus: *Flavivirus,* Species: *Kunjin virus* (KUN) (Thiel et al 2005).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Exotic.

15.1.4. Epidemiology

KUN is considered to be almost identical to West Nile virus (Spurr and Sandlant 2004). It occurs widely in Australia where it is considered endemic in the tropical regions of northern Western Australia, the Northern Territory, and northern Queensland (Hall et al 2001; Mackenzie et al 2003).

Human infections give rise to mild fever, headache, photophobia, rash, arthraligia, myalgia and lymphadenopathy, although severe encephalitis has also been described (Hall et al 2001).

The principal mosquito vector of KUN is considered to be *Culex annulirostris*, although virus has also been recovered from *Cx. australicus, Cx. squamous, Cx. Quinquefasciatus,* and *Aedes tremulus* (Mackenzie et al 1984). Birds are the major vertebrate hosts of KUN, particularly ardeid waterbirds of the order Ciconiiformes (Marshall et al 1982).

Spradbrow (1972) described low-titre serological evidence of exposure of pigs to KUN in northern Queensland. Similarly, serological evidence of infection of feral pigs in New South Wales was described by Gard et al (1976). Experimental inoculation of pigs is followed by low levels of detectable antibody (Williams et al 2001). There is no evidence to suggest that transmission of KUN in porcine semen should be considered likely.

15.1.5. Hazard identification conclusion

Although there is serological evidence of exposure of pigs in Australia to KUN, they are not considered to be a major vertebrate host of this virus and there is no evidence of virus being found in the semen of infected pigs. KUN is not identified as a hazard in porcine semen.

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16. Menangle virus

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

The International Committee for the Taxonomy of Viruses has identified Menangle virus (porcine paramyxovirus) as a tentative species in the genus *Rubulavirus* of the family *Paramyxoviridae* (Lamb et al 2005).

16.1.2. OIE list

Not listed.

16.1.3. New Zealand status

The virus is classified as "other exotic virus" (MAF 2008).

16.1.4. Epidemiology

The virus has only been found in Australia, where it caused a single outbreak of disease in 1997 (Chant et al 1998; Kirkland and Stephano 2006; Philbey et al 1998). All information in this section comes from these sources.

Infection has been associated with reproductive disease including abortions, mummified foetuses and congenital defects. During the initial outbreak, returns to mating occurred and pseudopregnancy was common. The incubation period has not been accurately determined and the period for which sows remain infected is not known. A carrier state does not occur. Therefore, it can be assumed that serologically-positive animals are not carriers of the virus. Sows become immune and pass antibody to newborn piglets in their colostrum. Once the population had become immune, only young pigs were infected after their maternal immunity had declined.

The disease did not spread to other pig herds except to two associated growing farms that did not have any breeding sows and received pigs from the parent farm. A colony of fruit bats roosted within 200 meters of the parent farm. Virus neutralising antibody was found in approximately one-third of fruit bats in the colony on the affected farm and from fruit bats in a variety of locations in Australia. Therefore fruit bats were identified as the probable natural reservoir host of the virus. Pigs were infectious and the virus spread slowly through the entire pig population. Six months after the estimated time of entry, 90% of pigs were positive to a virus neutralization test and antibody titres remained high for at least 2 years.

Two workers on the affected farm became seropositive and developed a severe febrile illness with a rash and had high convalescent-phase antibody titres to the virus. There was no serological evidence of an alternative cause. It was concluded that there was strong evidence that the two men were infected with Menangle virus.

The virus does not survive long in the environment and there have been no other recorded outbreaks of the disease. There is no evidence to suggest that transmission in the semen of infected pigs should be considered likely.

16.1.5. Hazard identification conclusion

Menangle virus occurs only in Australia. Only one outbreak of the disease has occurred in Australia in the last 10 years, and the virus cannot be regarded as endemic in the pig population. There is no evidence that transmission occurs via semen. Menangle virus is not identified as a hazard in the commodity.

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17. Murray Valley encephalitis virus

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Family: *Flaviviridae*, Genus: *Flavivirus*, Species: Murray Valley encephalitis virus (MVEV) (Thiel et al 2005).

17.1.2. OIE list

Not listed

17.1.3. New Zealand status

Exotic unwanted organism (MAF 2008).

17.1.4. Epidemiology

MVEV is found in Australia, Papua New Guinea, and probably islands in the eastern part of the Indonesian archipelago (Mackenzie et al 1994). Waterbirds are thought to be the major vertebrate host of the virus, especially members of the order Ciconiiformes, with the highest serological titres recognised in the rufous night heron (Boyle et al 1983).

Culex annulirostris is recognised as the major vector of MVEV in Australia. The virus has also been recovered from other mosquito species including *Aedes normanensis*, *Ae. pseudonormanensis*, *Ae. eidsvoldensis*, *Anopheles annulipes*, *Anopheles bancroftii*, *Cx. quinquefasciatus*, *Cx. australicus*, *Cx. palpalis*, and *Monsonia uniformis* (Mackenzie et al 1994). Of these, only *Cx. quinquefasciatus* (an introduced mosquito species known to be present in New Zealand [Holder 1999]) has been shown experimentally to transmit MVE.

Serological evidence of exposure to MVE has been described in domestic pigs in northern Queensland (Doherty et al 1964) and in feral pigs in New South Wales (Gard et al 1976). Experimental inoculation studies have shown that pigs which develop low-grade viraemia following infection with MVEV should be considered low-grade amplifiers of this virus (Kay et al 1985). There is no evidence to suggest that transmission of MVEV in porcine semen should be considered likely.

17.1.5. Hazard identification conclusion

Although there is serological evidence of exposure of pigs in Australia to MVEV, pigs have been shown to develop only low-grade viraemia following infection and there is no evidence of virus being found in the semen of infected pigs. MVEV is not identified as a hazard in porcine semen.

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18. Nipah virus

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

Nipah virus (NiV) is a paramyxovirus in the subfamily Paramyxovirinae, and is classified in the new genus *Henipavirus* (Eaton et al 2006; Wang et al 2012).

18.1.2. OIE list

Nipah virus encephalitis is an OIE-listed disease.

18.1.3. New Zealand status

Nipah virus is exotic to New Zealand and listed as a Notifiable Organism (Tana et al 2011).

18.1.4. Epidemiology

Nipah virus is a tropical disease that was first reported in Malaysia in 1998 and subsequently in Singapore, Bangladesh, and India (Tan and Wong 2003; Katu 2004; Epstein et al 2006). The disease is rare, appearing sporadically in tropical climates where the natural reservoir host *Pteropid* fruit bat species are found.

Respiratory disease in pigs caused by NiV is often subclinical (OIE 2010). Pigs excrete the virus in urine and respiratory droplets. Chronic infections do not appear to be a feature of the disease. Experimental infections showed that virus was not excreted by pigs once neutralising antibodies appeared 14-18 days post-infection (Middleton et al 2002). Direct, close contact with pigs was the primary source of a major public health crisis, with the death of 105 people, when NiV first appeared (Katu 2004). Nipah virus attacks the central nervous system and respiratory systems. Encephalitis is the main cause of death in humans. The movement of infected pigs from Malaysia to an abattoir in Singapore resulted in the development of symptoms of encephalitis in 35 abattoir workers with confirmed infection by NiV demonstrated in 11 of these individuals. One of these infected individuals died whilst four were reported to have persistent neurological deficits (Paton et al 1999; Chua et al 2000).

In Bangladesh from 2001 to 2005, five outbreaks were attributed to NiV infection. These involved much smaller numbers of affected humans and no animal disease was evident, differing from the Malaysian epidemic. These outbreaks appear to have been due to spillover of virus directly from bats to humans (Epstein et al 2006). One outbreak was reported in 2001 in India, close to the Bangladesh border (Chadha et al 2006). Since 2001, almost annual human outbreaks of fatal encephalitis caused by NiV in Bangladesh and sporadic outbreaks in India have been reported. Although human-to-human transmission was not seen in the Malaysia and Singapore outbreak, the recent outbreaks in Bangladesh have led to the suspicion of human-to-human and foodborne transmission of NiV (Lo et al 2012). The threat to human health is significant since NiV infection has a high mortality rate of 33-75% recorded in different outbreaks (Center for Food Security and Public Health 2007).

Virus isolation of NiV from unfixed samples followed by identification procedures such as immunostaining, serum neutralisation, or molecular characterisation are described. Real-time polymerase chain reaction is also available as a diagnostic test. Immunohistochemistry of fixed tissue can also be used to detect NiV antigens. Virus neutralisation tests and enzyme-linked immunosorbent assays are also available for serological diagnosis of NiV infection (OIE 2010).

Virus spread between farms is usually associated with pig movements although transmission in semen has been suggested (Center for Food Security and Public Health 2007). NiV was not found in the urine of experimentally infected pigs although virus was detected in the kidneys of clinically infected individuals suggesting that urinary excretion of the virus may be possible (Middleton et al 2002). NiV has been recovered from the urine of infected bats (Wacharapluesadee and Hemachudha 2007), cats (Mungall et al 2007), and humans (Chua et al 2001). Natural infection of pigs with NiV leads to lesions in both the brain and lungs of an individual with tracheitis, pneumonia, and meningoencephalitis described. The lungs and meninges are recognised as the most important target organs following experimental infection with vascular degeneration also described in the gastric submucosa and muscle, ureteral submucosal, spleen, and hepatic arterioles. The spread of NiV from infected pigs is principally through coughed-up sputum and expired air, with excretion also possible via the placenta (Hooper et al 2001). There is no evidence that suggests NiV is shed in the semen of infected pigs.

18.1.5. Hazard identification conclusion

There is no evidence to support the suggestion that NiV may be spread in the semen of infected boars. NiV is not identified as a hazard in porcine semen.

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19. Porcine reproductive and respiratory syndrome virus

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Family: *Arteriviridae*, Genus: *Arterivirus*, Species: *Porcine reproductive and respiratory syndrome virus* (PRRSv) (Snijder et al 2005).

19.1.2. OIE list

PRRS is listed in the *Code*, but it does not contain a chapter on this disease (OIE 2008a).

19.1.3. New Zealand status

Exotic, notifiable disease (MAF 2008).

19.1.4. Epidemiology

The disease/virus occurs in the USA, Canada and many EU countries but not in Norway, Sweden, or Australia (OIE 2008b). The virus is highly infectious and spread rapidly through Europe and North America in the early 1990s. In infected regions 60-80% of herds are typically infected (Zimmerman et al 2006).

Infection with virulent strains results in viraemia within 12 to 24 hours. Viral titres reach maximal levels by the seventh day after infection and are highest in the lung. Signs of infection are typically those of viraemia, followed by respiratory signs and reproductive problems such as abortion, foetal deaths and reproductive failure (Zimmerman et al 2006).

Infected pigs shed the virus in saliva, nasal secretions, urine and semen (Zimmerman et al 2006). Pigs may remain persistently infected for periods of at least 150 days after infection (Allende et al 2000), with virus multiplying in the cells of subclinically infected pigs for several months (Zimmerman et al 2006). The persistence of the virus in infected pigs is independent of the route or time of infection and occurs despite the concomitant presence of antibody.

Transmission commonly occurs through skin wounds. Oral and nasal transmission is less efficient, and airborne transmission over short distances (1-2.5 meters) has been demonstrated (Zimmerman et al 2006). Airborne transmission over longer distances is controversial but recent studies have shown that airborne spread from an infected herd is possible over distances of around 120 meters (Pitkin et al 2007). Viral RNA has been demonstrated in semen 92 days after infection and the virus has been isolated from the bulbourethral gland 101 days after infection (Christopher-Henning et al 1995). Artificial insemination has been a common method of transmission of the virus (Zimmerman et al 2006).

Diagnosis can be confirmed by isolation of the virus or by demonstration of viral RNA by PCR (Ludeman and Lager 2008; Zimmerman et al 2006). Antibodies develop within 1-2 weeks, are maximal 30-50 days post infection, and decline to undetectable levels 3-5 months after infection (Zimmerman et al 2006). ELISA kits are commercially available with some using antigen prepared from both American and European strains of the virus (Ludeman and Lager 2008).

19.1.5. Hazard identification conclusion

Transmission of PRRSV in semen is well documented. The virus is identified as a potential hazard in porcine semen imported from countries where the virus is present.

19.2. RISK ASSESSMENT

19.2.1. Entry assessment

PRRS is endemic in Canada, the USA, and most EU countries where the prevalence in many areas is high. The organism is commonly excreted in semen. Therefore the likelihood of entry of the virus in imported semen is non-negligible.

19.2.2. Exposure assessment

Since imported semen will used to inseminate sows, exposure of naïve New Zealand pigs to virus in imported semen would be inevitable.

19.2.3. Consequence assessment

Insemination of New Zealand pigs with infected imported semen would almost certainly result in infection of the recipients. It can be expected that the virus would establish in herds that are infected by imported semen and spread rapidly as it did in other countries. The disease would initially cause high losses to farmers and as the infection became endemic would lead to a continuous erosion of profits. It may be expensive and difficult to eradicate from New Zealand.

The virus is not zoonotic and there would be no consequences for human health. The only animals known to be infected by the virus are pigs and feral pigs would be the only susceptible species of wild or feral animals. However, transmission from domestic to feral pigs is unlikely. Furthermore it is likely that the disease would be self-limiting since under the conditions in which feral pigs live the virus may not re-cycle between litters of piglets. Therefore the likelihood of deleterious effects on humans or the environment is assessed to be negligible.

19.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for PRRS virus is non-negligible and it is classified as a risk in the commodity. Therefore risk management measures can be justified.

19.3. RISK MANAGEMENT

19.3.1. Options

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

- The virus is excreted in pig semen and transmission in semen is common.
- Since pigs may remain infected for at least 5 months, quarantine is not an option for preventing the introduction of the virus.
- Serological tests and viral RNA detection tests (PCR) are available for diagnosis.
- Introduction of the disease could have severe consequences for the pig industry.

Of all the OIE-listed porcine diseases that are considered to be a hazard in porcine semen, it is noteworthy that PRRS is unique in that no *Code* chapter has been written to outline standards for sanitary measures to manage the risk of international transmission of this disease.

Chapters 4.5 and 4.6 of the *Code* (OIE 2008a) contain requirements for PRRSv for boars standing at semen collection centres. Under these requirements, boars from a country or zone that is not free of PRRS must be clinically healthy, physiologically normal, and subject to testing

complying with the standards in the Terrestrial Manual. Boars must remain in this quarantine station for at least 28 days and, at least 21 days after entering the quarantine station must be tested negative for PRRSv using a test compliant with the standards of the Terrestrial Manual. Any boars resident in the semen collection facility must also be tested at least annually using a test compliant with the standards in the Terrestrial Manual.

These requirements from the *Code* may provide some assurance that infected boars are not introduced into a stud, although ensuring a stud remains free of infection is recognised to be extremely challenging (Huinker 2002; Connor 2003; Polson and Reicks 2009) so annual testing alone (as described above) cannot be relied upon to ensure semen is free from PRRSv.

Previously, PCR testing of semen has been used routinely in boar studs to monitor for PRRS (Connor 2003; Torremorell 2003). However, following infection, virus can be detected sooner in blood than in semen (Christopher-Hennings et al 2001). Serum PCR (using either a TaqMan PCR test or a nested PCR test) is more sensitive than semen PCR for PRRSv detection during the first six days following infection and boars are detected as PRRSv-positive by serum PCR before semen PCR (Reicks et al 2006a; Reicks et al 2006b). For testing boars, serum is currently considered to be the preferred sample to use as PRRSv is in much higher levels in serum than in semen and is detectable at least 1-2 days sooner (Reicks 2009). PCR testing of pooled serum samples have been shown to be as effective for detection of PRRSv in boar studs as testing individual samples (Rovira and Munoz-Zanzi 2006).

The current state-of-the-art principles for early detection of PRRSv infection in boar studs include prohibition of vaccination together with avoidance of any seropositive animals, monthly serological testing of the stud population, antigen testing of donors at (weekly) collection, antigen testing of pooled semen samples (daily), and no use of semen until all individual animal tests have been completed. However, despite these stringent measures, biosecurity breakdowns in boar studs are recognised (Clement 2010).

In order to minimise the risk of distributing semen containing PRRSv, it has been recommended that boars should be sampled each day of collection. Additionally, a statistical sampling of all boars on a stud should be collected each day, any boar showing clinical signs such as anorexia or pyrexia should be sampled immediately, and all semen should be withheld until negative results (using PCR testing of serum) are obtained. Due to viral mutation, some strains of PRRSv may be undetected by PCR testing so additional weekly testing of boars by serum ELISA is also recommended (Reicks 2005; Reicks 2009).

NZPork have stated that it would also be desirable to require semen to be withheld for a period of three months after collection and only released if accompanied by certification that during the three months since the last collection date there has been no clinical or serological evidence of PRRS in the donor herds or the stud itself (Clement 2010).

Considering the above, the following options could be considered to effectively manage the risk of introducing PRRSv in imported porcine semen:

Option 1

Pig semen could be imported from countries that are recognised as free from PRRS without sanitary measures.

Option 2

a. Boars should be sourced from donor herds that do not vaccinate against PRRSv, and be shown to be negative for PRRSv before entering the stud as described in Chapters 4.5 and 4.6 of the *Code*.

b. Individual donor boars should be tested by serum PCR each day of collection with negative results.

c. During each day of the collection period, all boars on the stud should be tested for PRRSv by serum PCR, with the number of boars sampled sufficient to give a 95% confidence of detecting a 5% prevalence rate.

d. 30 to 50 days after the final sample collection, boars on the stud should be tested by a multi-valent serum ELISA for PRRSv that uses both European and American strain antigens, with the number of boars sampled sufficient to give a 95% confidence of detecting a 5% prevalence rate.

Option 3

a. Boars should be sourced from donor herds that do not vaccinate against PRRSv, and be shown to be negative for PRRSv before entering the stud as described in Chapters 4.5 and 4.6 of the *Code*.

b. Individual donor boars should be tested by serum PCR each day of collection with negative results.

c. During each day of the collection period, all boars on the stud should be tested for PRRSv by serum PCR, with the number of boars sampled sufficient to give a 95% confidence of detecting a 5% prevalence rate.

d. 30 to 50 days after the final sample collection, boars on the stud should be tested by a multi-valent serum ELISA for PRRSv that uses both European and American strain antigens, with the number of boars sampled sufficient to give a 95% confidence of detecting a 5% prevalence rate.

e. Semen should be accompanied by certification that during the three months since the last collection date there has been no clinical or serological evidence of PRRS in the donor herds or the stud itself.

Option 4 (the current measures in place based on an earlier risk analysis)

a. The semen collection centre could be required to have a documented absence from PRRS. All pigs entering the semen collection centre must originate from herds which have never recorded a clinical case of PRRS.

b. The semen collection centre could be required to have never used a modified live PRRS virus vaccine nor introduced pigs from herds that have used a modified live PRRS virus vaccine.

c. All pigs in the semen collection centre could be required to have completed a 5 week period of isolation. While undergoing this 5 week isolation period the pigs must be exposed throughout the isolation period to direct contact with at least an equal number of sentinel grower pigs and the total number of pigs in the semen collection centre undergoing isolation could be required to be at least ten at all times.

d. The sentinel grower pigs used could be required to be between 12 and 24 weeks of age and derived from 3 or more herds which have been shown by an approved multi-valent ELISA to be free of PRRS within two months prior to the commencement of the isolation period.

e. During the isolation period, all pigs undergoing isolation could be tested for PRRS using an approved multi-valent ELISA test, on two occasions at the start and finish of the isolation period, with a negative result in each case.

f. Semen collected from donor boars during the 5 week period of isolation (either during approval of the semen collection centre or prior to entry of a donor boar onto an approved semen collection centre) could become eligible for export to New Zealand upon successful completion of isolation (i.e. no positive test for PRRS in any donor boar or sentinel simultaneously undergoing isolation).

Option 5

Reflecting that breakdowns in boar studs still occur despite intensive monitoring, the importation of pig semen from countries with PRRSv could be prohibited.

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20. Porcine epidemic diarrhoea virus

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*, Species: *Porcine epidemic diarrhea virus* (Spaan et al 2005).

20.1.2. OIE list

Not listed.

20.1.3. New Zealand status

Listed as an "other exotic virus" (MAF 2008).

20.1.4. Epidemiology

Information given below is derived from a comprehensive review of the subject (Pensaert and Yeo 2006).

Porcine epidemic diarrhoea occurs in the EU but neither in North America nor Australia. The disease was probably first recognized in England in 1971, but the cause was not established until 1978. The disease spread from England to Europe. Outbreaks of diarrhoea occur in pigs of all ages. Older pigs generally recover within a week, while mortality in piglets under 7 days old is around 50% and may approach 100%. The morbidity may be close to 100% in naïve herds. The prevalence of the disease in Europe has declined and it is now rare. The disease may become endemic on a farm where the number of pigs is high and the virus is maintained by circulation in litters of piglets.

Experimentally-infected, colostrum-deprived piglets develop the disease in 22-36 hours, and on farms with susceptible populations of pigs the disease appears 4-5 days after introducing new pigs. The virus multiplies only in the cells of the digestive tract and is transmitted by the faecal-oral route. There is no evidence to indicate that the virus is excreted in semen or that semen is a vehicle for the transmission of virus.

20.1.5. Hazard identification conclusion

Porcine epidemic diarrhoea is predominantly a disease of very young piglets. The causative virus multiplies only in the cells of the digestive tract and there is no evidence that it is excreted in semen. Therefore, this virus is not identified as a hazard in the commodity.

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21. Porcine myocarditis virus

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

The aetiological agent of this new disease has been identified as a pestivirus and given the name Bungowannah virus (Kirkland et al 2007).

21.1.2. OIE list

Not listed.

21.1.3. New Zealand status

Exotic.

21.1.4. Epidemiology

A new disease occurred in pigs in Australia in 2003 (McOrist et al 2004). The disease was shown to be caused by an infectious agent and characterized by neonatal deaths and stillbirths. The pathological changes consisted of multifocal non-suppurative myocarditis and sometimes myonecrosis with crystalline arrays of virus particles identified in heart tissue (Kirkland et al 2007; McOrist et al 2004).

The condition was confined to two farms. There was some movement of breeding sows between the two properties (McOrist et al 2004). Since no further cases have been described it has apparently not spread.

The virus has characteristics of a pestivirus but shows significant divergence from other known pestiviruses (Kirkland et al 2007). It has been isolated in culture and identified by immunoperoxidase staining using polyclonal antibody from a pig infected with the virus. Sera from infected pigs react weakly with other pestiviruses in an agar gel immunodiffusion test but do not exhibit neutralizing activity against other pestiviruses.

The epidemic features of the disease outbreak suggest that the epidemic spread by contact through naïve breeding sows and ceased when naïve sows were no longer available. There was no evidence indicating that long term carriers of the disease occurred. Despite the severity of the outbreaks the herds made relatively rapid recoveries.

The disease has not been described in any other country. A recent report has confirmed Bungowannah-like Pestivirus is unlikely to be present in swine in the upper Midwestern USA (Abrahante et al 2012).

21.1.5. Hazard identification conclusion

As Bungowannah virus is a pestivirus, it is considered likely that it is present in semen from infected boars. Since the virus occurs only in Australia, it is identified as a potential hazard in the commodity from that country.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Although the disease occurs extremely rarely and there is no evidence suggesting that long-term carriers of virus occur, given that the Bungowannah virus has been identified as a Pestivirus, it is assumed that there is a non-negligible likelihood of transmission in semen (see Chapter 8).

21.2.2. Exposure assessment

Imported semen will be inseminated into New Zealand pigs. As it has been demonstrated that sows can be infected with CSFV by artificial insemination with infected semen (Floegel et al 2000), it is assumed that this is also likely for pigs that are infected with Bungowannah virus, the likelihood of exposure of New Zealand pigs is assessed to be non-negligible.

21.2.3. Consequence assessment

As described above, infection of pigs is characterized by neonatal deaths and stillbirths, with pathological changes described as multifocal non-suppurative myocarditis and sometimes myonecrosis with crystalline arrays of virus particles identified in heart tissue. The consequences of infection are therefore assessed to be non-negligible.

21.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for Bungowannah virus is non-negligible and it is classified as a risk in the commodity. Therefore risk management measures can be justified.

21.3. RISK MANAGEMENT

21.3.1. Options

Porcine myocarditis is confined to a single enterprise composed of a number of small properties in New South Wales and there have been no other reports of the disease outside this enterprise. This enterprise includes an AI facility which used boars from the positive site after testing with a protocol to ensure they are antibody positive and serum negative for Bungowannah virus (Biddle 2010). Since this enterprise has was placed under quarantine in 2003, approximately 275,000 semen doses each year have been used to inseminate 50,000 sows in both the positive site and in negative farms with no further spread of this disease. Considering there has been no further spread of the virus, it is reasonable to conclude that these current measures are effectively managing the risk of spread in porcine semen.

A peroxidise-linked assay to detect antibodies to Bungowannah virus and an RT-PCR test to detect Bungowannah virus RNA have been described (Finlaison et al 2009).

Option 1

Porcine semen could be imported from countries other than Australia without sanitary measures.

Option 2

Semen originating from Australian studs which can be certified as not including individuals from properties where porcine myocarditis has been identified could imported without any further sanitary measures.

Option 3

Donor boars originating from properties where porcine myocarditis has been recognised should be isolated and tested to demonstrate they are seropositive and negative for Bungowannah virus RNA before entering the collection centre.

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22. Porcine respiratory coronavirus

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*. The International Committee on Taxonomy of Viruses considers *Transmissible gastroenteritis virus* and *Porcine respiratory coronavirus* to be a single species (Spaan et al 2005).

22.1.2. OIE list

Not listed.

22.1.3. New Zealand status

Listed as an "other exotic organism" (MAF 2008).

22.1.4. Epidemiology

Porcine respiratory coronavirus (PRCV) is probably a deletion mutant of the transmissible gastroenteritis virus (TGEV) (Rasschaert et al 1990). PRCV was first isolated in 1984 following the discovery of unexplained antibody titres to TGEV in pigs (Pensaert et al 1986). The virus spread widely through Europe and later through North America (Saif and Sestak 2006). Natural infections with the virus are generally subclinical, but experimental infections with high challenges of virus may result in mild respiratory signs. Concomitant infections with other viruses, particularly porcine respiratory and reproductive syndrome virus, result in more severe signs (Saif and Sestak 2006). The emergence of the virus in Europe coincided with the decline in the occurrence of TGE and has lead to speculation that infection with PRCV provides protection against TGEV (Saif and Sestak 2006). Experimental studies have shown that infection with PRCV provides partial protection against TGEV (Cox et al 1993; Wesley and Woods 1993; Wesley and Woods 1996).

PRCV multiplies in the respiratory tract and tonsils and may be isolated from these sites. Limited multiplication may occur at enteric sites where only a few scattered cells containing virus may be demonstrated, even following direct inoculation of virus into the intestinal lumen (Saif and Sestak 2006). No reports could be found that suggest that PRCV is excreted in semen.

Infection with PRCV is of little economic importance as it usually causes only subclinical infections. It may even be beneficial in providing at least a partial immunity against TGEV. However, the presence of PRCV in a pig population complicates the diagnosis of TGE due to cross reactions.

22.1.5. Hazard identification conclusion

PRCV causes a mild, usually subclinical, infection in pigs. There is no evidence that the virus is excreted in semen. Therefore it is not identified as a hazard in the commodity.

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23. Rabies virus

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Family: *Rhabdoviridae*, Genus: *Lyssavirus*, rabies virus. In addition to the true rabies virus there are a number of closely related lyssaviruses such as the European bat *Lyssavirus* which cause similar diseases.

23.1.2. OIE list

Listed (OIE 2008a).

23.1.3. New Zealand status

Exotic, notifiable organism (MAF 2008).

23.1.4. Epidemiology

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

With some exceptions (particularly islands), rabies is found worldwide. The United Kingdom, Ireland, Sweden, Norway, Finland, Iceland, Japan, Australia, New Zealand, Singapore, most of Malaysia, Papua New Guinea, the Pacific Islands, the Indonesian island chains east of Java, and Iranian Jaya have been free of classical rabies virus for many years (Center for Food Security and Public Health 2005; OIE 2008b).

In all endemically-infected countries the virus is maintained in a population of domestic or wild carnivores, and in some European countries and Australia related lyssaviruses occur in bats (Fooks et al 2003; Swanepoel 2004; 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 footpads has been shown to prevent the virus from progressing to the brain (Swanepoel 2004). The passage of virus through the nervous system is a slow process and depending on the site of infection, the dose of virus and the animal concerned, the incubation period before the appearance of clinical signs may vary from weeks to years. In pigs the incubation period has been reported to vary from a few days to several weeks (Elvinger 2006).The occurrence of viraemia is an exceptional event, except in experimental infections of young mice with large doses of virus (Swanepoel 2004). The virus spreads to the salivary glands at about the stage that there is 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 be aggressive.

The disease in pigs lasts from a few days to a few weeks and invariably ends fatally, although it has been suggested that pigs may recover from the disease (Elvinger 2006). Typically animals become uncoordinated, may be aggressive and salivate excessively or develop a paralytic form of the disease (Swanepoel 2004). Pigs are generally dead-end hosts since they are unlikely to bite other animals or humans.

The virus is confined to the nervous tissues until the final stages of the disease when the clinical signs are dramatic and obvious. The likelihood of contamination of semen with virus collected from healthy animals which have remained healthy for 60 days after collection is negligible.

23.1.5. Hazard identification conclusion

Rabies virus is not identified as a hazard in semen collected from healthy boars housed at collection centres that have been approved by the veterinary authority of the exporting country.

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24. Ross River virus

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

Family: *Togaviridae*, Genus: *Alphavirus*, Species: *Ross River virus* (RRV) (Weaver et al 2005).

24.1.2. OIE list

Not listed.

24.1.3. New Zealand status

Exotic.

24.1.4. Epidemiology

RRV is a mosquito-borne alphavirus that occurs in Australia. It has not been reported in North America or Europe (Harley et al 2001; Russell 2002; Russell and Doggett 2006). RRV is a zoonotic virus but is not known to cause clinical disease in domestic animals.

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

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

RRV has not occurred in New Zealand. *Aedes notoscriptus*, a probable vector of Ross River virus (Russell and Doggett 2006) and *Aedes camptorhynchus*, a known vector of the virus, have become established in New Zealand (Derraik and Calisher 2004). However, *Aedes camptorhynchus* has subsequently been the subject of a successful eradication campaign[4].

[4] See: http://www.biosecurity.govt.nz/pests/southern-saltmarsh-mosquito

No records of natural infection of pigs with RRV have been found, although pigs in Taiwan have serological evidence of repeated exposure to a closely related alphavirus (Sagiyama virus). Experimental infection of pigs with Sagiyama virus is non-pathogenic and leads to a short-lived period of viraemia (Chang et al 2006). There is no evidence to suggest that Sagiyama virus is likely to be transmitted in porcine semen.

24.1.5. Hazard identification conclusion

Since there is no evidence that pigs can act as reservoirs of RRV and no evidence to suggest that transmission of alphaviruses in porcine semen should be considered likely, RRV is not identified as a hazard in the commodity.

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25. Teschovirus serotype 1

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Family: *Picornaviridae*, Genus: *Teschovirus*. Eleven serotypes of porcine Teschovirus (PTV) are recognised, PTV-1 to PTV-11. Virulent strains of PTV-1 are associated with Teschovirus encephalitis (Teschen disease) whereas less virulent strains of PTV-1 are associated with milder disease (Talfan disease, benign enzootic paresis, or poliomyelitis suum) (Zell et al 2001; Center for Food Security and Public Health 2009).

25.1.2. OIE list

Not listed.

25.1.3. New Zealand status

Enterovirus encephalomyelitis virus is exotic to New Zealand and listed as a Notifiable Organism (Tana et al 2011).

25.1.4. Epidemiology

Most of the eleven serotypes of PTV are widely distributed in pigs and cause few clinical problems. However, virulent strains of PTV-1 cause Teschovirus encephalomyelitis. Teschen disease (a fatal, nonsuppurative encephalomyelitis of pigs) was first described in the Czech republic in 1929 and later spread throughout Europe (OIE 2008). A milder form of disease (Talfan disease or poliomyelitis suum) was later recognised in Wales and Denmark (Yamada et al 2009). Clinical signs of infection with virulent strains of PTV-1 include fever, anorexia, depression and incoordination followed by hypersensitivity, paralysis and death. Less virulent strains of PTV-1 (and strains of PTV-2, -3, -4, -5, -6, -9, and -10) are associated with neurological disease in younger animals characterised by ataxia and paresis which may progress to paralysis (OIE 2008; Center for Food Security and Public Health 2009).

Teschovirus encephalomyelitis has not been reported in Western Europe since 1980. Since 1996, the disease has been reported in Belarus, Japan, Latvia, Madagascar, Moldavia, Romania, Russia, Uganda, and Ukraine (OIE 2008).

PTV enters the body via the oral route and multiplies in the gastrointestinal tract and associated lymphoid tissues before being shed in faeces and oral secretions (Center for Food Security and Public Health 2009).

Following experimental intravenous infection with PTV-1, viral antigen was detected in spinal ganglia, degenerate nerve fibres, brainstem grey matter, and ventral horn of the spinal cord as well as in bronchiolar epithelial cells, hepatocytes, tonsillar epithelium and myenteric nerve plexus of the small and large intestine. Viral antigen was only seen in the enterocytes of the duodenum and jejunum and in the tonsils following oral infection with PTV-1 (Yamada et al 2009). Histological studies on field outbreaks of disease suggest pathological lesions are limited to the brain and spinal cord (Pogranichniy et al 2003).

PTV infections only occur in swine. Other animal species are not known to be susceptible (OIE 2008).

25.1.5. Hazard identification conclusion

There is no evidence to support the suggestion that PTV-1 may be spread in the semen of infected boars. PTV-1 is not identified as a hazard in porcine semen.

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26. Torque teno virus

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

Genus: *Anellovirus*, Species: *Torque teno virus* (TTV) (Biagini et al 2005). Two species-specific genogroups have been described in pigs (TTV1 and TTV2) (Niel et al 2005).

26.1.2. OIE list

Not listed.

26.1.3. New Zealand status

Testing of a small number of samples from New Zealand pigs has not found positive animals, but no surveillance related to this virus has occurred in New Zealand (Clement 2009).

26.1.4. Epidemiology

TTV infection of pigs was first reported in the United States (Leary et al 1999) and later characterised in Japan (Okamoto et al 2002). Infection has subsequently been recognised in pigs in Canada, Spain, China, Korea, Thailand (McKeown et al 2004), and Brazil (Niel et al 2005). A study of TTV in six different countries demonstrated that approximately 66% of pigs were positive for TTV DNA, suggesting that TTV should be considered ubiquitous in swine populations (McKeown et al 2004).

Porcine TTV is not known to be associated with any swine disease (McKeown et al 2004) although experimental infection of gnotobiotic piglets resulted in mild histologic changes in the lung, liver and kidney which were not accompanied by overt clinical signs (Krakowka and Ellis 2008). There is some speculation that TTV may contribute to the expression of postweaning multisystemic wasting syndrome in porcine circovirus 2-infected pigs (Kekarainen et al 2006; Ellis et al 2008)

Vertical transmission of TTV has been described in pigs (Pozzuto et al 2009) and TTV can be found in porcine semen. 69% of TTV-seropositive boars were shown to have detectable TTV DNA in their semen using a nested PCR assay (Kekarainen et al 2007).

26.1.5. Hazard identification conclusion

TTV is ubiquitous and where surveillance for exposure to this virus has been undertaken a high seroprevalence is demonstrated. There is a high likelihood that TTV would be present in porcine semen and, in the absence of sanitary measures for TTV, it is inevitable that this virus has been introduced into New Zealand in this commodity. Given the absence of clinical disease associated with TTV infection, any claim that New Zealand should be considered free of this virus would have little credibility. TTV is not identified as a hazard in the commodity.

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27. Transmissible gastroenteritis virus

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Family: *Coronaviridae*, Genus: *Coronavirus*, Species: *Transmissible gastroenteritis virus* (TGEV) (Spaan et al 2005).

27.1.2. OIE list

Listed (OIE 2008a).

27.1.3. New Zealand status

Notifiable disease (MAF 2008).

27.1.4. Epidemiology

Transmissible gastroenteritis causes high mortality in neonatal pigs. It occurs in North America and in many European countries but not in Norway or Australia (OIE 2008b). The close relationship to PRCV has been discussed in Section 18. There is an overall nucleotide sequence homology of 96% between TGEV and PRCV, but the latter has a deletion of 621-681 nucleotides in the S gene (Saif and Sestak 2006).

When introduced into a naïve herd TGEV infects all age groups, and pigs under 7 days invariably die. Suckling pigs older than 7 days usually survive but remain stunted. Older pigs generally show inappetance and diarrhoea for a few days before recovering (Saif and Sestak 2006). Lactating sows may become very sick with signs of inappetance, vomiting, diarrhoea, and agalactia (Saif and Sestak 2006). Cats and dogs can be subclinically infected (Pensaert and Van Reeth 2004).

Pigs commonly carry the virus for about 2 weeks after infection (Pensaert and Van Reeth 2004), but chronic and/or persistent shedding of the virus for periods up to 18 months has been reported (Derbyshire et al 1969). After an outbreak of the disease some herds eliminate the virus, but in larger herds with frequent farrowings the virus may become endemic (Saif and Sestak 2006).

Transmission is usually by the faecal-oral route when susceptible pigs come in contact with infected pigs or when faeces are carried on fomites (Saif and Sestak 2006). There are no descriptions of transmission by semen or of insemination being significant in the epidemiology of the disease.

27.1.5. Hazard identification conclusion

The virus is not transmitted in semen, and therefore it is not identified as a hazard in the commodity.

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28. Rotavirus

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Rotavirus*, Species: *Rotavirus E* (Ramig et al 2005).

28.1.2. OIE list

Not listed.

28.1.3. New Zealand status

Group A rotavirus has been described in New Zealand pigs (Fu and Hampson 1987; Fu and Hampson 1989; Fu et al 1989; Fu et al 1990). However, due to the limited number of investigations reported and the confused state of the taxonomy of rotaviruses, it is uncertain if all types of rotaviruses that occur in pigs are present in New Zealand.

28.1.4. Epidemiology

Rotaviruses are generally described as occurring universally (Steele et al 2004; Yuan et al 2006) and there is considerable confusion about the naming and classification of the different species. The International Committee on the Taxonomy of Viruses recognises Rotavirus A-E with tentative species F and G. Group A, B, and C rotaviruses have been detected in pigs as well as in other animals and humans. Type E has been found only in pigs and groups D, F and G have been detected only in birds. The basis for recognition of a species is considered to be "the ability to exchange (reassort) genome segments during coinfection, thereby exchanging genetic information and generating novel progeny virus strains" (Ramig et al 2005). Rotaviruses are double-stranded RNA viruses in which reassortments occur. The basis for classifying species is more theoretical than practically useful and the taxonomy of the group is likely to be unstable. Serological characteristics are of more practical importance for classifying species. *Rotavirus* group A strains have been described in pigs in New Zealand (Fu and Hampson 1987; Fu and Hampson 1989; Fu et al 1989; Fu et al 1990). Most importantly, all the rotaviruses are enteric viruses transmitted by the faecal-oral route and have not been reported as being excreted in semen. Semen, natural breeding and artificial insemination have not been reported as being relevant to the epidemiology of the disease.

28.1.5. Hazard identification conclusion

The virus is not excreted in semen and therefore the likelihood of importing rotavirus virus in semen is negligible and it is not identified as a hazard in the commodity.

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29. Porcine reovirus

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agent

Family: *Reoviridae*, Genus: *Orthoreovirus*, Species: *Mammalian orthoreovirus* 1-4 (Chappell et al 2005).

29.1.2. OIE list

Not listed.

29.1.3. New Zealand status

Reoviruses have been isolated from poultry (Green et al 1976; Saifuddin et al 1980) and antibodies are frequently detected in poultry. Reovirus also occurs in cats. However, a retrospective electronic search of *Surveillance* and the *New Zealand Veterinary Journal* revealed no evidence that the virus has ever been found in pigs in this country.

29.1.4. Epidemiology

Reoviruses are regarded as orphan viruses occurring in the respiratory system and gut. They have been isolated from diseased and healthy pigs but experimental inoculations have not consistently caused disease (Yuan et al 2006).

29.1.5. Hazard identification conclusion

Reoviruses are not considered to be pathogenic in pigs and are not identified as a hazard in the commodity.

References

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30. Swine influenza virus

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

Family: *Orthomyxoviridae*, Genus: *Influenzavirus* A, Species: *Influenza A virus* (Kawaoka et al 2005).

30.1.2. OIE list

Avian influenza is listed in the *Code*, but swine influenza is not listed as a pig disease (OIE 2008).

30.1.3. New Zealand status

Antibody to H3N2 influenza A virus was found 88.6% of pig sera in a survey of 429 pigs. No antibodies were found to H1N1, the other strain of influenza A virus commonly found in pigs overseas (Stanislawek 2001).

30.1.4. Epidemiology

Influenza A, B, and C viruses are recognized as separate genera (Kawaoka et al 2005). Only influenza A viruses are important in pigs (Olsen et al 2006; Thompson and Easterday 2004). All influenza viruses are classified serologically according to the combination of the haemagglutinin (H) and neuraminidase (N) antigens they express. There are 16 different forms of H and 9 of N and all combinations of these are possible (Olsen et al 2006). Influenza viruses are segmented negative sense RNA viruses and therefore genetically unstable. Major reassortments of RNA segments are possible due to recombination events and smaller mutations cause antigenic drift. However, the situation in pigs has remained relatively stable and only H1N1, H3N2, and more rarely H1N2 are of known significance (Olsen et al 2006).

Because pigs are susceptible to both avian and human influenza viruses they are thought to be important hosts for the generation of recombinants that could potentially cause a pandemic in humans (Olsen et al 2006). The factors necessary for creating a new virulent strain are still only partially understood (Neumann and Kawaoka 2006).

Swine influenza is a mild disease of pigs characterised by a sudden onset of respiratory signs and an abrupt recovery about 5-7 days later (Olsen et al 2006). More severe signs can occur when infection with influenza virus is complicated by the simultaneous occurrence of other infections such as porcine respiratory and reproductive syndrome or various bacterial infections (Olsen et al 2006). However, the infection is often subclinical as evidenced by the fact that the disease has not been described in New Zealand but antibodies occur commonly in pigs.

Influenza in swine is primarily an infection of the respiratory tissues. In lung tissue the virus multiplies to a high titre (Olsen et al 2006; Thompson and Easterday 2004) but viraemia is not considered to be a feature of the disease (Zou 2006). However, it has been shown to occur irregularly in experimentally infected mice and in humans with subsequent transient infections of other organs including muscles, liver and brain (Davis et al 2000; Mori et al 1995; Tsuruoka et al 1997). No evidence could be found that the virus is transmitted in semen.

30.1.5. Hazard identification conclusion

Swine influenza is a comparatively mild infection of little economic importance. The disease has a short course or is subclinical and long-term carriers are not known. Consequently, the likelihood that semen donors resident in collection centres, where all pigs are healthy and quarantined on entry, would be infected with swine influenza virus at the time of semen donation is negligible. In addition the virus is not transmitted in semen. Therefore the virus is not identified as a hazard in the commodity.

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31. Swine pox virus

31.1. HAZARD IDENTIFICATION

31.1.1. Aetiological agent

Family: *Poxviridae*, Genus: *Suipoxvirus*, Species: *Swinepox virus* (Buller et al 2005).

31.1.2. OIE list

Not listed.

31.1.3. New Zealand status

Classified as an "other exotic organism" (MAF 2008).

31.1.4. Epidemiology

Information in this section is taken from two authoritative reviews (Delhon and Tulman 2006; Munz and Dumbell 2004).

Swine pox virus is widely distributed in the world and causes a mild disease of little economic significance. The disease is transmitted by contact between acutely infected pigs or by lice or biting flies which act as mechanical vectors. Lice remain infected for weeks or months. The incubation period of the disease is from 4-14 days and the virus may persist in dried scabs for months. Toward the end of the incubation period virus is also shed from saliva and lachrymal fluid. A viraemic state is postulated but has not been demonstrated. Pox lesions develop especially on the abdomen and thin skin regions. The lesions scab over and then heal in about three weeks. Diagnosis is usually made from the typical signs and lesions, and virus can be confirmed by isolation or electron microscopy. Antibody development is often poor or absent and serological tests are unreliable.

There is no evidence that pigs are long-term carriers of virus and there is no evidence that the virus can be transmitted in semen.

31.1.5. Hazard identification conclusion

The signs of infection are obvious and long-term carriers do not occur. Consequently, the likelihood that boars that have been quarantined before introduction to a semen collection centre will be infected at the time of collection is negligible. In addition the virus is not transmitted in semen. Therefore, the virus is not identified as a hazard in the commodity.

References

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32. Swine vesicular disease virus

32.1. HAZARD IDENTFICATION

32.1.1. Aetiological agent

Family: *Picornaviridae*, Genus: *Enterovirus*, Species: *Swine vesicular disease vir*us (SVDV). SVDV is considered to be a porcine variant of *Human coxsackievirus B5* (Stanway et al 2005).

32.1.2. OIE list

Listed (OIE 2008a).

32.1.3. New Zealand status

Exotic notifiable disease (OIE 2008b).

32.1.4. Epidemiology

Swine vesicular disease (SVD) first emerged in Italy in 1966 (Nardelli et al 1968) and was subsequently diagnosed throughout Europe and Asia (Lubroth et al 2006).

SVDV is exceptionally stable outside the host and indirect contacts such as transport vehicles or waste feeding play an important role in the spread of disease (Hedger and Mann 1989). Experimental inoculation or exposure to a contaminated environment is followed by the rapid development of viraemia (Dekker et al 1995).

Viral titres are greatest in the myocardium and brain of infected individuals, suggesting these are the most likely sites of viral replication (Chu et al 1979; Lai et al 1979). Experimental inoculation studies have demonstrated high viral titres in lymph nodes (Dekker et al 1995). SVDV has been recovered from the semen of infected boars up to 4 days post infection, although artificial insemination using infected semen failed to transmit disease to sows (Maes et al 2008).

32.1.5. Hazard identification conclusion

SVDV can be recovered from the semen of infected boars. SVDV is therefore identified as a potential hazard in semen imported from infected countries.

32.2. RISK ASSESSMENT

32.2.1. Entry assessment

SVDV has been reported in a number of European countries and is currently recognised in Italy (OIE 2008b; Sabirovic et al 2009; Sabirovic et al 2010a; Sabirovic et al 2010b). As SVDV can be found in the semen of infected pigs (Maes et al 2008), the likelihood of entry is assessed to be non-negligible.

32.2.2. Exposure assessment

Imported semen will be inseminated into New Zealand pigs. Maes et al (2008) noted that artificial insemination using SVDV-infected semen failed to transmit disease to sows. Similarly, van Rijn et al (2004) were unable to isolate virus directly from the semen of boars artificially infected with SVDV intravenously although virus isolation carried out following the blind passage of semen samples in cell culture did detect SVDV. PCR testing of semen from

artificially infected boars gave weak positive results, suggesting low numbers of SVDV RNA (van Rijn et al 2004).

The likelihood of exposure is very low.

32.2.3. Consequence assessment

Reflecting the results of van Rijn et al (2004) described above, there is a very low likelihood that insemination of pigs would result in the transmission of SVDV to the recipients of the semen.

Infection of pigs with SVDV results in vesicular lesions whose severity may be heavily influenced by environmental factors (Hedger and Mann 1989). These lesions may be accompanied by fever, inappetance and general malaise. SVDV infection may be clinically indistinguishable from foot and mouth disease so an initial presentation of clinical SVD (before laboratory confirmation of the viral aetiology) would be likely to trigger the massive initial response usually reserved for foot and mouth disease.

The consequences of exposure are assessed to be non-negligible.

32.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for SVDV is non-negligible and it is classified as a risk in the commodity. Therefore risk management measures can be justified.

32.3. RISK MANAGEMENT

32.3.1. Options

The *Code* defines SVD-free countries, and SVD-infected zones. The *Code* chapter on SVD contains the following recommendations relating to importation of pig semen:

> **Article 15.4.9.** Recommendations for importation from SVD free countries
>
> Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:
>
> 1. the donor animals:
>
> a. showed no clinical sign of SVD on the day of collection of the semen;
> b. were kept in an SVD free country for not less than 6 weeks prior to collection;
>
> 2. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.
>
> **Article 15.4.10.** Recommendations for importation from countries considered infected with SVD
>
> Veterinary Authorities should require the presentation of an international veterinary certificate attesting that:

1. the donor animals:

    a. showed no clinical sign of SVD on the day of collection of the semen and were subjected to the virus neutralisation test for SVD with negative results;

    b. were kept in the exporting country for the 28 days prior to collection, in an establishment or artificial insemination centre where no case of SVD was officially reported during that period, and that the establishment or artificial insemination centre was not situated in an SVD infected zone.

2. the semen was collected, processed and stored in conformity with the provisions of Chapter 4.5. and Chapter 4.6.

Chapters 4.5 and 4.6 of the *Code* (OIE 2009a) contain requirements for SVD for boars standing at semen collection centres. Under these requirements, prior to entry into isolation at the quarantine station of the semen collection facility, boars must be clinically healthy, physiologically normal, and either kept in a SVD-free country since birth or for the past six weeks, or kept since birth or for the past six weeks in an establishment outside an SVD infected zone where no case of SVD was officially reported during that period and been subject to a virus neutralisation test for SVD with negative results. Boars must remain in this quarantine station for at least 28 days and, at least 21 days after entering the quarantine station must be tested negative for SVD as per Articles 15.4.9 or 15.4.10 of the *Code*. In countries, zones or compartments which are not free of SVD, any boars resident in the semen collection facility must also be tested at least annually as per Articles 15.4.9 or 15.4.10 of the *Code*.

Available options for exclusion of SVD are:

Option 1

Semen originating from donor boars that have lived their entire lives in countries free from SVD could be imported without further sanitary measures.

Option 2

Semen originating from donor boars from countries considered infected with SVD could be required to comply with the recommendations contained in 15.4.10 of the OIE *Code*, including compliance with Chapters 4.5 and 4.6 of the *Code*.

Option 3

Every batch of semen to be imported could be tested by PCR as described by van Rijn et al (2004). A positive test on any batch of semen could result in disqualification of that semen.

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33. Venezuelan encephalitis virus

33.1. HAZARD IDENTIFICATION

33.1.1. Aetiological agent

Family: *Togaviridae*, Genus: *Alphavirus* (King et al 2012). Six subtypes of Venezuelan equine encephalomyelitis (VEE) virus are recognised (OIE 2008).

33.1.2. OIE list

Venezuelan equine encephalomyelitis is an OIE-listed disease.

33.1.3. New Zealand status

Equine encephalitis viruses (including VEE virus) are exotic to New Zealand and listed as Notifiable Organisms (Tana et al 2011).

33.1.4. Epidemiology

Venezuelan equine encephalomyelitis is an arthropod-borne inflammatory viral infection of equines and humans that results in mild to severe febrile and, occasionally fatal, encephalitic disease. Horses serve as an amplifying host for epizootic strains of VEE while enzootic VEE viruses cycle primarily between sylvatic rodents and mosquitoes (OIE 2008).

Bowen (1976) summarised experimental studies using VEE virus and concluded that pigs could be eliminated as important epizootic hosts because of their low susceptibility to infection or low viraemias. There are a few reports of serological evidence of VEE in pigs but isolation of virus has not been reported in this species. It appears that pigs are not significant in the epidemiology of the disease because they do not produce a significant viraemia (AQIS 2000).

The pig is generally regarded as a dead-end host, and there is no evidence suggesting that pigs might transmit the virus to mosquitoes. Overall, the likelihood of the VEE virus being present in the semen of a donor boar and being transmitted to a recipient sow was assessed by AQIS (2000) to be negligible.

33.1.5. Hazard identification conclusion

Consistent with the findings of AQIS (2000), VEE virus is not identified as a hazard in porcine semen.

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34. Vesicular exanthema virus

34.1. HAZARD IDENTIFICATION

34.1.1. Aetiological agent

Family: *Caliciviridae*, Genus: *Vesivirus*. Vesicular exanthema of swine virus (VESV) and vesiviruses isolated from marine species are phylogenetically grouped together as "marine vesiviruses" (King et al 2012).

34.1.2. OIE list

Not listed.

34.1.3. New Zealand status

Vesicular exanthema has never been decribed in New Zealand.

34.1.4. Epidemiology

The primary reservoir hosts of marine vesiviruses are thought to be certain fish species which can pass on infections to secondary hosts such as seals or pigs. Vesicular exanthema was first described in pigs in the United States in 1932 and outbreaks continued until it was eradicated in 1956. Only one outbreak has been described outside of the United States (in Iceland, associated with pigs fed raw garbage from a United States military post) (Anon 1988).

Vesicular exanthema is clinically indistinguishable from either foot and mouth disease or vesicular stomatitis. Infected pigs present with vesicles on the snout, lips, tongue, and mucosae of the oral cavity and on the sole, Interdigital spaces, and coronary band of the foot. Lesions directly attributable to VESV, other than vesicle formation, have not been described (Madin and Traum 1955).

VESV is principally transmitted between pigs by direct contact when ruptured vesicles release large amounts of infective virus into the environment. Transmission of virus associated with feeding waste (garbage) food to pigs is also recognised (Madin and Traum 1955). No references could be found that described the presence of VSEV in boar semen and VSEV is not listed in recent reviews of pathogens likely to be found in boar semen (Guérin and Pozzi 2005; Maes et al 2008).

34.1.5. Hazard identification conclusion

Vesicular exanthema has not been reported since 1956 and there is no evidence to support the suggestion that VSEV may be spread in the semen of infected boars. VSEV is not identified as a hazard in porcine semen.

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35. Vesicular stomatitis virus

35.1. HAZARD IDENTFICATION

35.1.1. Aetiological agent

Family: *Rhabdoviridae*, Genus: *Vesiculovirus*, Species: *Vesicular stomatitis Alagoas virus*, *Vesicular stomatitis Indiana virus*, *Vesicular stomatitis New Jersey virus* (Tordo et al 2005). Many authors regard the three species of virus as serotypes of the same species.

35.1.2. OIE list

Listed (OIE 2008).

35.1.3. New Zealand status

Exotic notifiable disease (MAF 2008).

35.1.4. Epidemiology

Vesicular stomatitis is primarily an insect-borne virus but it can also be transmitted by contact (Lubroth et al 2006). Outbreaks of disease occur sporadically in the USA and are always associated with insect transmission (Lubroth et al 2006; Rodriguez 2002; Rodriguez et al 1996). The virus is found in epithelial tissues of the mouth, nose, coronary region of the hooves, teats and lymph nodes (Lubroth et al 2006). It is not found in blood (Lubroth et al 2006). There are no references to it being excreted in semen.

35.1.5. Hazard identification conclusion

Since the virus is an arbovirus and is not excreted in semen it is not identified as a hazard in the commodity.

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36. West Nile virus

36.1. HAZARD IDENTIFICATION

36.1.1. Aetiological agent

Family: *Flaviviridae*; Genus: *Flavivirus*, Species: *West Nile virus* (WNV) (Thiel et al 2005).

36.1.2. OIE list

Listed.

36.1.3. New Zealand status

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

36.1.4. Epidemiology

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

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

Experimental inoculation studies of pigs using mosquitoes infected with WNV have shown that adult pigs are very unlikely to show clinical disease when infected with this virus and rarely develop a very low level transient viraemia. Weanling pigs are more likely to develop viraemia following infection but this is also of short duration and insufficient to infect mosquitoes. Pigs are therefore considered unlikely to serve as amplifying hosts of WNV (Teehee et al 2005).

36.1.5. Hazard identification conclusion

Since pigs are dead-end hosts for WNV, the likelihood that virus would be present in imported semen is assessed to be negligible. Therefore, the organism is not identified as a hazard in the commodity.

References

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37. Western equine encephalitis virus

37.1. HAZARD IDENTIFICATION

37.1.1. Aetiological agent

Family: *Togaviridae*; Genus: *Alphavirus*, Species: Western equine encephalitis virus (WEEV). (Weaver et al 2005).

37.1.2. OIE list

Listed.

37.1.3. New Zealand status

Exotic notifiable disease (MAF 2008).

37.1.4. Epidemiology

WEEV is found predominantly in the western United States and Canada (Radostits et al 2007) and is normally maintained in a host-vector relationship by cycling between mosquitoes (*Culex tarsalis* in the western United States and *Culiseta melanura* in the eastern and southern United States) and the definitive host (wild birds, especially passerines) (Hayes and Wallis 1977; Iversen 1994). Pigs are an accidental host of the virus (Radostits et al 2007).

Although surveys have described evidence of exposure to WEEV in pigs (Scherer et al 1966), there is no evidence that would suggest this species should be considered anything other than a dead-end host. An extensive literature search has found no evidence of clinical disease in pigs associated with WEEV, nor any suggestion that this virus is likely to be found in the semen of infected pigs.

37.1.5. Hazard identification conclusion

Since pigs are dead-end hosts for WEEV, the likelihood that virus would be present in imported semen is considered to be negligible. Therefore, the organism is not identified as a hazard in the commodity.

References

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38. Bacillus anthracis

38.1. HAZARD IDENTFICATION

38.1.1. Aetiological agent

Bacillus anthracis.

38.1.2. OIE list

Listed (OIE 2008).

38.1.3. New Zealand status

Exotic notifiable disease (MAF 2008).

38.1.4. Epidemiology

Anthrax is generally an acute bacterial infection of many species including pigs. Pigs are more resistant to infection than ruminants (Taylor 2006). Animals are infected by the oral route with spores which occur in the soil, but infected animals are not contagious (Taylor 2006). Bacilli sporulate when blood from infected animal carcasses is exposed to air, and spores can remain viable in soil for many years (De Vos and Turnbull 2004; Turner et al 1999a; Turner et al 1999b). Anthrax may present as the pharyngeal, intestinal, or septicaemic form in pigs. The first two syndromes may be mild and animals may recover spontaneously. The septicaemic form is usually an acute or peracute infection and death may occur without any signs being observed (Taylor 2006).

Diagnosis is made by identification of the organism in blood smears taken from dead animals, or if the carcass has been opened from lymph nodes, spleen or kidney. The organism can be cultured from blood or tissues.

There are no reports indicating that the organism is excreted in semen of healthy animals, and the *Code* gives no recommendations for trade in semen, thereby indicating that no trade restrictions are required.

38.1.5. Hazard identification conclusion

The likelihood that a healthy boar housed at a semen collection centre would be excreting *Bacillus anthracis* spores in its semen is negligible. Therefore *Bacillus anthracis* is not identified as a hazard in the commodity.

References

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39. Brucella suis

39.1. HAZARD IDENTFICATION

39.1.1. Aetiological agent

Brucella suis.

39.1.2. OIE list

Listed (OIE 2008a).

39.1.3. New Zealand status

Exotic, notifiable organism (MAF 2008).

39.1.4. Epidemiology

Brucellosis is a venereal disease in pigs, with sows readily infected when mated with infected boars or inseminated with semen containing *B. suis* (MacMillan et al 2006). Infected boars may shed 10^4 to 10^5 CFU of *B. suis* per ml of semen (Lord et al 1998). Infection of semen cannot be eliminated by using antibiotics, as the amount of antimicrobial required may be incompatible with semen survival (Algers et al 2009)

Apart from domestic pigs, reservoirs of *B. suis* infection are recognised in European hares (Bendtsen et al 1954) and feral pigs (Drew et al 1992). Transmission of infection from wild boars to domestic pigs is thought to be through the venereal route. Waste feeding of hare offal has also been implicated as a method of introduction (Algers et al 2009).

Bacteraemia follows 1 to 7 weeks after exposure to *B. suis* and persists for an average of about 5 weeks, although intermittent bacteraemia lasting as long as 34 months has been decribed. Bacteraemia may precede the development of a detectable serological response by as much as 6 to 8 weeks (MacMillan et al 2006).

Infection with *B. suis* is associated with reproductive failure characterised by abortion, stillbirth, infertility, and testicular lesions. *B. suis* localises to the placenta of pregnant sows, leading to placentitis with subsequent malnutrition and hypoxia, and abortion in mid-to-late pregnancy (Algers et al 2009). However, the clinical evidence of infection varies considerably between herds and clinical signs may be only transient or absent. Death is rare (MacMillan et al 2006).

Porcine brucellosis is a significant public health concern, with occupational *B. suis* infections described in farmers, veterinarians, abattoir workers, and individuals who have direct contact with feral swine (MacMillan et al 2006).

39.1.5. Hazard identification conclusion

Following infection, *B. suis* is commonly found in porcine semen and is therefore identified as a potential hazard.

39.2. RISK ASSESSMENT

39.2.1. Entry assessment

B. suis has never been recognised in Norway. The disease may be present in the other countries although it appears to be restricted to wildlife in the United States, Canada, Australia, and some EU member states (OIE 2008b; 2008c). Pigs originating from those countries where *B. suis* is present could be subclinically infected with *B. suis* and could excrete the organism in their semen. The likelihood for entry of *B. suis* in pig semen from these countries is therefore non-negligible.

39.2.2. Exposure assessment

Imported semen will be inseminated into New Zealand pigs. Sows are readily infected when mated with infected boars or inseminated with semen containing *B. suis* (MacMillan et al 2006). The likelihood of exposure of New Zealand pigs is assessed to be non-negligible.

39.2.3. Consequence assessment

Insemination of pigs would result in the transmission of *B. suis* to the recipients of the semen and the establishment of infection. Although affected herds may have no clinical signs of brucellosis, reproductive losses, infertility, paralysis and lameness may be seen as a result of infection. Once introduced into the environment, *B. suis* can persist in organic matter at near-freezing temperatures for over 2 years (MacMillan et al 2006), and subsequent spread to other domestic herds would be likely. Infection may also become established in New Zealand's feral pig population.

Occupational exposure may result in infection of farm staff, abattoir workers, and veterinarians.

The consequences of introduction are assessed to be non-negligible.

39.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for *B. suis* is non-negligible and it is assessed to be a risk in the commodity. Therefore risk management measures can be justified.

39.3. RISK MANAGEMENT

39.3.1. Options

The *Code* chapter on porcine brucellosis contains the following recommendations relating to importation of pig semen:

> **Article 15.3.5.** Recommendations for the importation of semen of pigs
>
> Veterinary Authorities of importing countries should require the presentation of an international veterinary certificate attesting that:
>
> 1. the donor animals showed no clinical sign of porcine brucellosis on the day of collection of the semen;
> 2. the donor animals were kept in a herd free from porcine brucellosis;

3. the donor animals were subjected to a diagnostic test for porcine brucellosis with negative results during the 30 days prior to collection;
4. the semen does not contain Brucella agglutinins;
5. the donor animals were kept in the exporting country, for the 60 days prior to collection, in an establishment or artificial insemination centre where the herd is free from porcine brucellosis;
6. the semen was collected, processed and stored in conformity with the provisions of chapters 4.5. and 4.6.

Chapters 4.5 and 4.6 of the *Code* (OIE 2009a) contain requirements for *B. suis* for boars standing at semen collection centres. Under these requirements, prior to entry into isolation at the quarantine station of the semen collection facility, boars must be clinically healthy, physiologically normal, kept in a herd free of porcine brucellosis and subject to a diagnostic test for porcine brucellosis with negative results. Boars must remain in this quarantine station for at least 28 days and, at least 21 days after entering the quarantine station, must be tested negative for *B. suis* as per Articles 15.3.5 of the *Code*. In countries, zones or compartments which are not free of porcine brucellosis, any boars resident in the semen collection facility must also be tested at least annually as per Articles 15.3.5 of the *Code*.

The OIE Manual (Olsen 2009) describes the indirect and competitive ELISAs, as well as the Rose Bengal test, complement fixation test, and fluorescence polarisation assay as suitable serological tests for international trade. None of the serological tests described are considered reliable for diagnosis in individual pigs, although all of these tests are sensitive enough to be used as screening tests to detect an infected herd (Algers et al 2009). For international trade, the disease status of the herd and of the area in which the herd is situated are of more importance than tests on individual animals (Olsen 2009).

Available options for exclusion of *B. suis* are:

Option 1

The requirements of Article 15.3.5. (including compliance with Chapters 4.5 and 4.6) would ensure that imported semen was free from *B. suis*.

Option 2

Semen from donor boars that have lived their entire lives in countries that are free from *B. suis* could be imported without restrictions.

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40. Burkholderia pseudomallei

40.1. HAZARD IDENTIFICATION

40.1.1. Aetiological agent

Burkholderia pseudomallei.

40.1.2. OIE list

Not listed.

40.1.3. New Zealand status

Listed as an exotic unwanted organism (MAF 2008).

40.1.4. Epidemiology

Burkholderia pseudomallei is the cause of melioidosis, a disease of humans and animals. It occurs predominantly in the tropical and subtropical regions of Asia, northern Australia and in some foci in Africa (Groves and Harrington 1994; Inglis 2004; Inglis et al 2004), and was recorded over 20 years ago in a piggery in southeastern Queensland (Ketterer et al 1986). It occasionally spills over into temperate regions and an imported case has occurred in New Zealand (Corkill and Cornere 1987). The aetiological agent occurs in the environment and is widely distributed in water and soil (Sprague and Neubauer 2004). It has been transmitted to animals via oral mucosa, nasal mucosa, ingestion, parental inoculation, and skin scarification (Groves and Harrington 1994). Infection in natural cases is probably by contact with infected water and mud, especially through abrasions and wounds. Water was implicated as a possible source of infection in six locations in one study (Inglis et al 2004). The disease has been described in pigs (Taylor 2006; Thomas 1981). Many cases are subclinical, but fever and occasional deaths may occur. The infection is usually discovered at post-mortem when abscesses are found in the internal organs and lymph nodes.

There is no evidence that the infection is transmitted in pigs by semen. Theoretically the organism could be excreted in semen during the bacteraemic phase of the disease. However, this has not been described and it is considered extremely unlikely to occur in a healthy pig used as a semen donor.

40.1.5. Hazard identification conclusion

Burkholderia pseudomallei is an organism found in the environment in tropical and subtropical areas, but has not established in temperate climates. It appears to be an opportunistic pathogen and there are no descriptions of it being transmitted by pig semen. For these reasons it is not identified as a hazard in the commodity.

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41. Clostridium botulinum

41.1. HAZARD IDENTFICATION

41.1.1. Aetiological agent

Clostridium botulinum. Types A, B, Cα, Cβ, D E, F, and G have been identified (Kriek and Odendaal 2004).

41.1.2. OIE list

Not listed.

41.1.3. New Zealand status

Type C botulism has been identified in New Zealand (Fairley 1997; Gardner 1990; Gardner 1992). It is not clear whether other types occur in New Zealand.

41.1.4. Epidemiology

Animals are poisoned when they eat decaying meat, bones, vegetable matter or other food items that are contaminated with botulinum toxin (Cobb et al 2002; Kriek and Odendaal 2004; Songer and Taylor 2006). Therefore botulism is a toxicosis, not an infectious disease, except in rare cases of toxicoinfectious botulism where the organism multiplies in the gut or in wounds (Kriek and Odendaal 2004). Affected animals are not infectious. There are no reports of *C. botulinum* or its toxin being excreted in semen. Pigs are highly resistant to botulism and the disease is rarely reported in them (Songer and Taylor 2006).

41.1.5. Hazard identification conclusion

C. botulinum is an environmental contaminant and not a disease agent transmitted by animals. Therefore, it is not identified as a hazard in the commodity.

References

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42. Other *Clostridium* spp.

42.1. HAZARD IDENTIFICATION

42.1.1. Aetiological agents

The following clostridial species are associated with syndromes occurring in swine (Songer and Taylor 2006):

Clostridium perfringens type A
C. perfringens type C
C. difficile
C. tetani
C. novyi
C. septicum
C. chauvoei

42.1.2. OIE list

Not listed.

42.1.3. New Zealand status

The following have been identified as occurring in New Zealand (Gardner 1990):

C. tetani
C. botulinum
C. difficile
C. chauvoei
C. septicum
C. oedematiens (*novyi*)
C. perfringens (types A and D)

C. haemolyticum (a synonym for *C. novyi* type D) (Anonymous 1979) and more recently *C. sordellii* (Simpson 1999) have also been isolated.

42.1.4. Epidemiology

Clostridia are generally regarded as being universally distributed, occurring as vegetative organisms or as spores in soil and the environment. Of the organisms identified as being important in pigs, all are already present in New Zealand, except possibly *C. perfringens* type C. The disease syndrome in young pigs caused by *C. perfringens* type C, has also not been described in New Zealand (Fairley 1997). Therefore only *C. perfringens* type C is considered in this section.

The peracute and acute forms of the disease in young pigs are characterised by haemorrhagic diarrhoea and death within 12-48 hours. At post mortem there is necrohaemorrhagic enteritis (Songer and Taylor 2006). The organism is probably carried in the gut of infected sows where it occurs in low numbers and is difficult to identify, as it is massively outnumbered by the total population of *C. perfringens* of other types (Songer and Taylor 2006). The lethal necrotising beta

toxin of *C. perfringens* type C is the primary factor involved in the pathogenesis of the disease, but other factors may also be involved (Songer and Taylor 2006). The organism is transmitted by the oral route and there is nothing in the literature to suggest that it is transmitted by semen. Indeed, if any animal were to be septicaemically infected by the organism it would be clearly sick and obviously unsuitable for semen donation.

42.1.5. Hazard identification conclusion

Since the organism is not transmitted in semen it is not identified as a hazard in the commodity.

References

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43. Leptospira spp.

43.1. HAZARD IDENTIFICATION

43.1.1. Aetiological agent

The species *Leptospira interrogans* contains over 200 *Leptospira* serovars classified into 23 serogroups (Bolin 2004). A newer and alternative taxonomic scheme based on genomic characteristics classifies the pathogenic organisms into eight species, and the species are subdivided into serovars. For the purposes of this risk analysis the older method of classifying leptospires as serovars of *Leptospira interrogans* is used and serovars are written as if they were single species, e.g., *Leptospira hardjo, L. pomona,* etc.

43.1.2. OIE list

Although leptospirosis is listed by the OIE, from 2004 to 2009 the *Code* chapter did not contain recommendations for leptospirosis, only a statement that it was "under study". At the OIE General Session in May 2009, the International Committee accepted the recommendation of the Terrestrial Animal Health Standards Commission that the empty *Code* chapter on leptospirosis should be deleted from the *Code*.

43.1.3. New Zealand status

L. hardjo, L. pomona, L. balcanica, L. copenhageni, *L. ballum* and *L. tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). A single isolation of *L. australis* has been reported from a human (Thompson 1980). Serological diagnosis indicates that five of the species that have been found in farm animals can also infect humans, but *L. balcanica* which is associated with possums has not been diagnosed in humans (ESR 2004). Other *Leptospira* spp. are classified by MAF as "other exotic organisms" (MAF 2008).

L. pomona and *L. tarassovi* have been identified in pigs in New Zealand (Fairley 1997).

43.1.4. Epidemiology

Leptospira spp. occur throughout the world, but it is not possible to accurately define which serovars occur in each country. Exotic *Leptospira* spp. that have been found in pigs include *L. bratislava, L. canicola, L. icterohaemorrhagiae, L. sejroe* and *L. grippotyphosa.*

Leptospirosis is not a single disease but a complex of diseases caused by many different leptospires. Most serovars are adapted to a particular host species in which they may exist for long periods without causing disease signs. Species other than the maintenance host may be more resistant to infection, but if infected are more susceptible to disease. *L. pomona,* for example, causes subclinical infection in pig herds but may be responsible for causing sporadic cases of disease in other species such as humans (accidental hosts). In maintenance hosts, *Leptospira* localise in the kidneys and continue to be excreted in urine for protracted periods.

The organisms are shed in urine and infection can occur through mucous membranes, venereally, by mouth or through the skin, particularly through abrasions and wounds (Hunter 2004). *Leptospira* spp. are also excreted in semen and remain viable in semen prepared for artificial insemination (Hunter 2004; Masri et al 1997). Clinically diseased animals shed more organisms and are more important sources of infection than chronic carriers (Horsch 1989).

In accidental hosts the incubation period ranges from 2-16 days and is followed by a period of bacteraemia (Hunter 2004). Most cases of leptospirosis in pigs are subclinical. A variety of signs may be shown by diseased animals including abortion, haemolytic anaemia, icterus and nephritis. Chronic leptospirosis is characterised by abortions, stillbirths and the birth of weak piglets. The disease can be diagnosed by the isolation of the organism, but because this is a difficult process it is usually diagnosed by serological methods. A rising titre suggests a recent infection while a stable, often low-level titre indicates resolution or a chronic infection. The microscopic agglutination test is still the most commonly used herd test. A number of variations of ELISA are also available, but generally lack serovar specificity (Bolin 2004). Leptospirosis is seldom the cause of economically serious disease in animals. It is a zoonotic disease that occasionally causes serious disease in humans (Thornley et al 2002).

Leptospira spp. are sensitive to several antibiotics (Alt et al 2001; Gerritsen et al 1994; Gerritsen et al 1993; Hodges et al 1979; Ministry of Agriculture and Forestry 2007; Murray and Hospenthal 2004; Oie et al 1983). Leptospiruria has been successfully treated using streptomycin (Alt et al 2001; Gerritsen et al 1994; Hodges et al 1979). Streptomycin and penicillin have been used extensively for prophylaxis and treatment of live animals, semen and embryos in international trade. Streptomycin was found to be the drug of choice in pigs and tetracycline was also effective (Alt and Bolin 1996).

43.1.5. Hazard identification conclusion

Since *Leptospira* spp. exotic to New Zealand occur in the countries included in this risk analysis, they are identified as a potential hazard.

43.2. RISK ASSESSMENT

43.2.1. Entry assessment

Acutely infected animals or chronic carriers of infection may excrete the organism in their semen (Hunter 2004; Masri et al 1997). Therefore the likelihood of entry in imported semen is non-negligible.

43.2.2. Exposure assessment

Carriers and acutely infected animals shed the organism in their semen and inseminated sows are likely to become infected. Therefore the likelihood of exposure of sows inseminated with imported semen is high.

43.2.3. Consequence assessment

Introduction of new *Leptospira* serovars is unlikely to have a major impact on the New Zealand pig population or other farmed animals although cases of disease and reproductive failure may occur.

The establishment of a new *Leptospira* serovar to which humans are susceptible could lead to the sporadic occurrence of leptospirosis in humans. The number and severity of the cases would depend on the serovars involved and contact with infected animals. Some serovars are not important as human pathogens. For example, in New Zealand *L. balcanica* is common in its maintenance host the brush-tailed possum, but infections of humans have not occurred despite close contact between possums and possum hunters (ESR 2004).

There are not likely to be noticeable consequences for feral or wild animals, but some serovars such as *L. grippotyphosa*, *L. canicola*, *L. sejroe* and *L. saxkoebing* could become established in mice and rats (Horsch 1989) and subsequently infect humans.

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. Cases of disease and reproductive failure may occur in pigs.

43.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for *Leptospira* spp. is non-negligible and they are classified as a risk in the commodity. Therefore risk management measures can be justified.

43.3. RISK MANAGEMENT

43.3.1. Options

When drafting options for managing the risk posed by *Leptospira* spp. in semen, the following points were considered:

- Because of the occurrence of long-term carriers of infection, quarantine is not a suitable option.
- Diagnosis in donor boars 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 cross reactions and low-titre reactions.
- Testing of semen samples by culture or PCR is problematic because isolation of organisms is difficult and selection of primers for PCR that will recognise all serovars has not yet been achieved.
- *Leptospira* are sensitive to a variety of antibiotics, and treatment of animals or inclusion of antibiotics in prepared semen has traditionally been used to prevent dissemination of *Leptospira* spp. by international trade.
- The OIE Terrestrial Animal Health Standards Commission considers that international trade does not increase the risks to human or animal health. The *Code* gives no recommendations for leptospirosis.

Option 1

The disease could be considered to be of negligible risk to human or animal health, and trade without restrictions could be permitted.

Option 2

Donor boars could be tested serologically with a variety of antigens that occur in the exporting country and not in New Zealand, with negative results.

Option 3

Donor boars could be treated with effective antibiotics within one week of semen collection. Streptomycin is the antibiotic of choice although it may not be available in all countries.

Option 4

Semen diluents containing antibiotics that are effective against *Leptospira* spp. could be used in the preparation of the semen.

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44. Mycoplasma spp.

44.1. HAZARD IDENTIFICATION

44.1.1. Aetiological agent

Mycoplasma hyopneumoniae, *M. hyorhinis*, and *M. hyosynoviae* are recognized pathogens of pigs. Other *Mycoplasma* spp. and related Mollicutes are regarded as non-pathogenic (Thacker 2006).

44.1.2. OIE list

Not listed.

44.1.3. New Zealand status

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

44.1.4. Epidemiology

M. hyopneumoniae, *M. hyorhinis*, and *M. hyosynoviae* are widely distributed in the world (Thacker 2006). Of these, only *M. hyosynoviae* has not been described in New Zealand. Since there have been no restrictions in the past to prevent the introduction of this organism, it seems unlikely that it is not already here. It commonly occurs along with *M. hyorhinis*, which is faster growing and easier to culture. Unless a culture medium that contains inhibitors for *M. hyorhinis* is used, *M. hyosynoviae* may be overlooked (Friis 1979; Friis et al 1991).

M. hyosynoviae colonises the upper respiratory tract. In the acute phase of infection, which lasts 1-2 weeks, it spreads systemically and infects joints and other tissues. Infection can persist indefinitely in the tonsils (Friis et al 1991; Hagedorn-Olsen et al 1999a; Hagedorn-Olsen et al 1999b; Thacker 2006).

Signs of infection with *M. hyosynoviae* occur after an incubation period of 4-9 days and include lameness in 3-5 month old pigs, with acute signs persisting for 3-10 days. Many animals recover with no further lameness. In some animals stiffness or arthritis may persist and may be complicated by osteochondrosis (Thacker 2006). The diagnosis can be confirmed by isolation of the organism from joint fluid during the acute phase, or antibody can be detected by complement fixation or ELISA. These tests are not generally available in the USA (Thacker 2006), and are unlikely to be available in New Zealand. In some herds no signs of disease are seen and infection is confined to the tonsils (Hagedorn-Olsen et al 1999b).

The available evidence suggests that the organism persists in tonsils and synovial surfaces. The organism spreads systemically during the early stages of infection, indicating bacteraemia. However, a search of the literature including three electronic databases produced no evidence that the organism is excreted in semen or that semen or artificial insemination is implicated in the transmission of the disease.

44.1.5. Hazard identification conclusion

M. hyosynoviae has not been described as occurring in New Zealand, but surveillance has not been conducted in a systematic way. There is no evidence to suggest that the organism is excreted in semen and therefore it is not identified as a hazard in the commodity.

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45. Pasteurella multocida

45.1. HAZARD IDENTIFICATION

45.1.1. Aetiological agent

Pasteurella multocida strains that produce a dermonecrotic protein toxin (Penrith 2004).

45.1.2. OIE list

Listed (OIE 2008a).

45.1.3. New Zealand status

Notifiable organism (MAF 2008).

45.1.4. Epidemiology

Progressive atrophic rhinitis occurs in all countries that are considered in this risk analysis (OIE 2008b). The disease is caused by toxigenic strains of *P. multocida* and is most commonly seen in young pigs about 4-12 weeks old, but older pigs can also be infected. The first signs are those of acute rhinitis with snuffling and sneezing. As the disease progresses, snout deformities are seen and retardation of growth rate is common (De Jong 2006; Penrith 2004). In some cases pigs may be subclinically infected. The organism may be found in the nasal passages and it usually colonises the tonsils (De Jong 2006; Penrith 2004). Colonisation of the nasal mucosa causes the most severe lesions, but lesions can develop as a result of toxin produced in the tonsils without colonisation of the nasal passages (De Jong 2006; Penrith 2004). The organism may be isolated from tonsils, nasal passages and lungs, but has also been isolated from the vagina (De Jong 2006).

An extensive review of the literature revealed no description of the isolation of the organism from the male genital tract or of excretion of the organism in semen. The Australian Quarantine and Inspection Service (AQIS) found no information on excretion of *P. multocida* in semen (AQIS 1999). Further, in reply to a question about their draft import risk analysis, AQIS said "transmission of *P. multocida* via artificial insemination is considered a very unlikely event" (AQIS 2000). Therefore, it is concluded that the organism is not excreted in semen. In addition, the organism is sensitive to a wide range of antibiotics including those that are used in semen diluents (Penrith 2004) and would likely be inactivated in semen prepared for artificial insemination.

45.1.5. Hazard identification conclusion

P. multocida is not excreted in semen and is not identified as a hazard in the commodity.

References

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46. Salmonella spp.

46.1. HAZARD IDENTIFICATION

46.1.1. 32.1.1 Aetiological agent

There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2008). All organisms considered in this section belong to the species *enterica* and the subspecies *enterica* except for *Salmonella arizonae*, which belongs to the subspecies *arizonae*. Using correct conventions, the names such as Dublin and Typhimurium, which do not have species status, should not be italicised. The correct name for the serovar Typhimurium is *Salmonella enterica* subsp. *enterica* serovar Typhimurium. However, in this risk analysis the simplified form such as *Salmonella* Typhimurium is used. Phage typing of *Salmonella* spp. is also commonly used to classify strains. The definitive phage type (Alley et al 2002) number is given after the species name, e.g., *Salmonella* Typhimurium DT104. In this chapter the term species or its abbreviations sp. (singular) or spp. (plural) are used broadly to include species, subspecies, serotypes and phage types, e.g., *Salmonella* spp.

46.1.2. OIE list

Not listed.

46.1.3. New Zealand status

Salmonella Abortus ovis, *S. arizonae*, *S.* Dublin, *S.* Enteritidis DT4, *S.* Gallinarum, *S.* Pullorum, *S.* Typhimurium DT44 and DT104 and *Salmonella* spp. (exotic affecting animals), are listed as unwanted organisms (MAF 2008).

46.1.4. 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 2007). Isolations from both medical and animal health laboratories are included.

S. Typhimurium DT104 is of particular importance because it exhibits multiple resistance to commonly used antibiotics and is a threat to human health (Hogue et al 1997; Jones et al 2002; Plagemann 1989). *S.* Typhimurium is endemic in New Zealand in both animals and humans, but in 2006 the definitive phage type DT104 occurred only three times and DT44 did not occur in 1,404 isolates from humans. These page types were not identified in 1,417 isolates from animals. *S.* Enteritidis DT4 was isolated six times in humans and not at all from animals in the same period (ESR 2007). The occurrence of these phage types in a few cases in humans may indicate that they have not become established in the New Zealand animal population and are rare in humans.

S. Choleraesuis is the main cause of salmonellosis in pigs in the USA and *S.* Typhimurium is the principle pathogen in Europe (Griffith et al 2006; Penrith et al 2004). *S.* Choleraesuis is a pig-adapted strain and has rarely been isolated from other animals. It is not present in New Zealand (ESR 2007). Disease in pigs is usually characterised by septicaemia and pneumonia and sometimes meningoencephalitis. Enterocolitis occurs infrequently (Griffith et al 2006; Penrith et al 2004). *S.* Typhimurium commonly causes enterocolitis and may cause septicaemia and abortion (Griffith et al 2006; Penrith et al 2004).

Outbreaks of disease in pigs caused by *S.* Typhisuis have been reported from Asia, Europe and the USA (Penrith et al 2004). Other *Salmonella* spp. are commonly isolated from healthy and diseased pigs and, although they are usually not a significant cause of disease in pigs, pigs can act as a source of infection for humans (Penrith et al 2004).

Once a herd becomes infected with a *Salmonella* sp. the organism spreads through the herd and many pigs become carriers that excrete the organism intermittently. Identification of carrier animals is difficult and requires repeated culturing of faeces.

Since septicaemia occurs in infected animals, infection of semen could occur.

46.1.5. Hazard identification conclusion

S. Choleraesuis is an exotic, zoonotic organism and *S.* Typhimurium type DT104 and DT44 and *S.* Enteritidis DT4 are unwanted and zoonotic organisms. Other exotic zoonotic *Salmonella* spp. may occur in pigs. Therefore these organisms are identified as a potential hazard in the commodity.

46.2. RISK ASSESSMENT

46.2.1. Entry assessment

Animals infected with *Salmonella* spp. may carry them for long periods and excrete them intermittently in faeces. Donor boars could be chronic carriers or be recently infected with *Salmonella* spp. Therefore the likelihood that donors could be infected with *Salmonella* organisms is non-negligible. In sheep *S.* Abortus ovis may be transmitted during mating (Vodas and Marinov 1986) and *S.* Morbificans has been described as a secondary invader of the bovine reproductive tract (Boryczko and Furowicz 1971). It is therefore possible that septicaemic pigs could excrete *Salmonella* spp. in semen. Although the likelihood that *Salmonella* spp. could be present in pig semen is low, it is assessed to be non-negligible.

46.2.2. Exposure assessment

Imported semen would be inseminated into sows and therefore exposure of New Zealand farmed pigs is inevitable.

46.2.3. Consequence assessment

Salmonella spp. could be transmitted by the faecal-oral route amongst pigs and infected pigs could develop disease or become subclinically-infected carriers. Newly introduced *Salmonella* spp. could spread widely due to stock movements and could result in the establishment of production-limiting salmonellosis in livestock. An example of how an emerging *Salmonella* sp. can effect New Zealand animal industries can be seen in the case of *S.* Brandenburg, which first emerged in 1997 and subsequently spread to many farms causing abortions in hundreds of sheep flocks within a few years (Kerslake and Perkins 2006). Introduction of new *Salmonella* spp. could therefore have significant economic consequences for the animal industries. Establishment of exotic zoonotic *Salmonella* spp. in animal populations, e.g., *S.* Typhimurium DT 104, could cause human disease requiring expensive treatment and possibly death in rare cases (Davies 2001; Hogue et al 1997).

There would be no particular consequences for the environment other than possibly sporadic cases of salmonellosis in wild or feral animals such as deer, goats, and birds. *S.* Typhimurium (DT160) was reported to be associated with several hundred deaths in sparrows (Alley et al 2002). The outbreak was self-limiting and did not cause lasting damage to the sparrow

population. However, *Salmonella* infections can establish in wild bird populations and cause mortalities over many years (Pennycott 2001).

Introduction of infected semen could lead to the establishment of new *Salmonella* spp. that have the potential to cause disease in humans, production-limiting disease of domestic animals and disease in wild and feral animals. Therefore the consequences are non-negligible.

46.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result the risk estimate for exotic *Salmonella* spp. is non-negligible and they are assessed to be a risk in the commodity. Therefore risk management measures can be justified.

46.3. RISK MANAGEMENT

46.3.1. Options

When drafting options for managing the risk posed by *Salmonella* spp. in semen the following points were considered:

- Donor boars should be healthy and show no signs of salmonellosis.
- Culturing of faeces from donor animals is unreliable due to the intermittent excretion of the organism by carrier animals. Therefore isolation attempts could be repeated several times to increase the likelihood of detecting carriers.
- Serological tests are not presently available that can reliably detect a wide variety of *Salmonella* serovars in individual carrier animals.
- Processed semen will generally contain antibiotics but, since resistance to antibiotics is widespread, they cannot be relied upon to inactivate *Salmonella* spp. (Jones et al 2002; Wray et al 1991).
- Culture of *Salmonella* spp. from a variety of animals and animal sources is well documented and comparatively simple to perform (Davies 2008).

There are no recommendations in the *Code* relating to *Salmonella* in semen.

The following options, given in ascending order of stringency, are available:

Option 1

Semen from healthy pigs not showing clinical signs of salmonellosis and housed on an artificial insemination centre could be imported without restriction.

Option 2

Faecal samples from donor boars could be cultured using suitable pre-enrichment and enrichment media (Davies 2008), on at least 3 occasions, twice in the 3 weeks prior to semen collection and once within 1 week of the completion date of collection of semen. All isolated *Salmonella* spp. could be identified to serotype and for *S.* Typhimurium and *S.* Enteritidis to phage type. The results could be reported to the director of Biosecurity New Zealand. Semen from boars carrying exotic *Salmonella* spp. could be prohibited from importation. The decision whether to import semen from boars infected with endemic species could be made by the importer.

Option 3

An aliquot of semen from each batch could be cultured using suitable pre-enrichment and enrichment media (Davies 2008). When culturing processed semen it could be assumed that if *Salmonella* spp. are not isolated they are not present or have been inactivated by the antibiotics used in preparation of the semen. Culturing from processed semen samples could therefore be recommended. Any isolated *Salmonella* spp. could be fully identified to serotype, and to phage type for *S.* Typhimurium and *S.* Enteritidis, and the results reported to the director of Biosecurity New Zealand. Semen infected with exotic *Salmonella* spp. could be prohibited from importation. The decision whether to import semen infected with an endemic *Salmonella* spp. could be made by the importer.

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