Import risk analysis: Llamas (*Lama glama*) and alpacas (*Vicugna pacos*) from specified countries

FINAL

ISBN 978-0-478-37526-8 (print)

ISBN 978-0-478-37527-5 (online)

14 December 2010

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Policy and Risk
MAF Biosecurity New Zealand

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Approved for general release

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Acronyms

CDC	United States Centers for Disease Control and Prevention
CF(T)	complement fixation (test)
DNA	deoxyribonucleic acid
ELISA	enzyme-linked immunosorbent assay
IFA(T)	indirect fluorescent antibody (test)
IHS	Import Health Standard
MAF	New Zealand Ministry of Agriculture and Forestry
MAFBNZ	Ministry of Agriculture and Forestry Biosecurity New Zealand
OIE	World Organisation for Animal Health
PCR	polymerase chain reaction
PEQ	pre-export quarantine
RNA	ribonucleic acid
RT-PCR	reverse transcriptase polymerase chain reaction
WTO	World Trade Organization

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Executive Summary

This risk analysis examines the risks involved with the importation of llamas (*Lama glama*) and alpacas (*Vicugna pacos*) from specified countries: Australia, USA, Canada, the European Union and South America.

An extensive hazard list of organisms of potential concern that could be associated with camelids has been collated in Table 1. Preliminary hazards are identified within Table 1 as those that have meet specified criteria. *Mycobacterium bovis* is the only endemic organism retained as a preliminary hazard since it is the subject of an official control programme under the Biosecurity Act 1993.

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

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

Risk management is not warranted for disease agents that are exclusively arthropod-borne, mainly through specific ticks, flies and mosquitoes that are not present in New Zealand. However, if new vector species were to establish here, measures may then become necessary to prevent introduction of such organisms. For organisms that are classified as hazards as a result of risk estimation, in the risk management step the options that could be used to effectively manage the risk are presented. The risk management options include quarantine, testing, vaccination and treatment as appropriate.

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

In the case of *Babesia* spp. of livestock, *Anaplasma* spp., bluetongue virus and epizootic haemorragic disease virus, the risk analysis concludes that the risks posed by these organisms in alpacas and llamas are negligible. As a result, the testing requirements for these organisms in the currently issued IHSs are not justifiable.

Thirty three individual organisms or groups of organisms were identified as preliminary hazards from the organisms of potential concern listed in Table 1. As a result of the individual risk assessments, 20 of these preliminary hazards were classified as hazards in the commodity and for each of these risk management measures are presented:

Bovine viral diarrhoea virus type 2

Bovine herpesvirus type 1

Equine herpesvirus type 1

Foot and mouth disease virus

Rabies virus

Vesicular stomatitis virus

Bacillus anthracis

Brucella spp.

Chlamydophila abortus

Coxiella burnetii

Leptospira serovars

Mycobacterium bovis

Mycoplasma haemolamae

Salmonella spp.

Trypanosoma spp.

Echinococcus granulosus

Other internal parasites (trematodes and nematodes)

External parasites (mites, fleas, lice and ticks)

Screwworm and other myiasis infestations

Hitch-hiker weeds and seeds

1. Introduction

The importation of alpacas and llamas is increasing each year, from around 50 animals several years ago to more than 550 during 2008 and 2009. This increase in the number of animals moving internationally is expected to continue, along with an increase in the number of countries from which alpacas and llamas are sourced.

Conditions have been developed in the past to allow importations from Chile and Argentina. However, importations only ever occurred from Chile and the conditions were not based on a risk analysis, but rather detailed bilateral negotiation between MAF and the Chilean Authority. The last consignment from Chile (1992-1993) had problems when seropositive animals to foot and mouth disease were detected in post-arrival quarantine (false-positive test results). Australia and Canada also experienced similar problems with foot and mouth disease seropositive animals being detected in post-arrival quarantine. In those cases, it was thought that vaccinated animals had been inadvertently included in the consignments. These episodes raised serious concerns regarding the ability of Chile to provide the necessary pre-export assurances required.

Because of this history when importing from Chile, New Zealand put in place a moratorium on the issuing of import health permits for llamas and alpacas from all of South America.

Alpacas and llamas are presently imported under the following Import Health Standards (IHSs):

Alpacas and Llamas from United States of America (July 2005)

Alpacas and Llamas from Australia (May 2006)

The measures in these IHSs are based on the MAF document "Disease Risk Analysis for the Importation of Llamas (*Lama glama*) and Alpacas (*Lama pacos*) into New Zealand" that was finalised in 1997 and up-dated in 1998. That risk analysis examined the risks involved with the importation of animals from the USA, Canada and Australia. The 1997 risk analysis *specifically excluded* South American countries from consideration.

The 1998 risk analysis was produced using procedures and policies that have meanwhile changed. The analysis identified potential hazards and provided a short discussion, including some recommendations from the *Terrestrial Animal Health Code*. The measures that were presented were based on other animal species, not specifically camelids. For the identified agents of concern, no risk assessment step was undertaken.

A new risk analysis is therefore required because of the changes in the risk analyses process, technical advances over the last 12 years and the increased number of countries requiring assessment.

Note that an importing country is entitled to expect validity in the veterinary certification of export. However, it must be made clear that an evaluation of an exporting country’s standards and performance is not made in this risk analysis. MAF may conduct an evaluation of veterinary services when drafting IHSs developed from this import risk analysis, particularly for countries with which there is no existing trade.

2. Scope

This qualitative risk analysis examines the risks involved with the importation of llamas (*Lama glama*) and alpacas (*Vicugna pacos*). Llamas and alpacas are hereafter referred to as "camelids".

This risk analysis is restricted to camelids imported from Australia, Canada, the United States of America (USA), Central and South America and the European Union (EU)[A]. Hereafter these countries are referred to as "relevant countries".

3. Commodity Definitions

The Family *Camelidae* contains three genera: *Camelus*, *Lama* and *Vicugna*.

Guanacos (*Lama guanicoe)* are a wild species and are not traded. The genus *Vicugna* includes the species *V. vicuna* which is a wild ancestor of the alpaca and an endangered species that will not be traded.

The dromedary (one-humped) and bacterian (two-humped) camel belong to the *Camelus* genus and are not part of the commodity definition.

Llamas (*Lama glama*) and alpacas (*Vicugna pacos*) (domesticated species) that have been certified on the day of shipment to be showing no clinical signs of infectious or parasitic disease are the commodity to be traded.

4. Risk Analysis Methodology

The methodology used in this risk analysis follows the 2006 MAF Biosecurity New Zealand *Risk Analysis Procedures- Version 1*. These procedures combine the guidelines in the *Terrestrial Animal Health Code* 2009 (hereafter referred to as the *Code*) of the World Organisation for Animal Health (OIE) and International Plant Protection Convention guidelines. The procedures provide a framework which adheres to the requirements set out under the World Trade Organization's Agreement on the Application of Sanitary and Phytosanitary (SPS) measures, 1995 and of the Biosecurity Act, 1993.

The process followed is shown in Figure 1 (overleaf).

[A] The EU presently includes Austria, Belgium, Bulgaria, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, and the United Kingdom.

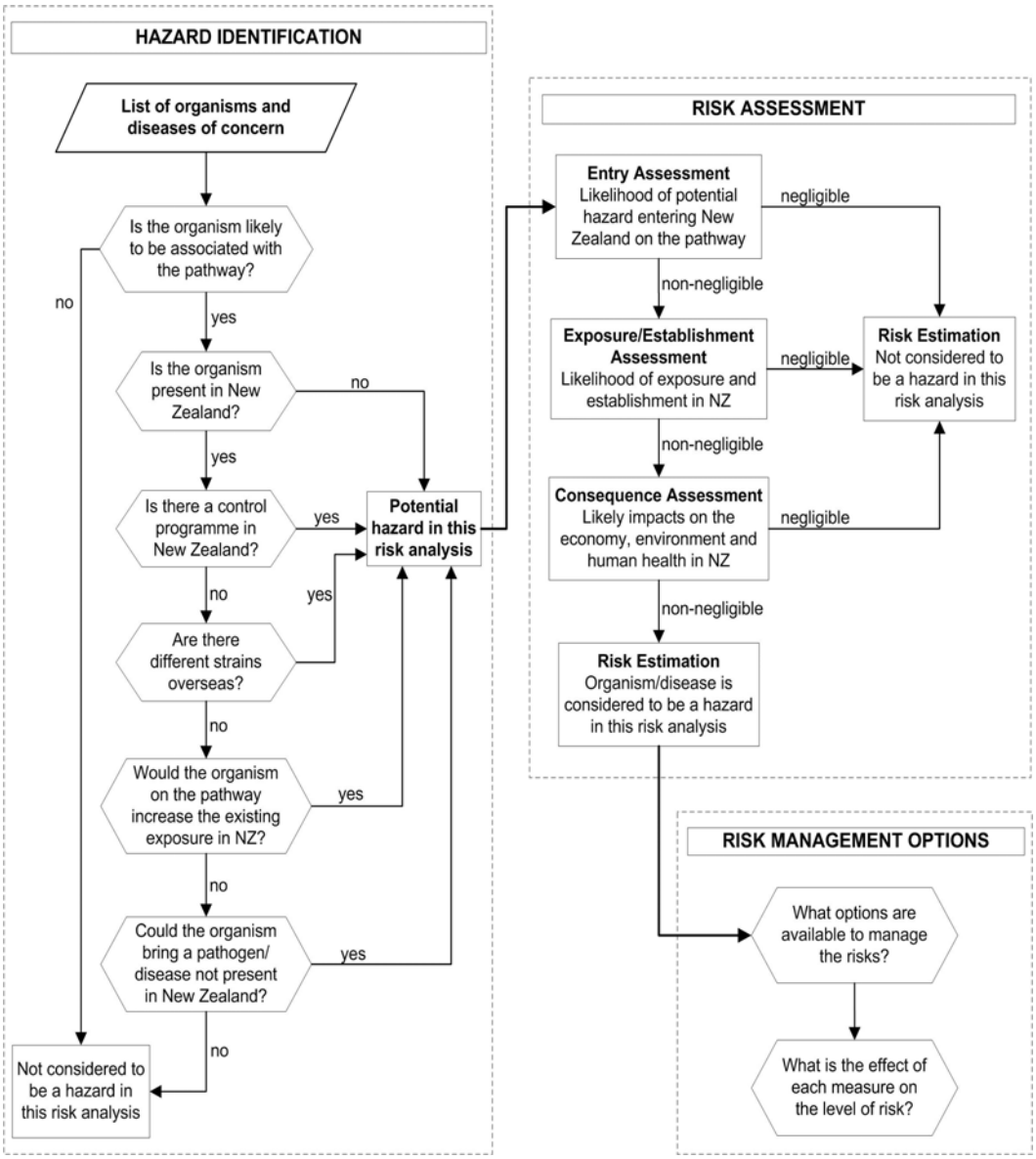

Figure 1. The risk analysis process.

4.1. Preliminary Hazard List (Organisms of Potential Concern)

From consulting authoritative texts and electronic data-bases an extensive list of organisms known to infect or infest camelids has been collated. From all the potential organisms of concern listed, preliminary hazards are identified by applying specific criteria to each organism to eliminate those that clearly do not constitute any risk. The remaining organisms are collated into a preliminary hazard list. The organisms in this list are subjected to hazard identification.

4.2. Hazard Identification

Each organism in the preliminary hazard list is subjected to a hazard identification step. This includes formal identification of the organism, whether it is an OIE listed disease, its New Zealand status, and a discussion on the relevant aspects of the epidemiology and characteristics of the organism. The hazard identification section is concluded by an

assessment of whether the organism is a potential hazard or not. All potential hazards are subjected to risk assessment.

4.3. Risk Assessment

Risk assessment consists of:

a) *Entry assessment*: The likelihood of a potential hazard (pathogenic organism) being imported with the camelids.
b) *Exposure assessment*: Describes the biological pathway(s) necessary for exposure of susceptible animals or humans in New Zealand to the potential hazard. Further, a qualitative estimation of the probability of the exposure occurring is made.
c) *Consequence assessment*: Describes the likely potential consequences of entry, exposure and establishment or spread of an imported potential hazard.
d) *Risk estimation*: An estimation of the risk posed by the potential hazard associated with importing camelids. This is based on the entry, exposure and consequence assessments. If the risk estimate is non-negligible, then the potential hazard is classified as a hazard and risk management measures may be justified to reduce the level of risk to an acceptable level.

Not all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of entry is negligible for a certain potential hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. The same situation arises when the likelihood of entry is non-negligible but the exposure assessment concludes that the likelihood of susceptible species being exposed is negligible, or when both entry and exposure are non-negligible but the consequences of introduction are assessed to be negligible.

4.4. Risk Management

For each organism classified as a hazard, a risk management step is carried out, which identifies the options available for managing the risk. Where the *Code* lists recommendations for the management of a hazard, these are described alongside options of similar, lesser or greater stringency where available. In addition to the options presented, unrestricted entry or prohibition may also be considered for all hazards. Recommendations for the appropriate sanitary measures to achieve the effective management of risks are not made in this document. These will be determined when an IHS is drafted.

As obliged under Article 3.1 of the WTO's SPS Agreement the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

4.5. Risk Communication

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

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

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

5. Organisms of Potential Concern and the Preliminary Hazard List

The first step in the risk analysis is hazard identification to ensure that all organisms of potential concern have been considered. For this risk analysis a list of organisms of potential concern was made comprising all the diseases/disease agents of alpacas and llamas located from the following sources:

- **OIE (2009).** *Terrestrial Animal Health Code*. Available at: http://www.oie.int/eng/normes/mcode/en_sommaire.htm
- **Taylor MA, Coop RL, Wall RL (2007).** *Veterinary Parasitology*. Blackwell Publishing, Oxford. pp 874.
- **Fowler M (1998).** *Medicine and Surgery of South American Camelids*. Iowa State University Press, Ames. pp 391.
- **Wernery U, Kaaden O-R (2002).** *Infectious Diseases in Camelids*. 2nd edition, Blackwell Science, Berlin. pp 404.
- **Ministry of Agriculture and Forestry (1997).** Disease Risk Analysis for the Importation of Llamas (*Lama glama*) and Alpacas (*Lama pacos*) into New Zealand. Amendments dated 21st April 1998.
- **Australian Quarantine and Inspection Service (2000).** Import Risk Analysis for the Importation of Camelids from Chile and Peru. Canberra. pp 19.
- **PubMed electronic scientific journal data-base.** Provides access to bibliographic information published in journals in the US and more than 80 other countries.
- **Additional diseases or disease agents.** As suggested by experts employed by MAFBNZ and interested parties that were consulted on the subject or were involved in reviewing this risk analysis.

Organisms/diseases identified as of potential concern from the above sources are listed in Table 1 (below).

The organisms of particular interest are those that are zoonotic and those that could be transmitted from camelids to domestic, feral or wild animals and humans.

The table indicates whether the organisms are zoonotic and whether they are known to occur in New Zealand. In Column 7 of the table, each organism is classified as to whether it is a preliminary hazard or not. An organism classified as a preliminary hazard meets the following criteria:

- All disease agents that are exotic to New Zealand and present in an exporting country or about which there is some uncertainty.

- In addition, organisms that occur in New Zealand for which there are known sub-species or strains or host associations that do not occur in New Zealand but do occur in an exporting country and are potentially harmful.
- Organisms that occur in New Zealand and an exporting country and for which an eradication programme administered by a Pest Management Strategy under the Biosecurity Act is in place. However, measures taken to prevent entry of the organism must not be more stringent than the measures adopted in the control programme for the eradication of the disease.
- Diseases that are of concern to human health.

Disease agents are not preliminary hazards if:

- After exhaustive searching no evidence is found that they are able to infect camelids or that they act as potential carriers of the pathogen concerned.
- The disease agent is known to occur in New Zealand and does not meet the criteria defined above for classification as a preliminary hazard.

Table 1. Organisms of potential concern

Disease agent	OIE Listed	Zoonotic	Disease or potential carrier	Present in NZ	Present in any relevant country	Preliminary hazard
Viruses						
Adenovirus	No	No	Yes	Yes (MAF 1997)	Yes (AQIS 2000)	No
African horse sickness virus	Yes	No	Yes (OIE 2009b)	No	No (OIE 2009b)	Yes+
Bluetongue virus	Yes	No	Yes (OIE 2009c; OIE 2009a)	No	Yes (OIE 2009b)	Yes
Borna disease virus	No	No	Yes (Wernery & Kaaden 2002b)	No	Yes (Wernery & Kaaden 2002b)	Yes
Bovine viral diarrhoea virus	Yes	No	Yes (Puntel1997; Puntel et al 2002)	Some strains	Exotic strains (Potgieter 2004)	Yes
Bovine herpesvirus type 1	No	No	Yes (Thedford & Johnson 1989)	Some strains	Exotic strains (Babuik et al 2004)	Yes
Camelpox virus	No	No	Yes (OIE 2009a)	No	No (OIE 2009b)	No
Contagious ecthyma virus	No	Yes	Yes	Yes (MAF 1997)	Yes	No
Crimean Congo haemorrhagic fever virus	Yes	Yes	Yes (OIE 2009a)	No	No (OIE 2009b)	No
Ephemeral fever virus	No	No	No (Chiu 1984)	No	Yes (St George 2004)	No
Epizootic haemorrhagic disease virus	No	No	Yes (OIE 2009a)	No	Yes (The Center for Food Security and Public Health 2006; Maclachlan & Osburn 2004)	Yes
Equine herpesvirus type 1 (Equine rhinopneumonitis)	Yes	No	Yes (Thedford & Johnson 1989)	Yes (Julian 1992; Dunowska et al 2002)	Yes (OIE 2009b; Allen et al 2004)	Yes #
Eastern, Western and Venezuelan equine encephalomyelitis viruses	Yes	Yes	Yes for EEEV only (Nolen-Watson et al 2007)	No	Yes (OIE 2009b)	Yes for EEEV only

Disease agent	OIE Listed	Zoonotic	Disease or potential carrier	Present in NZ	Present in any relevant country	Preliminary hazard
Foot and mouth disease virus	Yes	No	Yes [Wernery & Kaaden 2004)	No	Yes (OIE 2009b)	Yes
Influenza A viruses	No	Yes	No* (Wernery & Kaaden 2002e;Fowler 1992)	Yes	Yes	No
Japanese encephalitis virus	Yes	Yes	No	No	No (OIE 2009b)	No
Parainfluenza virus III	No	No	Yes	Yes (MAF 1997)	Yes	No
Peste des petits ruminants virus	Yes	No	Yes (OIE 2009a)	No	No (OIE 2009b)	No
Lumpy skin disease virus	Yes	No	No	No	No (OIE 2009)	No
Louping ill virus	No	Yes	Yes (Cranwell et al 2008)	No	Yes (Cranwell et al 2008)	Yes
Rabies virus	Yes	Yes	Yes (Wernery & Kaaden 2002h)	No	Yes (OIE 2009b)	Yes
Respiratory syncytial disease virus (bovine)	No	No	Yes	Yes (MAF 1997)	Yes	No
Rift Valley fever virus	Yes	Yes	Yes (The Center for Food Security and Public Health 2006)	No	No (OIE 2009b)	No
Rinderpest virus	Yes	No	Yes (OIE 2009a)	No	No (OIE 2009b)	No
Rotavirus and Coronavirus	No	No	Yes (Parreno et al 2001)	Yes (MAF 1997)	Yes	No
Enzootic bovine leukosis virus	Yes	No	No (Wernery & Kaaden 2002j)	Yes	Yes	No
Vesicular stomatitis virus	Yes	Yes	Yes (Wernery & Kaaden 2002m)	No	Yes (OIE 2009b)	Yes
West Nile virus	Yes	Yes	Yes (Kutzler et al 2004)	No	Yes (OIE 2009b)	Yes
BACTERIA, RICKETTSIA AND SPIROCHAETES						
Bacillus anthracis	Yes	Yes	Yes (Wernery & Kaaden 2002a)	No (Gill J 1992)	Yes (OIE 2009b)	Yes
Actinomyces lamae	No	No	Yes (MAF 1997)	Yes (MAF 1997)	Yes (AQIS 2000)	No
Brucella spp.	Yes	Yes	Yes (Wernery & Kaaden 2002c)	No (Mackereth G 2003)	Yes (OIE 2009b)	Yes
Burkholderia pseudomallei	No	Yes	Yes (Janmaat et al 2004)	No (Corkill & Cornere 1987)	Yes (Thomas 1981)	Yes
Clostridium tetani	No	No	Yes	Yes	Yes	No
Escherichia coli	No	Yes	Yes	Yes	Yes	No
Chlamydia spp.	No	Yes	Yes (Fowler 1992)	Some	Some	Yes
Clostridium botulinum	No	No	Yes	Yes	Yes	No
Clostridium perfringens	No	No	Yes	Yes (MAF 1997)	Yes	No
Clostridium septicum	No	No	Yes	Yes (MAF 1997)	Yes	No
Coxiella burnetii	No	Yes	Yes (Wernery & Kaaden 2002k)	No (Worthington 2001)	Yes	Yes
Fusobacterium	No	Yes	Yes	Yes (MAF 1997)	Yes	No

Disease agent	OIE Listed	Zoonotic	Disease or potential carrier	Present in NZ	Present in any relevant country	Preliminary hazard
necrophorum						
Pasteurella multocida types 6B and 6E	Yes	No	Yes (OIE 2009a; Wernery & Kaaden 2002i)	No	Yes (OIE 2009b)	Yes
Mannheimia haemolytica	No	No	Yes (Dwan et al 2008)	Yes	Yes	No
Leptospira spp. (exotic)	Yes	Yes	Yes Wernery & Kaaden 2002g)	Few serovars (Midwinter 1999)	Yes (OIE 2009b)	Yes
Listeria monocytogenes	No	Yes	Yes	Yes (MAF 1997)	Yes	No
Anaplasma (Ehrlichia) phagocytophilum	Yes	No	Yes Wernery & Kaaden 2002k)	No	Yes (Grzeszczuk et al 2004)	Yes
Mycobacterium avium subsp. *paratuberculosis*	Yes	No	Yes	Yes (MAF 1997)	Yes	No
Mycobacterium bovis	Yes	Yes	Yes (Wernery & Kaaden 2002i)	Yes**	Yes (OIE 2009b)	Yes
Corynebacterium pseudotuberculosis sheep/goat strain	No	No	Yes (Wernery & Kaaden 2002f)	Yes (Radostits et al 2007)	Yes (Radostits et al 2007)	No
Mycoplasma capricolum subsp. *capripneumoniae* (biotype F-38)	Yes	No	No (OIE 2009a)	No (OIE 2009b)	No (OIE 2009b)	No
Mycoplasma (Eperythrozoon) haemolamae	No	No	Yes (Reagan et al 1990)	No	Yes	Yes
Mycoplasma mycoides subsp. *mycoides* SC	Yes	No	No (OIE 2009a)	No (OIE 2009b)	No (OIE 2009b)	No
Nocardia asteroides	No	Yes	Yes	Yes (MAF 1997)	Yes	No
Salmonella spp.	No	Yes	Yes (Wernery & Kaaden 2002d)	Some spp.	Yes	Yes
Rhodococcus equi	No	Yes	Yes	Yes	Yes	No
Streptococcus spp.	No	Yes	Yes	Yes	Yes	No
Yersina pestis	No	Yes	Yes (Orloski & Lathrop 2003)	No (MAF 2009)	Yes (CDC 2009)	Yes
FUNGI						
Aspergillus fumigatus	No	Yes	Yes	Yes	Yes	No
Trichophyton verrucosum, *T. mentagrophytes*, and *Microsporum* spp.	No	Yes	Yes	Yes (MAF 1997)	Yes	No
Candida albicans	No	Yes	Yes	Yes	Yes	No
Coccidioides immitis	No	Yes	Yes	No (MAF 1997)	Yes	Yes
Rhizopus spp.	No	Yes	Yes	Yes	Yes	No
Dermatophilus congolensis	No	Yes	Yes	Yes	Yes	No

Disease agent	OIE Listed	Zoonotic	Disease or potential carrier	Present in NZ	Present in any relevant country	Preliminary hazard
PROTOZOA						
Eimeria alpacae, *E. lamae, E. punoensis, E. macusaniensis, E. peruviana*	No	No	Yes (Taylor , Coop & Wall 2007)	Some species (McKenna 2006)	Yes (OIE 2009b; MAF 1997)	No
Anaplasma centrale and *A. marginale*	Yes	No	No (Wernery & Kaaden 2002k; Taylor Coop & Wall 2007)	No	Yes	No
Babesia spp. of livestock	Yes	No	No (Taylor, Coop & Wall 2007, Wernery & Kaaden 2002l)	No	Yes	No
Encephalitozoon cuniculi	No	Yes	Yes	Yes (Anonymous 1980)	Yes	No
Sarcocystis aucheniae and *S. lamacenis*	No	Yes	Yes (Taylor , Coop & Wall 2007; More et al 2008)	Yes [(Mason & Orr 1993)	Yes	No
Toxoplasma gondii	No	Yes	Yes (More et al 2008)	Yes (McKenna 2009)	Yes	No
Neospora caninum, *Cryptosporidium parvum*, and *Giardia intestinalis*	No	Yes	Yes (Taylor , Coop & Wall 2007; More et al 2008)	Yes (McKenna 2009)	Yes	No
Theileria spp.	No	No	No (Taylor, Coop & Wall 2007)	Yes	Yes	No
Trypanosoma spp.	Yes	Yes	Yes (Wernery & Kaaden 2002)	No	Yes	Yes
INTERNAL PARASITES						
Cestodes: several species including *Echinococcus granulosus*	Yes	Yes	Yes (Taylor , Coop & Wall 2007)	No (MAF 1997)	Yes	Yes
Nematodes: many species	No	No	Yes (Taylor, Coop & Wall 2007)	Some	Yes	Yes
Trematodes:						
Fasciola hepatica	No		Yes (Taylor , Coop & Wall 2007)	Yes (McKenna 2009)	Yes	No
Fasciola gigantica			Yes (Taylor , Coop & Wall 2007)	No (McKenna 2009)	Yes	Yes
Fascioloides magna			Yes (Taylor , Coop & Wall 2007)	No (McKenna 2009)	Yes	Yes
Dicrocoelium dendriticum			Yes	No	Yes	Yes
EXTERNAL PARASITES						
New World Screwworm	Yes	Yes	Yes (Taylor, Coop & Wall 2007)	No (McKenna 2009)	Yes	Yes
Other myiasis infestations	No	Yes	Yes (Mattoon et al 1997; Radostits et al 2007)	Some spp.	Yes	Yes

Disease agent	OIE Listed	Zoonotic	Disease or potential carrier	Present in NZ	Present in any relevant country	Preliminary hazard
Exotic tick spp.	No		Yes (Taylor , Coop & Wall 2007)	No	Yes	Yes
Mites:						
Sarcoptes scabiei				Yes	Yes	No
Psoroptes ovis	No	Yes	Yes (Taylor , Coop & Wall 2007)	No	Yes	Yes
Chorioptes bovis				Yes	Yes	No
Lice:						
Microthoracius mazzai				No		Yes
M. minor			Yes (Taylor , Coop & Wall 2007)	No		Yes
M. praelongiceps				No		Yes
Bovicola breviceps				Yes (Palma et al 2006; Reagan et al 1990)	Yes	No
Fleas:						
Vermipsylla spp.			Yes (Fowler 1992)	No		Yes
HITCH -HICKERS						
Exotic weeds and seeds	No	No	No	No	Yes	Yes

+ Outbreaks have occurred outside Africa in Portugal and Spain (OIE 2009b).
* Exotic species or serotype/strains occur in humans and other animals overseas that are not known to be present here. Only a serological response detected, but no disease in camelids.
** An official control programme (National Pest Management Strategy) exists for cattle and deer.
# EHV-1 is a common endemic infection of horses. However, there is evidence to indicate exotic strains of greater pathogenicity occur in other countries.

5.1.1.1. Preliminary hazard list

Viruses

African horse sickness virus
Bluetongue virus
Borna disease virus
Bovine viral diarrhoea virus
Bovine herpes virus type 1
Epizootic haemorrhagic disease
Eastern equine encephalomyelitis virus
Equine herpesvirus type 1
Foot and mouth disease virus
Louping ill virus
Rabies virus
Vesicular stomatitis virus
West Nile virus

Bacteria, rickettsias and spirochaetes
Anaplasma phagocytophilum
Bacillus anthracis
Brucella spp.
Burkholderia pseudomallei
Chlamydia spp.
Coxiella burnetii
Leptospira spp.
Mycobacterium bovis
Pasteurella multocida types 6B and 6E
Mycoplasma haemolamae
Salmonella spp.
Yersinia pestis

Fungi
Coccidioides immitis

Protozoa
Trypanosoma spp.

Internal parasites
Echinococcus granulosus and other cestodes
Nematodes and trematodes

External parasites
Mites, lice and fleas
Ticks
Screwworm and other myiasis infestations

Hitch-hikers
Weeds and seeds

All organisms in the preliminary hazard list are subjected to hazard identification, and those concluded to be potential hazards are subjected to risk assessment.

References

References marked * were sighted as abstracts in electronic data-bases.

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6. African horse sickness virus

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: *Reoviridae*; Genus: *Orbivirus*, Species: *African horse sickness virus* (AHSV). There are 9 serotypes of AHSV (Mertens et al 2005).

6.1.2. OIE list

Listed as a disease of equidae.

6.1.3. New Zealand status

Listed on the Unwanted Organisms Register as a notifiable organism.

6.1.4. Epidemiology

African horse sickness (AHS) is an infectious but noncontagious viral disease affecting all species of equidae. Camels and dogs can also be infected. Humans are not natural hosts and it is not a zoonotic disease.

The virus is transmitted by midges in the genus *Culicoides* with *C. imicola* and *C. bolitinos* considered to be the principal vectors (Sanchez-Vizcaino 2008).

AHS is endemic in sub-Saharan central and east Africa and sometimes spreads to southern Africa and occasionally to northern Africa and Mediterranean countries. Outbreaks have occurred in Europe (Spain, 1966, 1987-1990 and Portugal, 1989) (Sanchez-Vizcaino 2008). The most serious infections occur in horses and mules. Zebras often do not show clinical signs of infection and are considered to be the natural reservoir hosts in Africa (Coetzer & Guthrie 2004; CFSPH 2006).

Dogs may become naturally infected by consuming contaminated horse meat but play no role in the epidemiology of the disease (Coetzer & Guthrie 2004). Reports of infection in camels appear to be uncommon and infection is not associated with clinical disease (CFSPH 2006). Few details are available as to the level and duration of viremia in camels and their role, if any, in the epidemiology of AHS (Guthrie 2008). The OIE ad hoc group on Camelidae diseases considers that camelids could potentially act as carriers of the virus (OIE 2009).

A *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al 1991). To date, seroconversion to arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped (Tana & Holder 2008).

AHS has not been associated with disease in camelids or camels. It is not known if any Camelidae develop a viraemia sufficient to infect feeding *Culicoides*.

Imported camelids would not be able to infect other animals in New Zealand since the virus can only be transmitted by vectors that are not present.

6.1.5. Hazard identification conclusion

Camelids are not known to play any role in the epidemiology of AHS. It is a noncontagious infection and transmission of the virus to other animals would not be possible due to New Zealand's freedom from *Culicoides* spp.

Therefore, AHSV is not considered to be a hazard in the commodity.

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7. Bluetongue virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

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

7.1.2. OIE list

Listed as a disease of multiple species.

7.1.3. New Zealand status

Listed on the Unwanted Organisms Register as an exotic, notifiable organism.

7.1.4. Epidemiology

BTV can infect many ruminant species and antibodies to the virus have been found in camelids. It is not a zoonotic disease therefore there is no threat to public health.

Bluetongue occurs in most tropical and sub-tropical countries. The global BTV distribution is currently between latitudes of approximately 53°N and 34°S but is known to be expanding in the Northern hemisphere (OIE 2009).

The virus causes disease mainly in sheep, occasionally in goats and rarely in cattle and deer (Verwoerd & Erasmus 2004). In camelids, disease associated with BTV infection is extremely rare. Few reports of clinical signs suggestive of bluetongue disease in camelids could be found. During the recent epizootic spread of BTV in Europe, a fatal case was described in an alpaca in Germany (Heinrich 2007) and the death of two llamas in France was attributed to BTV infection (Meyer et al 2009). An acute, fatal infection in an alpaca has recently been reported in the USA (Ortega et al 2010). An earlier report describes a suspected case of bluetongue affecting a llama (Fowler 1992).

BTV is transmitted by *Culicoides* spp. (midges) and several serological surveys have investigated seroconversion rates in South American alpacas. Prevalances of 20 % have been described in some reports, whereas other surveys have failed to detect any antibody. Camelids are susceptible to infection and seroconvert, however, there is no evidence that they act as a reservoir for the virus (Fowler 1992). The OIE ad hoc group on Camelidae diseases considers that camelids could potentially act as carriers of BTV (OIE 2007).

A *Culicoides* surveillance programme has been operating in New Zealand since 1991. Sentinel cattle are monitored for seroconversion to viruses transmitted by *Culicoides* spp. (bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses). To date, seroconversion to arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped (Tana & Holder 2008).

Camelids are resistant to disease and unlikely to show clinical signs of infection with BTV. Even if viraemic they would not be able to infect other animals since the virus can only be transmitted by *Culicoides* vectors that are not present in New Zealand.

Furthermore, the OIE *Terrestrial Animal Health Code* states that "A BTV free country or zone in which surveillance has found no evidence that *Culicoides* likely to be competent BTV vectors are present will not lose its free status through the importation of vaccinated, seropositive or infective animals" (OIE 2009).

7.1.5. Hazard identification conclusion

Camelids are not thought to play any role in the epidemiology of bluetongue. BTV transmission to other animals would not be possible due to New Zealand's freedom from *Culicoides* spp. Even if an animal were discovered to be infected or seropositive, the *Code* states that New Zealand would not lose its BTV-free status.

Therefore, BTV is not considered to be a hazard in the commodity.

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8. Borna virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family: *Bornaviridae*: Genus: *Bornavirus*; Species; *Borna disease virus* (Schwemmle et al 2005).

8.1.2. OIE List

Not listed.

8.1.3. New Zealand's status

Listed on the Unwanted Organisms Register as an exotic, unwanted organism.

8.1.4. Epidemiology

Classical Borna disease virus (BDV) encephalomyelitis, known as Borna disease (BD) in horses, cattle and sheep, is restricted to endemic regions in Germany, Switzerland and Austria (Staeheli 2000). A range of other animals from birds to primates, including cats, dogs and possibly humans, can be infected. The definitive host for BDV has not been identified, but rodents and birds are suspected (Greene & Berg 2006).

Antibody to BDV has been found in humans suffering from psychiatric disorders. However, the significance of the virus in human infections and as a cause of psychiatric disorders remains controversial (Carbone 2001).

Virus is excreted in nasal secretions, saliva and conjunctiva of infected horses and sheep. Natural transmission is presumed to occur by direct contact with contaminated fomites, including food, which leads to inhalation and ingestion of the agent. In recent studies, however, all attempts to demonstrate infectivity in secretions of horses have failed (Staeheli et al 2000). There is no clear evidence that transmission from horse to horse occurs. Infection does not appear to spread between cats either and there are no reports of vertical transmission occurring in any species (Staeheli et al 2000).

The virus is highly neurotropic, similar to rabies virus, and reaches the central nervous system (CNS) by intraaxonal transport. Injecting virus into the feet of neurectomized rats fails to lead to infection as virus is prevented from reaching the CNS (Carbone & Duchala 1987). Intravenous injection of rats also failed to establish infection, reinforcing the exclusiveness of the neural pathway. Experimentally the disease has been transmitted from infected rats and mice to naïve rats and mice through the olfactory route (Carbone & Duchala 1987). This lends support to the theory that rodents may be the reservoir hosts of BDV and that the olfactory nerves carry the virus to the brain. However, overall the transmission route(s) of BDV remain largely unknown (Kamhieh & Flower 2006).

Despite the fact that the disease has been known for more than 250 years, there is controversy regarding diagnosis and relative significance of BDV in animals (Staeheli et al 2000).

Borna disease was diagnosed in a group of camelids that died at two zoos in Erfurt, Germany in the mid 1970s. In these outbreaks both llamas and alpacas were affected and

died acutely or within 3 weeks. Clinical signs did not involve neurological signs, but anorexia and wasting was observed. Diagnosis of BD was confirmed from histological changes seen in the hippocampus and the presence of BDV demonstrated by immunohistochemistry (Wernery & Kaaden 2002).

More recently, genetic detection techniques are employed to demonstrate the presence of viral RNA using a RT-PCR test. However, the accuracy and reliability of the RT-PCR test has been questioned (Staeheli et al 2000; Carbone 2001). Although viral RNA has been demonstrated in an increasing number of countries and animal species, the occurrence of the disease is still mainly confined to parts of Germany and surrounding countries. Since studies using RT-PCR have not generally been confirmed by viral isolation, it is not known whether closely related viruses occur and what role they might play in causing disease and stimulating antibody production.

Detection in the CNS of BDV antigen by immunohistochemistry, of BDV RNA by *in situ* hybridization, or both in combination with neurohistopathological alterations is considered the most reliable method of confirming active CNS classical Borna disease (Greene & Berg 2006). The sensitivity and specificity of serological assays varies considerably between laboratories. A reason for this is that titres are usually very low (1:5 to 1:320) as the immune response to viral antigens is weak and these antibodies may have been induced by infection with an antigenetically related agent of unknown identity or exposure to some other related immunogen (Staeheli et al 2000).

8.1.5. Hazard identification conclusion

The currently available diagnostic tests for BDV are not well suited to diagnosing *intra vitam* (during life) infections in animals or humans. The epidemiology of BDV remains unclear and several key questions, including whether it causes psychiatric disease in humans and the extent of its distribution worldwide, are controversial.

Alpacas and llamas in German zoos have been diagnosed with BD with fatal consequences. Therefore BDV is considered to be a potential hazard in the commodity.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

BD is a rare disease primarily affecting horses and sheep in recognised endemic regions of Europe (Kolodziejek et al 2005). It appears that reported disease in alpacas and llamas is limited to the one report from German zoos in the mid 1970s. Infection is difficult to diagnose, with a largely unknown epidemiology and distribution. Serology remains controversial since seropositivity does not necessarily mean the animal is carrying the virus.

Since BDV is reported extremely rarely in alpacas and llamas, and death results quickly if affected, it is unlikely they are reservoir hosts. It is more likely that they are incidental hosts and are unlikely to be important in the epidemiology of BD.

The likelihood of importing an infected alpaca or llama is remote therefore the likelihood of entry is assessed to be negligible.

8.2.2. Risk estimation

Since entry is assessed to be negligible, the risk of importing llamas or alpacas infected with BDV is estimated to be negligible. BDV is therefore not classified as a hazard in the commodity.

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9. Bovine viral diarrhoea virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family: *Flaviviridae*; Genus: *Pestivirus*, Species: *Bovine viral diarrhea virus* (Thiel et al 2005). There are two genotypes BVDV1 and BVDV2 (Booth et al 1995). In each genotype both cytopathic and non-cytopathic biotypes occur.

9.1.2. OIE list

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

9.1.3. New Zealand status

Bovine viral diarrhoea virus genotype 1 (BVDV1) is endemic in New Zealand but genotype 2 (BVDV2) is exotic (Vilcek et al 1998; Horner 2000).

9.1.4. Epidemiology

BVDV1 has a world-wide distribution, including New Zealand and Australia (Horner 2000; Vilcek et al 1998). In New Zealand, most cattle have been exposed to BVDV1 and the prevalence of antibodies is around 60 % (Littlejohns & Horner 1990). BVDV2 occurs in North America (Potgieter 2004), Italy (Falcone et al 2001), the Netherlands (Barkema et al 2001) and in the United Kingdom (David et al 1994; Barkema et al 2001; Drew et al 2002; Nettleton & Gunn 2002; Cranwell et al 2005). The only isolation of a BVDV2 strain in New Zealand was from a batch of foetal calf serum imported from the USA (Horner 2000). The virus was contained in the laboratory. BVDV2 has not been described in Australia.

BVDV is normally transmitted by direct contact between infected animals and/or possibly by aerosol transmission over short distances (Potgieter 2004). It is also transmitted in semen, particularly from persistently infected bulls which shed virus in their semen for years (Potgieter 2004).

In cattle, the incubation period is usually about 3-7 days (Brownlie 2005) and the animals may remain viraemic for 4-15 days after initial infection (Potgieter 2004). Viraemia seldom exceeds 10-14 days (Brownlie 2005). Antibodies develop 2-4 weeks after infection. The incubation period and viraemic period of natural infections in camelids is not known. It is assumed that they are likely to be similar to those in cattle infections.

BVDV1 infection of non-pregnant cattle usually results in a mild infection typified by pyrexia and leukopenia from about 3-7 days, with viraemia and nasal excretion of the virus occurring during this period (Brownlie 2005). The clinical signs are often so mild that they are not observed or only mild signs and occasionally diarrhoea is seen (Potgieter 2004). Since BVDV1 is widely distributed in most herds, cattle are commonly infected before they become pregnant, resulting in a population that is mostly immune and does not carry the virus. Infection of naïve pregnant animals, particularly during the first trimester, may result in death of the conceptus or full term, or near full term, delivery of immunotolerant persistently infected calves.

It has been suggested that as many as 7 % of foetal deaths in Swiss dairy cattle may be caused by infection with BVDV (Rufenacht et al 2001). BVDV2 strains that cause a more severe form of the disease have been described in the USA (Pellerin et al 1994). In these cases the mortality rate was up to 10 % (Potgieter 2004) and the disease was characterised by severe leucopenia and haemorrhagic disease (Brownlie 2005).

Immunotolerant persistently infected cattle may be clinically normal or may not thrive and die within a year. They are always infected with non-cytopathic strains of the virus (Brownlie 2005). Superinfection of persistently infected animals with a cytopathic BVDV strain results in the development of mucosal disease (MD) (Potgieter 2004; Brownlie 2005). The cytopathic strain that re-infects the persistent carrier animals may result from a mutation of the persistent non-cytopathic strain or from infection with a new extrinsic cytopathic virus (Potgieter 2004; Brownlie 2005). Mucosal disease is invariably fatal. In acute cases death occurs within 2-21 days while in chronic cases the animal may survive for up to 18 months (Potgieter 2004).

An ELISA is available to detect BVDV antibody (Drew 2008). Despite the fact that serologically positive animals are usually no longer infected, exceptions are known to occur and a minority of persistently infected animals are also serologically positive. Some bulls that develop antibody titres continued to excrete infectious virus in semen for at least 5 months after experimental infection (Givens et al 2003). Further, a single case of a bull that was serologically positive and had no detectable virus in its blood but consistently excreted virus in its semen (Voges et al 1998). This led to a change in the recommendations made in the *Code*. It is now necessary for bulls that are antibody positive when they enter an AI station to have their semen tested for virus and for bulls that seroconvert to have every batch of semen that they have produced since their last negative serological test, tested for BVDV.
It is assumed that male camelids could similarly be persistently infected although seropositive when imported. The antibody ELISA will not detect persistently infected animals that are immunotolerant. Antigen detection ELISAs are available but less sensitive than the RT-PCR. Although there is no *Code* chapter for BVDV, the *Manual* lists virus isolation or antigen-detection ELISA as the prescribed tests for international trade. An RT-PCR is available to detect viral RNA in blood (Rufenacht et al 2001; Stokstad et al 2003). Kapil et al (2009) state that the commercial antigen ELISA can give false positive results when testing camelid serum. He notes “screening new world camelids by BVD viral-specific PCR on whole blood will detect active infection/viraemia”, although PCR testing is not validated for international trade.

BVD and MD are primarily diseases of cattle. Historical reports describe several serological studies that confirm camelids are susceptible to infection with BVDV. In a serological survey conducted in Peru on 117 alpacas that grazed with cattle, the prevalence of antibodies to BVDV was 11 %. In 270 llamas from Oregon, USA, the seroprevalence was reported to be 4.4 % (Wernery & Kaaden 2002). A survey of crias (unweaned baby camelids) in the USA found that 25 % of the herds studied were seropositive to BVDV (Topliff et al 2009). Historically, disease caused by BVDV in camelids had been described only once, in 1994. The affected llamas suffered excessive nasal discharge and diarrhoea before death. Experimental infection of four pregnant llamas during gestation did not result in clinical signs of disease or foetal infection or persistent BVDV infection of crias (Wentz et al 2003).

However, the first report describing a naturally persistently infected (PI) Canadian alpaca cria was published in 2005 (Carman et al 2005). Subsequently persistent infections have been reported by several workers (Mattson et al 2006; Foster et al 2007; Byers et al 2009; Kim et al 2009; Topliff et al 2009) Among 63 herds studied in the USA, herd prevalence for persistent infection of crias was found to be about 6.3 % (Topliff et al 2009). It is not known whether persistently infected crias go on to develop MD. Where virus isolation has been carried out, in all cases noncytopathogenic BVDV type 1 strains have been isolated from camelids. However, this is not unexpected since these strains are more prevalent than the cytopathogenic strains in cattle (Goyal et al 2002).

Although there is no evidence that proves conclusively that BVD2 is present in camelids, the OIE has classified BVD viruses as significant pathogens of camelids (OIE 2008) and recent studies suggest that persistent infections occur in these animals. Therefore BVD viruses could be considered an emerging disease in camelids.

9.1.5. Hazard identification conclusion

It is concluded that BVDV2 could infect camelids since they are susceptible to infection with BVDV1. Therefore BVDV2 is considered to be a potential hazard in camelids from countries where the virus occurs.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Although there is no direct evidence of BVD2 virus in camelids, it is assumed that, similar to BVD1 infections, animals either in the acute stage of infection or persistently infected could be excreting BVDV2 when imported into New Zealand. Therefore, the likelihood of entry in the commodity is considered to be non-negligible.

9.2.2. Exposure assessment

After importation, infectious carriers of BVDV2 would be in contact with, and could infect, naïve New Zealand camelids and cattle. The likelihood of exposure is therefore assessed to be non-negligible.

9.2.3. Consequence assessment

BVDV2 is exotic to New Zealand and, if introduced, it would be expected to spread amongst susceptible camelids and to cattle. Even those immune to BVDV1 may not be fully protected. Although some BVDV2 strains are of low virulence, mortalities of up to 10 % could result in cattle from initial infection with virulent BVDV2 strains (Potgieter 2004). It is therefore considered that the consequences of introducing the virus would be non-negligible.

As the virus does not infect humans, there would be no consequences for human health.

BVDV1 is known to infect deer and goats (Horner 2000). Antibody to the virus is known to develop in these species but disease has not been described. It is therefore likely that BVDV2 would also infect deer and goats, but it is not known whether the virus would cause significant disease in these species. The virus is not a risk to birds and the likelihood that there would be any consequences for the environment is considered to be negligible.

Therefore, since the the consequences to New Zealand livestock, particularly cattle, could be significant if BVD2 were introduced in any species of imported animal, the consequences are assessed to be non-negligible.

9.2.4. Risk estimation

Under the assumption that BVDV2 may be present in imported camelids and is epidemiologically similar to BVDV1 in these animals, the entry, exposure, and consequence assessments are all non-negligible, the risk estimate for BVDV2 is non-negligible and it is classified as a hazard in the commodity. Therefore, risk management measures may be justified.

9.3. RISK MANAGEMENT

9.3.1. Options

The following points were considered when drafting options for the effective management of BVDV2 in the commodity:

- It is assumed that BVDV2 is present in camelids and is epidemiologically similar to BVDV1 in these animals.
- Australia is the only relevant country considered to be free from BVDV2.
- As there is no *Code* chapter for BVD, there are no international risk management standards for any species. This notwithstanding, the *Manual* lists virus isolation and antigen-detection ELISA as the prescribed tests for international trade.
- Routine diagnostic tests are not available to distinguish BVDV1 and BVDV2. Therefore, any animal that is carrying BVDV and comes from a country in which BVDV2 is present could potentially be carrying BVDV2.
- Serologically negative immunotolerant, viraemic camelids have been reported (Carman et al 2005). In addition, viraemia in cattle may persist for several months after infection. Since it is possible for both antibody positive and negative animals to be viraemic, serological tests alone are not effective for screening camelids for importation.
- Antigen detection ELISAs are less sensitive than the RT-PCR. Further, the commercial antigen ELISA is known to give false positive results when testing camelid serum (Kapil et al 2009). Therefore the RT-PCR could be used for detecting camelids viraemic with BVD viruses in general. However this is not an OIE-prescribed test for international trade.

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

1. Camelids could be subjected to an antigen detection test within 14 days of shipment, with negative results. The test could be:
    - Virus isolation; or
    - Antigen ELISA; or
    - RT-PCR

2. Camelids undergo a pre-export isolation period of 28 days, and within 14 days of shipment they are subjected to an an antigen detection test, with negative results. The test could be:
    - Virus isolation; or
    - Antigen ELISA; or
    - RT-PCR
3. Animals eligible for importation into New Zealand could be only from countries that are free from BVDV2 virus (Australia).

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10. Bovine herpesvirus type 1

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agents

Family: *Herpesviridae*; Subfamily: *Alphaherpesvirinae*; Genus: *Varicellovirus*; Species: *bovine herpesvirus* 1 (BHV-1) (Davison et al 2005).

BHV-1 is associated with infectious bovine rhinotracheitis (IBR) and infectious pustular vulvovaginitis/infectious pustular balanoposthitis (IPV/IPB). Subtypes BHV-1.1 and BHV-1.2 can be identified by restriction endonuclease analysis of DNA (Engels et al 1981; Wentink et al 1993; Babuik et al 2004). Rhinitis and respiratory signs are associated with subtype 1.1, pustular vulvovaginitis and balanoposthitis is associated with subtype 1.2. Subtype 1.2 strains can be further classified as BHV-1.2a and BHV-1.2b strains. Some subtype 1.1 and 1.2a strains are abortifacient (Miller et al 1991). Subtype 1.2b strains are associated with respiratory and genital infections but not with abortions (Miller et al 1991; van Oirschot 1995).

Table 2 Bovine herpesviruses

Type	**Syndrome**		
	IBR	**IPV/IPB**	**Abortion**
BHV-1.1	+	-	+
BHV-1.2a	+	+	+
BHV-1.2b	+	+	-

10.1.2. OIE list

Infectious bovine rhinotracheitis and infectious pustular vulvovaginitis are listed as diseases of bovidae.

10.1.3. New Zealand status

Only BHV-1.2b has been isolated in New Zealand (Wang et al 2006). Abortions caused by bovine herpesviruses have not been seen in New Zealand (Horner 1990; Fairley 1996), and an attempt to cause abortion by experimental infection with the New Zealand strain of the virus was unsuccessful (Durham et al 1975). At the present time identification of abortifacient strains of the virus from either subtype 1 or 2 strains would require experimental infection of pregnant cows and a more pragmatic approach is to regard BHV-1.1 and BHV-1.2a as exotic organisms. Abortifacient strains are classified as unwanted notifiable organisms (MAF 2009).

10.1.4. Epidemiology

IBR/IPV has a world-wide distribution. Australia reports that BHV-1.2b is present but BHV-1.1 and BHV-1.2a has never occurred (Animal Health Australia 2010).The virus is endemic and widespread in New Zealand (Neilson & Grace 1988). Both the IBR and the IPV syndrome have been described (Horner 1990; Fairley 1996; Vermunt & Parkinson 2000). However, in the vast majority of cases there are no, or only mild, clinical signs (Vermunt & Parkinson 2000).

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

Camelids have been found to have antibody to BHV. Rarely BHV-1 has been isolated from diseased and dead llamas (Williams et al 1991; Wernery & Kaaden 2002). In Peruvian llamas and alpacas a seroprevalence of about 16% was found in herds grazed on the same pastures as cattle, sheep and goats. Alpacas that were managed separately from other ruminants were found to have a 5% seroconversion rate. In Oregon, a very low rate of 0.7% was found in 270 alpacas surveyed (Wernery & Kaaden 2002). In another serosurvey of 390 llamas in Argentina an antibody prevalence of 0.77% was reported. The positive samples were from a herd where the seroprevalence in the cohabitating cattle was found to be 60% (Puntel et al 1999).

Although camelids are susceptible to infection with BHV-1, disease is rare. It is not known whether natural transmission occurs in camelids and infection in camelids may be a spill-over from cattle (Puntel et al 1999). Indeed, the prevalences in camelids would be expected to be higher if natural transmission was occurring within infected flocks. The studies with very low prevalences are likely to reflect false positive or non-specific reactions. Although BHV-1 is considered non-pathogenic in camelids (Wernery & Kaaden 2002), the possibility that they could be infectious cannot be excluded. In other species such as goats, clinical signs of infection are mild but natural transmission can occur (Straub 1990).

The OIE ad hoc group lists BHV-1 as a significant disease of camelids and recommends investigation of their susceptibility to BHV-1 (OIE 2008). In view of the uncertainty around the epidemiology of this virus in camelids and in light of the evidence for transmission from infected goats, this risk analysis assumes that transmission from infected camelids can occur.

10.1.5. Hazard identification conclusion

Abortifacient strains of IBR/IPV virus are exotic, notifiable organisms that can be associated with camelids. Under the assumption that transmission to other susceptible animals may occur, these organisms are classified as potential hazards.

Since tests suitable for export/import certification are not available to identify abortifacient strains in the laboratory, it is necessary to regard all BHV-1 strains as potential hazards.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

In latently infected cattle, the virus remains in the neurons of the ganglia and can be periodically re-activated. It is assumed that this also occurs in camelids.

Camelids that have become infected without showing clinical signs and are still shedding virus or are latently infected could be imported from endemic areas. Therefore the likelihood of entry of BHV-1 in the commodity is assessed to be non-negligible.

10.2.2. Exposure assessment

Imported llamas and alpacas are likely to be kept close by, or to share pasture with, herds of susceptible camelids and cattle. It is assumed that transmission occurs between camelids and that they may act as a reservoir of infection for cattle. Recently infected cattle may excrete the virus in nasal secretions and aerosols for up to 14 days after infection (Babuik et al 2004). However, disease is rarely observed in camelids, so viral shedding is probably lower than that of infected cattle. Experimental studies conducted on goats showed that their clinical reaction was mild, but nevertheless natural transmission occurred (Straub 1990). The same situation is assumed to be possible in camelids. Therefore, it is assumed that viral shedding can occur from camelids and that this is capable of infecting other animals.

Further, in times of stress such as transport, parasite infestations, treatment with corticosteroids or normal parturition, latent infections may be reactivated in camelids as occurs in other species. This could result in aerosol viral shedding or contamination of the environment from infectious birth products.

Therefore the likelihood of exposure of naïve indigenous camelids and cattle to potentially exotic strains of BHV-1 associated with the commodity is considered to be non-negligible.

10.2.3. Consequence assessment

Introduction of abortifacient strains of BHV-1.1 or 1.2a may result in rare sporadic outbreaks of abortion in llamas and alpacas. Reproductive wastage and economic losses would be significantly greater should infection be introduced from infected imported camelids into the country's cattle herds.

Since clinical disease is rare in camelids, the disease is unlikely to be of direct concern to other camelids but introduction of the virus could have significant consequences for the cattle industries. The consequences for the camelid and cattle industries are assessed to be non-negligible.

There is no evidence to suggest that the virus would cause significant disease in deer, feral goats, thar or indeed camelids themselves. Other animal species, including birds are not known to be infected. Therefore, it is considered that the effect of introduction of the virus on the environment would be negligible.

There would be no consequences for public health since the virus does not infect humans.

10.2.4. Risk estimate

Based on the assumption that transmission may occur from infected camelids, the entry, exposure, and consequence assessments are all non-negligible. Therefore the risk estimate for BHV-1 is non-negligible. As such, BHV-1 strains are classified as hazards in the commodity and risk management measures may be justified.

10.3. RISK MANAGEMENT

10.3.1. Options

The following points were considered when drafting options for the effective management of BHV-1 in the commodity:

- Acute infections are generally subclinical and chronically infected animals are likely to be latent carriers of the virus.
- Latent infections may persist for the life of the animal. Therefore quarantine is not an option for preventing introduction of the virus.
- The *Manual* recommends the virus neutralisation test and various ELISAs as prescribed tests for international trade. These have been developed for use in cattle, but have also been applied to camelids[B]. These tests do not distinguish between antibodies induced by different BHV-1 strains. As a result, any animal that is serologically positive could be considered infected with an exotic strain.
- For trade, demonstrating absence of infection is necessary and positive animals should not be eligible for importation. The *Manual* states that the immune response develops in 7-10 days and it is presumed the immune response persists for life, although it may fall below the detectable limit of some tests. To detect recently infected animals, testing should be carried out after ata least 10 days in quarantine to allow for antibody development.
- The *Code* considers the clinical syndromes of IBR and IPV in cattle, but there are no international standards that are applicable when trading camelids.

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

1. Since camelids appear to be very rarely affected by BHV-1, and it has not been conclusively shown that they are capable of transmitting infection to other susceptible animals, it could be considered that no measures are necessary.
2. Camelids could be subjected to a period of PEQ with serological testing to be carried out at least 10 days after entering the facility, with negative results.
3. Camelids could be imported from countries that are considered free from BHV-1.1 and BHV- 1.2a.

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11. Epizootic haemorrhagic disease virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Family: *Reoviridae*; Genus: *Orbivirus*, Species: *Epizootic haemorrhagic disease virus*. There are 10 serogroups/strains (Mertens et al 2005).

11.1.2. OIE list

Epizootic haemorrhagic disease is an OIE listed disease but there is no *Code* chapter (OIE 2009).

11.1.3. New Zealand status

Listed on the Unwanted Organisms Register as an "other exotic organism" (MAF 2009).

11.1.4. Epidemiology

Epizootic haemorrhagic disease (EHD) is primarily a disease of white tailed deer in the southeast USA. Outbreaks have also been reported in wild pronghorn antelope and bighorn sheep. Although infection with the virus has been reported in ruminant species in other regions of the world, it is not regarded as pathogenic in domestic ruminants (Maclachlan & Osburn 2004). Although the virus is listed as occurring in camelids (Mertens et al 2005; OIE 2008) no references were located to suggest that it causes disease in camelids or that camelids are maintenance hosts. The virus is transmitted by a variety of *Culicoides* spp. (Maclachlan & Osburn 2004; Paweska et al 2005), which are not present in New Zealand. Evidence from other species indicates that animals infected with Orbiviruses are not infectious and that transmission is only by *Culicoides* spp. vectors.

11.1.5. Hazard identification conclusion

Epizootic haemorrhagic disease virus rarely infects camelids. Infected camelids would not be infectious and the vectors for the disease are not present in New Zealand (Tana & Holder 2007; Frazer & Green 2008). Therefore, introduction of infected animals would not result in establishment of EHD virus in New Zealand and it is not considered to be a hazard in the commodity.

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12. Eastern equine encephalitis virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: *Togaviridae*; Genus: *Alphaviridae*; Species: *Eastern equine encephalitis virus* (EEEV) (Weaver et al 2005).

12.1.2. OIE list

Listed as a disease of equidae.

12.1.3. New Zealand status

It is listed as notifiable on the Unwanted Organisms Register.

12.1.4. Epidemiology

Eastern equine encephalitis virus is an arthropod-borne virus transmitted by mosquitoes. The disease has remained confined to the southern United States, central and northern South America where the virus is maintained in a mosquito/bird cycle. Virus titres build up in birds during the summer with the ornithophilic mosquito *Culiseta melanura* acting as the primary vector. When high levels of virus are present, so called 'bridge vectors' that feed on both birds and mammals transmit the virus to mammals. Virus has been isolated from at least 20 species of mosquitoes from six genera (Gibbs 2004). Birds that are indigenous to infected areas are not susceptible to the disease but may act as carriers of the virus. Introduced species of birds and mammals including man, horses and South American camelids (Nolen-Watson et al 2007) are susceptible and may develop a usually fatal disease when infected. The overwintering mechanism in temperate parts of America remains obscure as transovarial transmission is not considered to be important. In tropical and subtropical areas the mosquito/bird cycle may be continuous (Gibbs 2004). As with horses and humans, infected camelids are considered to be non-contagious dead-end hosts. The disease has not established anywhere in the world outside of the known endemic regions, indicating that conditions required for the maintenance of the virus are specific to these geographic areas.

12.1.5. Hazard identification conclusion

Culisetia melanura is not present in New Zealand and the likelihood that other critical elements for the maintenance of the virus to be able to establish in New Zealand is considered to be negligible. Since infected camelids are not contagious and vectors are not present, the virus could not be transmitted from introduced camelids or establish here. Therefore, EEEV is not considered to be a hazard in the commodity.

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13. Equine herpesvirus type 1

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Family: *Herpesviridae*; Subfamily: Alphaherpesvirinae; Genus: *Varicellovirus*; Species: *Equid herpesvirus* 1 (EHV-1) (Davison et al 2005).

13.1.2. OIE list

"Equine rhinopneumonitis" is listed in the *Code* as a disease of horses, and the *Code* contains a chapter on this disease, which states in Article 12.9.1.:

> Equine rhinopneumonitis (ER) is a collective term for any one of several highly contagious, clinical disease entities of equids that may occur as a result of *infection* by either of two closely related herpesviruses, equid herpesvirus-1 and -4 (EHV-1 and EHV-4).

The *Code* chapter and the *Manual* restrict their discussion to equids only.

13.1.3. New Zealand status

Both EHV-1 and EHV-4 are present in New Zealand, but reports of serious disease associated with these viruses are rare (MAF 2000). Since there are reports of EHV-1 strains of greater pathogenicity in other countries (Nugent et al 2006), this chapter focuses on exotic strains of EHV-1 that are more pathogenic than strains already present in New Zealand.

13.1.4. Epidemiology

EHV-1 infects equids and occurs world-wide causing a wide range of diseases ranging from inapparent respiratory infection to abortion and potentially fatal neurological disease.

EHV-1 is highly infectious, and transmission is by the inhalation of infected droplets or by the ingestion of material contaminated by nasal discharges or aborted foetuses. Horses, and presumably camelids recovering from EHV-1 infection are likely to become long-term latent carriers of the virus, and they would not show signs of infection and would not excrete the virus except when it is reactivated due to stress or steroid treatment at which time it is shed in nasal secretions (Allen et al 2004; Radostits et al 2007).

Differences exist between EHV-1 viruses in their ability to disseminate and to establish infection at vascular endothelial sites, particularly within the endometrium and the central nervous system, and a sustained cell-associated viraemia appears to be responsible for the development of disease in EHV-1 infected horses (Goodman et al 2007). Outbreaks of neurological disease in horses caused by EHV-1 have been reported with increasing frequency in the USA in recent years (Allen 2008). A point mutation of a single amino acid of the DNA polymerase is strongly associated with these outbreaks of neurological disease (Nugent et al 2006). Sophisticated DNA technology has been used to detect the virus in tissues of animals latently infected with the mutant strain (Allen 2007; Allen et al 2008). However, a recent investigation of archived EHV-1 isolates collected from equine abortions in Kentucky dating

back to 1951, has revealed that the so-called new mutant strain did, in fact, exist in the 1950s, but at a low prevalence (Smith et al 2010). The investigation concluded that since that time the prevalence of the neuropathogenic genotype has been increasing, at least in the USA. It is assumed that the neuropathogenic strain has some sort of selection advantage over non-pathogenic strains, but the mechanism of this is not understood (Smith et al 2010).

Although EHV-1 is considered a disease of equids, it has been isolated from a natural disease outbreak that occurred in camelids. An outbreak in 1984 causing total blindness in 22 of 100 alpacas and llamas occurred on a quarantine farm in New York state (USA). The animals had originated from Chile 6 months previously and over a 30 day period they developed blindness characterised by retinitis and optic neuritis (Rebhun et al 1988). Some of the affected animals developed a generalised encephalitis and died. A herpesvirus indistinguishable from EHV-1 was isolated from the brain or ocular tissues of 4 alpacas and 1 llama. All but one of the affected animals had detectable antibody titres against EHV-1 and antibody testing of cohorts also detected EHV-1 antibody (Rebhun et al 1988).

An experimental study carried out at the Plum Island Animal Disease Center, confirmed that EHV-1 infects alpacas, causing blindness, severe neurological signs and death (House et al 1991). Severe clinical signs and death occurred acutely by day 8 post-inoculation in 2 of 3 llamas infected. The third animal developed a mild depression and fever between day 6-8 post-inoculation with a decreased visual acuity. Except for the decrease in visual acuity, this animal remained clinically normal between days 10-32 post-infection when the study concluded.

No vaccination efficacy trials have been carried out in camelids and there are no vaccines registered for use in these animals. Further, vaccination does not eliminate latent infections.

13.1.5. Hazard identification conclusion

The neurological form of EHV-1 disease is considered to be an emerging disease in horses, especially in the Northern hemisphere (Neubauer et al 2004; Perkins 2009). Reports of equine neurological disease in New Zealand are rare and no cases have been attributed to the neuropathogenic strain of EHV-1. It is assumed that the neurological disease syndrome is caused by an exotic neuropathogenic strain of EHV-1 and that reports of EHV-1 in camelids could indicate the presence of that exotic neuropathogenic strain. EHV-1 is therefore considered to be a potential hazard in the commodity.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

Acutely infected camelids do not necessarily show obvious clinical signs of disease (Rebhun et al 1988; House et al 1991). However, as with horses, camelids recovering from EHV-1 infection are considered likely to become long-term latent carriers of the virus.

Therefore, animals that meet the commodity definition may be infected with exotic strains of EHV-1, and the likelihood of introduction is assessed to be non-negligible.

13.2.2. Exposure assessment

Assuming that camelids recovering from acute infection are likely to become long-term latent carriers of the virus, it is considered that such animals would not show signs of infection and would not excrete the virus except when it is reactivated due to stress or steroid treatment. Shedding of the virus would be primarily in nasal secretions (Allen et al 2004; Radostits et al 2007).

Any susceptible animals (other camelids and horses) exposed to imported animals that are shedding virus are likely to become infected, allowing establishment of potentially exotic strains of EHV-1.

Exposure and establishment is therefore assessed to be non-negligible.

13.2.3. Consequence assessment

Equine herpes viruses predominantly affect equids. The literature is sparse of natural disease outbreaks occurring in camelids. However, severe disease in camelids does result from infection.

Infection by EHV-1 is normally characterised by a primary respiratory tract disease of varying severity that is related to age and immunological status of the infected animal. Severe neurological clinical signs, including death can occur in the infected camelid or equid. Since it is not a zoonotic disease there are no consequences for human health.

Consequences are assessed to be non-negligible.

13.2.4. Risk estimation

Since entry, exposure and consequence assessments for the introduction of exotic neurovirulent strains are non-negligible, the risk is assessed to be non-negligible and EHV-1 is classified as a hazard in the commodity. Therefore, risk management measures may be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

The following points were considered when drafting options for the effective management of exotic EHV-1 in the commodity.

- There are no treatments or vaccines that can be used to eliminate infections from carrier camelids.
- There are no practical measures that allow latently infected camelids to be identified. Therefore, it is not possible to restrict the importation of camelids to non-infected animals.
- Requiring the premise of origin to have had no clinical cases is probably the only practical measure available to reduce the likelihood of imported camelids harbouring the virus.
- There are no OIE-prescribed tests for equine rhinopneumonitis, however the virus neutralisation test is listed in the *Manual* as an alternative test.

- The *Code* does not discuss strains of equine herpes viruses and considers them only under the clinical syndrome 'equine rhinopneumonitis'. The *Code* makes recommendations for the safe trade in equids, but not camelids.
- The *Code* recommends that equids should comply with the following:

Article 12.9.2.

Recommendations for the importation of equines

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

1. showed no clinical sign of equine herpes virus type 1 infection (abortigenic and paralytic forms) on the day of shipment and during the 21 days prior to shipment;
2. were kept for the 21 days prior to shipment in an *establishment* where no *case* of equine herpes virus type 1 infection (abortigenic and paralytic forms), was reported during that period.

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

1. Camelids could be imported without measures for EHV-1.

N.B: This recognises that camelids appear to be rarely affected by EHV-1 and are not implicated in the international spread of the virus.

2. Camelids could be imported provided they comply with the *Code* recommendations for equine rhinopneumonitis.

N.B. This implies that, although there are no international standards that are directly applicable to camelids, it is reasonable to apply the *Code* chapter on equine rhinopneumonitis since they are a susceptible species.

3. Camelids could be subjected to a virus neutralisation test during PEQ with negative results.

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14. Foot and mouth disease virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Picornaviridae*; Genus: *Aphthovirus*; Species: *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 (Stanway et al 2005).

14.1.2. OIE list

Listed as a disease of multiple species.

14.1.3. New Zealand status

Listed on the Unwanted Organisms Register as an exotic notifiable disease.

14.1.4. Epidemiology

Extensive reviews on foot and mouth disease are available (Sanson 1994; Thomson & Bastos 2004) and much of the information given below is taken from these. The disease has been eradicated from, or has not occurred in, countries relevant to this risk analysis except for South American countries. It can infect all cloven hoofed animals and is considered to be 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.

Infected animals excrete the virus in saliva, faeces, urine, milk, semen, ocular and nasal discharges (Sanson 1994; Thomson & Bastos 2004), and virus is also discharged in aerosol in expired air. The incubation period is usually 2-14 days (Sanson 1994). Viraemia usually continues from 1 day before until 11 days after signs of disease first appear. Transmission can be from direct contact, contact with infected fomites, ingestion of infected animal products or from inhaling aerosolized virus (Sanson 1994; Thomson & Bastos 2004). Long- term carriers excrete small amounts of virus from the pharynx. Cattle may excrete virus in this way for up to 3 years. However, the amount of virus excreted by persistent carriers is low and the ability of persistently infected cattle to spread the disease is controversial (Thomson & Bastos 2004).

Camelids are susceptible to foot and mouth disease. However, several investigations indicate that they are much more resistant to the infection than cattle, carry the virus for only short periods and are not highly infectious (Fowler 1992; David et al 1994; Viera et al 1995; Wernery & Kaaden 2002; Wernery & Kaaden 2004). According to Fowler (1992) Mancini infected South American camelids experimentally and was able to transmit the disease by injection into the epidermis of the tongue, intramuscular and intravenous injection and by cohabitation. The first lesions developed 48-72 hours after infection. Some llamas infected intramuscularly or intravenously died but transmission by cohabitation was not always successful. It was concluded that camelids are less susceptible than cattle and sheep. In another study only two out of 10 llamas developed slight lesions when placed in contact with infected pigs and infected llamas carried the virus in the oesophageal-pharyngeal region for only short periods compared to cattle (David et al

1993). According to Fowler (1992), Lubroth demonstrated that the virus could be transmitted from cattle to llamas and vice versa, but could not be isolated from llamas 14 days after infection.

Inactivated virus vaccines are available. However, vaccination may mask clinical infections while animals may still be carrying virus. In addition, the efficacy of vaccination in camelids is not known. The OIE recognises freedom where vaccination is practised, but countries must cease vaccination for at least 12 months and not have introduced vaccinated animals before qualifying for inclusion in the list of FMD free countries where vaccination is not practised (OIE 2009).

14.1.5. Hazard identification conclusion

Foot and mouth disease is a debilitating highly contagious disease and the virus is an exotic, notifiable organism. Camelids can be infectious for short periods. Therefore, the virus is considered to be a potential hazard in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Animals from infected countries (some South American countries) and those that can not comply with the definition for country freedom without vaccination in the *Code* could be carrying the organism and could be infectious at the time of importation. The likelihood of entry in the commodity is therefore assessed to be non-negligible.

14.2.2. Exposure assessment

Infected animals are contagious and excrete virus in all body discharges and in aerosols. They could infect animals they are in contact with, or via contaminated fomites. Long distance transmission by aerosols is probably unlikely in the case of camelids. However, if camelids were to infect pigs, the pigs could spread the virus over long distances by aerosols (Gloster et al 1982). Therefore, the likelihood of exposure is assessed to be non-negligible.

14.2.3. Consequence assessment

Animals that become infected could become the focal point for a serious outbreak of foot and mouth disease. Such an outbreak would cause serious disruption to the livestock industries, economic losses to individual farmers, very large expenses for an eradication campaign, and severe disruption to export markets for both animals and animal products. The overall effects could be catastrophic as demonstrated dramatically 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). A scenario analysis of the likely macroeconomic impacts of a limited FMD outbreak in New Zealand estimates that the cumulative loss in nominal gross domestic product to be around $6 billion after 1 year, and around $10 billion after 2 years. Further, it is considered likely that there would be a large initial drop in the dollar (around 20 %) with the exchange rate expected to remain below the baseline for at least 2 years (The Reserve Bank of New Zealand and the Treasury 2003).

Foot and mouth disease infection of humans is extremely rare and of negligible 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 virus does not infect birds so there would be no consequences for native species.

14.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible for animals from countries where foot and mouth disease occurs, the risk is assessed as non-negligible and FMDV is classified as a hazard in the commodity. Therefore, risk management measures may be justified.

14.3. RISK MANAGEMENT

14.3.1. Options

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

- Foot and mouth disease is an economically devastating disease.
- Vaccination and treatment are not options for the effective control of the virus in the commodity.
- Long-term carriers of virus occur in cattle and although camelids are considered to carry the virus for much shorter periods than cattle the length of time for which they can remain infected has not been adequately defined.
- For the purposes of the *Code* chapter relating to foot and mouth disease, ruminants include animals of the family of Camelidae. The *Code* chapter relating to ruminants and therefore Camelidae is covered in Articles relating to the importation of animals from infected and free countries or zones, with or without the practice of vaccination. The options listed include the wording taken directly from the *Code*.

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

1. Consistent with Article 8.5.10. of the *Code*, camelids from FMD free countries or zones where vaccination is not practised could be required to:

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

   1. showed no clinical sign of FMD on the day of shipment;
   2. were kept since birth or for at least the past 3 months in a FMD free country or *zone* where vaccination is not practised;
   3. have not been vaccinated.

2. Consistent with Article 8.5.11. of the *Code*, camelids from FMD free countries or zones where vaccination is practised could be required to:

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

1.showed no clinical sign of FMD on the day of shipment;
2.were kept in an FMD free country or *zone* since birth or for at least the past 3 months; and
3.have not been vaccinated and were subjected, with negative results, to tests for antibodies against FMD virus, when destined to an FMD free country or *zone* where vaccination is not practised.

3. Consistent with Article 8.5.12. of the *Code*, camelids from FMD infected countries or zones could be required to:

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of FMD on the day of shipment;
2.were kept in the *establishment* of origin since birth, or
a. for the past 30 days, if a *stamping-out policy* is in force in the *exporting country*, or
b. for the past 3 months, if a *stamping-out policy* is not in force in the *exporting country*,
and that FMD has not occurred within a ten-kilometre radius of the *establishment* of origin for the relevant period as defined in points a) and b) above; and
3.were isolated in an *establishment* for the 30 days prior to shipment, and all animals in isolation were subjected to diagnostic tests (probang and serology) for evidence of FMDV *infection* with negative results at the end of that period, and that FMD did not occur within a ten-kilometre radius of the *establishment* during that period; or
4.were kept in a *quarantine station* for the 30 days prior to shipment, all animals in quarantine were subjected to diagnostic tests (probang and serology) for evidence of FMDV *infection* with negative results at the end of that period, and that FMD did not occur within a ten-kilometre radius of the *quarantine station* during that period;
5.were not exposed to any source of FMD *infection* during their transportation from the *quarantine station* to the *place of shipment*.

4. Importation of camelids from countries that are infected with foot and mouth disease or where vaccination is practised could be prohibited. Therefore animals only from countries that are on the OIE list of FMD free countries where vaccination is not practised would be eligible for export.

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15. Louping-ill virus

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological identification

Family: *Flaviviridae*; Genus: *Flavivirus*; Species: *Louping ill virus*. British, Irish, Spanish and Turkish subtypes are recognised (Thiel et al 2005).

The viruses causing tick-borne encephalitis (TBE) are a closely related group of viruses including the agents of Louping-ill, Central European TBE, Far Eastern TBE, Omsk haemorrhagic fever in Siberia, Kyasanur Forest disease in the Indian subcontinent, Langat in Malaysia, Negishi in Japan, Powassan in North America and parts of Russia, and four viruses from Asia that have no known veterinary or medical significance (Gresikova & Beran 1981; Korenberg & Kovalevskii 1999; Gilbert et al 2000).

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Listed as an unwanted organism (MAF 2009).

15.1.4. Epidemiology

Louping-ill is a tick-borne disease that occurs in the United Kingdom. Closely related viruses cause tick-borne encephalitis in various parts of Europe and the USA (Powassan virus).

Louping-ill in the United Kingdom is primarily a disease of sheep but other species can be infected. It has been suggested that at least 32 vertebrate species and a wide variety of ticks can be infected with louping-ill virus (Reid 1990). The TBE viruses that occur in Eastern Europe and Russia are primarily pathogens of humans. In Russia, 11,000 cases occur annually and another 3,000 cases occur in the rest of Europe (Gritsun et al 2003a).

In animals, transmission of TBE viruses is entirely by ticks (Gresikova & Beran 1981). The main tick vectors are *Ixodes ricinius* and *Ixodes persulcatus*, although other tick species may also be involved (Gresikova & Beran 1981; Korenberg & Kovalevskii 1999). Antibody has been demonstrated in, or virus has been isolated from, a wide range of animals including small rodents, wildlife, and domestic animals such as deer and cattle (Swanepoel & Laurenson 2004). TBE viruses of Russia and Eastern Europe are believed to be sustained mainly in a tick/small mammal cycle, although transovarial transmission through multiple generations of ticks also occurs (Gresikova & Beran 1981). In the case of louping-ill, small mammals are probably of lesser importance in maintaining the virus (Gilbert et al 2000). In sheep, louping-ill has an incubation period of 2-5 days. In experimental infection of sheep and goats viraemia lasts 1-5 days and shedding of virus in milk 2-7 days (Gresikova & Beran 1981).

The disease has been described in camelids in the United Kingdom (Macaldowie et al 2005; Cranwell et al 2008). Detailed studies in this species have not been carried out and information must be extrapolated from data from sheep.

15.1.5. Hazard identification conclusion

Animals that are infected with louping ill and their related viruses are not contagious. These diseases are transmitted by tick species that do not occur in New Zealand. Therefore, louping-ill and related viruses in camelids are not considered to be hazards in the commodity.

N.B. measures to manage the risks of introducing ticks associated with the commodities are discussed in the ticks section.

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16. Rabies virus

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Family: *Rhabdoviridae*; Genus: *Lyssavirus*; Species: *Rabies virus* (Tordo et al, 2005).

16.1.2. OIE list

Listed as a disease of multiple species.

16.1.3. New Zealand status

Listed as an exotic notifiable disease (MAF 2009).

16.1.4. Epidemiology

Rabies is a disease of all mammals including camelids (Fowler 1992; Wernery & Kaaden 2002) and humans. It is characterised by severe neurological signs and is invariably fatal.

Rabies occurs widely around the world but there are a number of countries that are free, mainly island and peninsular countries.

In all endemically infected countries rabies virus is maintained in a population of domestic or wild carnivores or bats. True rabies in bats is confined to the Americas (Swanepoel 2004). In South America vampire bats are important vectors (Swanepoel 2004). Infections of bats with related lyssaviruses occur in Europe (Fooks et al 2003), Africa (Swanepoel 2004) and Australia (Thompson 1999). Australia, the United Kingdom and some European countries are free from rabies, but it occurs in both North and South America.

The virus is carried mainly by carnivores. In the final stages of the disease they excrete the virus in their saliva and transmit it to other animals when they bite them. Other forms of transmission such as aerosol transmission in bat colonies (Swanepoel 2004) and *per os* infection of kudu (Hubschle 1988) are rare exceptions. Following deposition in a bite wound the virus enters peripheral nerves and is transported through the nerves to the central nervous system. After entering the peripheral nerves the virus is not found in any other body tissues or in the blood. Amputation of limbs of mice experimentally infected in the foot pads has been shown to prevent the virus from progressing to the brain (Swanepoel 2004). The passage of virus through the nervous system is slow 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. The occurrence of viraemia is an exceptional event other than in experimental infections of young mice with large doses of virus (Swanepoel 2004).

The virus spreads to the salivary glands at about the stage that there is generalised dissemination of infection in the brain. It then multiplies in the salivary glands and is excreted in the saliva. The *Code* states that “For the purposes of the *Terrestrial Code*, the *incubation period* for rabies shall be 6 months, and the *infective period* in domestic carnivores starts 15 days before the onset of the first clinical signs and ends when the animal dies”. Typically, animals become ataxic and aggressive, or develop a paralytic form of the disease (Radostits et al 2007).

Outbreaks of the disease have occurred in camelids (CDC 1990; Fowler 1992; Wernery & Kaaden 2002). In one outbreak, in which 20 alpacas were bitten by a rabid dog, 13 died or were euthanised. In these cases the incubation period varied from 15-34 days and animals died within 8 days of developing clinical signs. In another outbreak involving 29 cases the incubation period was from 15-30 days (Fowler 1992). In view of the protracted incubation periods sometimes seen in other animals it must be assumed those incubation periods of longer than 3 months are possible. Herbivores are almost invariably dead-end hosts as they seldom bite other animals.

Killed vaccines are safe for use in camelids although their efficacy is unproven. Modified live vaccines have been reported to cause post-vaccine paralysis in 10 % of cases (Fowler 1992).

16.1.5. Hazard identification conclusion

Rabies virus can infect camelids and causes an invariably fatal disease. Therefore, rabies virus is considered to be a potential hazard in the commodity.

16.1.6. Entry assessment

Camelids could be incubating the disease when imported. Incubation periods may be protracted and during this time animals show no signs of infection. Since the disease occurs in several countries relevant to this risk analysis, the likelihood of entry is assessed to be non-negligible.

16.1.7. Exposure assessment

Camelids are likely to be dead-end hosts and therefore exposure of other animals is unlikely. However, since there is a remote chance of transmission to animal attendants working with the camelids the likelihood of exposure is assessed to be non-negligible.

16.1.8. Consequence assessment

Establishment of rabies in New Zealand would be unlikely unless a carnivore became infected. The likelihood that a carnivore would be bitten by a camelid or have a wound infected with camelid saliva is extremely low. Humans working with an infected animal could possibly be infected by contamination of a wound with saliva. Since the untreated disease is invariably fatal the consequences are assessed to be non-negligible.

16.1.9. Risk estimation

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

16.2. RISK MANAGEMENT

16.2.1. Options

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

- Rabies is a serious zoonotic disease which is invariably fatal in untreated people. Post-exposure prophylactic treatment of humans exposed to rabies is expensive and stressful for the patient.
- There are no effective post-exposure prophylactic treatments that could be used in animals.
- The incubation period may be long and quarantine should be for a protracted period.
- Inactivated vaccines are safe but efficacy has not been proven for camelids.
- The *Code* makes the following recommendations that are relevant to the importation of camelids:

Article 8.10.2.

Rabies free country

A country may be considered free from rabies when:

1.the disease is notifiable;
2.an effective system of *disease surveillance* is in operation;
3.all regulatory measures for the prevention and control of rabies have been implemented including effective importation procedures;
4.no *case* of indigenously acquired rabies infection has been confirmed in man or any animal species during the past 2 years; however, this status would not be affected by the isolation of an Australian or European Bat Lyssavirus;
5.no imported *case* in carnivores has been confirmed outside a *quarantine station* for the past 6 months.

Article 8.10.3.

Recommendations for importation from rabies free countries

for domestic mammals, and wild mammals reared under confined conditions

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

1.showed no clinical sign of rabies on the day of shipment;
2.were kept since birth or for the 6 months prior to shipment in a rabies free country or were imported in conformity with the regulations stipulated in Articles 8.10.5., 8.10.6. or 8.10.7.

Article 8.10.6.

Recommendations for importation from countries considered infected with rabies

for domestic ruminants, equines and pigs

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

1.showed no clinical sign of rabies on the day of shipment;
2.were kept for the 6 months prior to shipment in an *establishment* where separation from wild and feral animals was maintained and where no *case* of rabies was reported for at least 12 months prior to shipment.

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

1. Animals could be imported provided that they:
   i. showed no clinical sign of rabies on the day of shipment; and

ii. were kept since birth or for the 6 months prior to shipment in a rabies free country.

N.B. This is equivalent to the *Code* recommendations made in Article 8.10.3.

2. Animals from countries in which rabies occurs could be imported provided that they:

   i. showed no clinical sign of rabies on the day of shipment; and
   ii. were kept for the 6 months prior to shipment in an establishment where separation from wild and feral animals was maintained and where no case of rabies was reported for at least 12 months prior to shipment.

N.B. This is equivalent to *Code* Article 8.10.6.

3. In addition to measures equivalent to *Code* Article 8.10.6. as in option 2 above, the animals could be vaccinated with an inactivated rabies vaccine at least 6 months prior to shipment.

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17. Vesicular stomatitis virus

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Family: *Rhabdoviridae*; Genus: *Vesiculovirus*; Species: *Vesicular stomatitis virus* (Tordo et al 2005). There are two main types; Indiana and New Jersey. Indiana has three sub-types and New Jersey contains only a single sub-type.

17.1.2. OIE list

Listed as a disease of multiple species.

17.1.3. New Zealand status

An exotic, notifiable disease.

17.1.4. Epidemiology

The disease occurs in horses, cattle and pigs and, more rarely, in sheep and goats (Swenson 2008; CFSPH 2009). Naturally occurring infections in South American camelids are rare (APHIS 2007; Bridges et al 1995; Schmidtmann et al 1999). Gomez (1964) infected camelids by injection into the dorsum of the tongue (Fowler 1992).

In addition to being a virus of vertebrates, the virus has also been shown to multiply in insects such as blackflies (*Simulium* spp.), sandflies (*Lutzomyia* spp.), mosquitoes (*Aedes aegypti*) and leafhoppers (*Peregrinus maidis*) (Mare & Mead 2004).

Vesicular stomatitis (VS) is important mainly because it is clinically indistinguishable from foot and mouth disease (Sellers & Daggupaty 1990; Rodriguez 2002; Mare & Mead 2004). Therefore, initial diagnosis of the disease before laboratory confirmation of the viral aetiology may trigger the massive initial response usually reserved for foot and mouth disease. Alternatively, if an outbreak of foot and mouth disease is incorrectly assumed to be VS, as occurred in Saskatchewan in 1951, the response to the foot and mouth disease outbreak can be delayed (Sellers & Daggupaty 1990).

The disease is endemic in Central and South America and thousands of outbreaks occur each year from southern Mexico to northern South America (Rodriguez 2002). In the USA the disease occurs sporadically in some southern states but is endemic in at least one location in Georgia (Stallknecht 2000). During 2009, 5 outbreaks occurred affecting horses resident in New Mexico and Texas (OIE 2009). In some seasons the disease spreads northward along riverbeds into northern locations in the USA (Schmidtmann et al 1999) and even as far as Canada (Wilks 1994).

Despite the large numbers of livestock exported from North America, the disease has only been reported outside the Americas on one occasion and this was in a large consignment of horses exported from North America to France during the First World War. The disease failed to establish in Europe (Mare & Mead 2004).

The most commonly held view is that the virus is transmitted by an insect vector. Virus has been isolated from the sand fly *Lutzomyia shannoni*, which is the most likely vector (Braverman 1994; Comer et al 1994; Rodriguez et al 1996; Schmidtmann et al 1999;

Stallknecht 2000). *Culicoides* spp. are also possible vectors and have been infected experimentally (Nunamaker et al 2000). Blackflies (*Simulium* spp.) have also been incriminated in the transmission of the disease (Mead et al 2000). The virus can also be transmitted by teat cups during milking of cows with teat lesions or by infection of wounds and abrasions (Wilks 1994).

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

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

The incubation period is 1-3 days (Wilks 1994), but for the purposes of international trade a period of 21 days is recommended by the *Code*.

Lesions on teats and feet are primary lesions caused by entry of the virus directly at these sites (Wilks 1994). Similarly, in experimental infection of pigs, lesions occurred at the injection sites but there was no viraemia (Howerth et al 1997). In a description of the pathogenesis of the disease it is stated that virus replicates in the lower layers of the epidermis and there is no description of viraemia (Mare & Mead 2004). Mead states that viraemia does not occur in mammalian hosts but demonstrated transmission of the virus to non-infected blackfly when infected and non-infected blackfly co-fed on the same host (Mead et al 2000). If viraemia does not occur in mammals, introducing the disease through trading clinically healthy animals would not be possible. This may account for the failure of the disease to spread beyond the Americas.

Serotype specific antibody develops within 5-8 days of infection. Blocking and competitive ELISAs, virus neutralisation and complement fixation tests are recommended for international trade (Swenson 2008).

17.1.5. Hazard identification conclusion

VS virus is an important exotic pathogen of cattle, horses and pigs and more rarely of other animals including llamas and alpacas. Therefore, it is considered to be a potential hazard in the commodity.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

There is a considerable body of opinion that suggests that viraemia does not occur in VS. Despite this, it is has been stated that “subclinical infection is frequent and subsequent excretion of the virus can occur with no clinical signs” (Mare & Mead 2004). The *Code* suggests that the incubation period for international trade “shall be 21 days” and recommends that a quarantine period of 30 days should be imposed on animals for export from infected countries. As many facts relating to the transmission, pathogenesis and excretion of the virus remain unknown for South American camelids, it is prudent to assume that llamas and alpacas could introduce the virus to New Zealand, while in the

incubation period of the disease. Therefore, the likelihood of entry of virus in the commodity is assessed to be low but non-negligible.

17.2.2. Exposure assessment

Infected animals introduced into New Zealand could transmit the virus to other animals through contact exposure involving minor abrasions of the oral mucosa or skin. However this would be an inefficient method of transmission and is unlikely to lead to establishment of the disease. The disease has never spread outside of the Americas, suggesting that there are factors unique to endemic regions that are necessary for the establishment of the disease. The disease is transmitted primarily by insect vectors. Whether any competent vectors occur in New Zealand is not known. However, blackflies, commonly known as 'sandflies' belonging to the family *Simuliidae* are present. Therefore, the likelihood that insect vectors in New Zealand could become infected and transmit the disease to naïve animals in New Zealand is assessed to be very low but non-negligible.

17.2.3. Consequence assessment

If the virus became established in competent vectors in New Zealand, sporadic cases of disease would be likely in animals, resulting in confusion with foot and mouth disease. Expensive control procedures normally reserved for cases of foot and mouth disease might be activated. There would also be losses due to interference with trade at least until foot and mouth disease could be ruled out. Individual farmers would also incur costs due to production losses.

The virus can cause disease in humans, as a result of direct contact or laboratory accidents (Wilks 1994; Letchworth et al 1999; Swenson 2008). Many people in endemic areas have antibody against the virus. Most cases of the disease go undiagnosed as the symptoms are similar to influenza. It is likely that the establishment of the disease in New Zealand would result in sporadic infections in humans during outbreaks of disease in livestock.

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

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

17.2.4. Risk estimation

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

17.3. RISK MANAGEMENT

17.3.1. Options

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

- VS is a rare disease of camelids.
- Quarantine in insect-free facilities for periods exceeding the incubation and viraemic periods could be effective.
- Conditional on a suitable quarantine period with protection from insect vectors, seropositive animals demonstrating a stable or declining titre could be safely imported. This is because viraemia has not been demonstrated in infected animals and there is no evidence for a carrier state in recovered animals (Pharo 1999).
- Australia, Canada and EU countries are free from the virus. The disease has never established outside of the Americas.
- Serological tests recommended by the OIE are available for diagnosis.
- There are no treatments or vaccines.
- The relevant sections from the *Code* are given below:

Article 8.15.2.

VS free country
A country may be considered free from VS when:
1.VS is notifiable in the country;
2.no clinical, epidemiological or other evidence of VS has been found during the past 2 years.

Article 8.15.4.

Recommendations for importation from VS free countries
for domestic cattle, sheep, goats, pigs and horses
Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of VS on the day of shipment;
2.were kept in a VS free country since birth or for at least the past 21 days.

Article 8.15.6.

Recommendations for importation from countries considered infected with VS
for domestic cattle, sheep, goats, pigs and horses
Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of VS on the day of shipment;
2.were kept, since birth or for the past 21 days, in an *establishment* where no *case* of VS was officially reported during that period; or
3.were kept in a *quarantine station* for the 30 days prior to shipment and were subjected to a diagnostic test for VS with negative results at least 21 days after the commencement of quarantine;
4.were protected from insect vectors during quarantine and transportation to the *place of shipment*.

N.B. The *Code* Article 8.15.6 offers the possibility for infected countries to provide certification similar to that recommended for VS free countries (by meeting points 1 and 2 only of Article 8.15.6.). There is no requirement for animals to be

quarantined with protection from insect vectors and diagnostic testing. Further, the infected country is not required to certify that no clinical, epidemiological or other evidence of VS has been found during the previous 2 years. This apparent difference in stringency is a reflection of the fact that while VS may be present, even widespread, in some countries, there are regions within endemically infected countries where the disease is absent. It is possible, therefore, for an establishment to have a good record of VS freedom.

Finally, the *Code* recommends that animals be subjected to a diagnostic test for VS with negative results. Serologically positive animals would not be eligible for importation.

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

1. Since natural infections are rare in camelids, it could be considered that no measures are necessary.
2. Animals from VS free countries could be imported provided the disease is notifiable and that no clinical, epidemiological or other evidence of VS has been found during the past 2 years; and
    i. showed no clinical sign of VS on the day of shipment; and
    ii. were kept in a VS free country since birth or for at least the past 21 days.

    N.B. This is equivalent to the *Code* Article 8.15.2.
3. Animals from VS infected countries could be imported provided that they:
    i. showed no clinical sign of VS on the day of shipment; and
    ii. were kept, since birth or for the past 21 days, in an establishment where no case of VS was officially reported during that period; and
    iii. were protected from insect vectors during quarantine and transportation to the place of shipment.

    N.B. These requirements are equivalent to those in the *Code* Article 8.15.6. (VS infected country) except that no serological test is required.
4. Animals from VS infected countries could be imported provided that they:
    i. showed no clinical sign of VS on the day of shipment; and
    ii. were kept, since birth or for the past 21 days, in an establishment where no case of VS was officially reported during that period; or
    iii. were kept in a quarantine station for the 30 days prior to shipment and were subjected to a diagnostic test for VS with negative results at least 21 days after the commencement of quarantine; and
    iv. were protected from insect vectors during quarantine and transportation to the place of shipment.

    N.B. These requirements are equivalent to those recommended in the *Code*, Article 8.15.6.

5. Camelids could be subjected to a diagnostic test for VS during the PEQ period and be protected from insect vectors. In the case of any positive result, all camelids could be re-tested not less than 14 days subsequently. The results of testing could indicate that all camelids have negative, stable or declining titres.

   N.B. This testing option allow serologically positive animals to be eligible for importation, reflecting IHS requirements for horses.

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18. West Nile virus

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

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

18.1.2. OIE list

West Nile fever is a listed disease of multiple species.

18.1.3. New Zealand status

An exotic organism that is not listed as unwanted or notifiable by MAF (2009).

18.1.4. 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 subsequently to 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 at the height of the US epidemic in 2003, 9,862 cases were reported and there were 264 deaths (CDC 2009a).

The virus is transmitted by mosquitoes and maintained in a mosquito/bird cycle (CDC 2009b). At least 43 species of mosquitoes have been suspected of acting as vectors of the virus (Gingrich & 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). Animals other than birds may be infected. Clinical cases are seen predominantly in horses in which the infection proves fatal in about 40 % of cases, but many horses are subclinically infected and develop antibody. However, in viraemic horses the virus titre is too low to infect mosquitoes.

According to CDC " People, horses, and most other mammals are not known to develop infectious-level viremias very often, and thus are probably 'dead-end or incidental-hosts' (CDC 2009b). Cattle are also dead-end hosts (Ilkal et al 1988). Particularly relevant is the *Code* statement Article 8.16.2. that "Members should not impose trade restrictions on dead-end hosts such as horses". The *Code* chapter makes recommendations for other susceptible species (birds) and specifically *excludes* horses from them (Articles 8.16.5., 8.16.6. and 8.16.7.). The *Code* also states that a free country or zone will not lose its free status through the importation of seropositive animals whether from natural infection, or vaccination induced (OIE 2009).

Fatal cases of the disease have been seen in alpacas (Dunkel et al 2004; Kutzler et al 2004b; Yaeger et al 2004). Alpacas and llamas produced antibody to the virus after vaccination. The vaccine proved to be safe but no challenge tests were done to prove immunity (Kutzler et al 2004a). It is highly likely that camelids will be dead-end hosts like horses, cattle and other mammalian species.

18.1.5. Hazard identification conclusion

West Nile virus is transmitted by mosquitoes and maintained in a mosquito/bird cycle. Mammals are dead-end hosts. Therefore, the likelihood that the virus would be introduced into New Zealand in imported camelids is considered to be negligible and the virus is not considered to be a hazard in the commodity.

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19. *Anaplasma phagocytophilum*

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Anaplasma phagocytophilum (synonym *Ehrlichia phagocytophilum*) is a gram-negative bacterium that is an obligate intracellular blood parasite of neutrophils. It is very closely related to *Ehrlichia equi* (Hulinska et al 2004) and may be the same species. Some authors refer to a genogroup of similar species (Barlough et al 1997a; Barlough et al 1997b).

19.1.2. OIE list

Not listed.

19.1.3. New Zealand status

Ehrlichia spp. are listed as exotic unwanted organisms (MAF 2009).

19.1.4. Epidemiology

Re-organisation of the taxonomy of the organisms in the family *Anaplasmataceae* as a result of new information on their genomic structure has resulted in significant changes in their classification (Dumler et al 2001). The family now contains four genera, *Ehrlichia*, *Anaplasma*, *Neorickettsia*, and *Wolbachia*. Changes to names and classification of the organisms in this group have been ongoing for several years. Uilenberg has suggested that classification of organisms based only on partial gene sequences may lead to misclassification of some species (Uilenberg et al 2004). The recent name changes have been summarised in an article on the internet (Anonymous undated). It has been suggested that knowledge about the *Ehrlichia* spp. is inadequate and that many new species may be found in the future. Attention has also been drawn to the problem of "perpetuation of many doubtful species names" (Sumption & Scott 2004).

This section is restricted to *Anaplasma phagocytophilum*. The genus and species names used in this chapter are those used by the authors of the articles referred to above.

A. phagocytophilum is the agent of tick-borne fever in animals and human granulocytic ehrlichiosis (McQuiston et al 2003; Grzeszczuk et al 2004). It has a world-wide distribution. In Europe the organism primarily affects young cattle and sheep and usually runs a mild course and inapparent infections occur. In the USA it causes subclinical infections in cattle and is the predominant cause of ehrlichiosis in horses (McQuiston et al 2003). Experimental infections with *A. phagocytophilum* in sheep and goats caused a non-fatal disease (Gokce & Woldehiwet 1999a). Infections with *A. phagocytophilum* may make animals more susceptible to concurrent infections with other organisms (Woldehiwet 1983; Gokce & Woldehiwet 1999b). Infected animals may carry the infection for 2 years (Woldehiwet 1983). In the USA the vectors are *Ixodes scapularis and Ixodes spinipalpis,* while in Europe the main vector is *Ixodes ricinus* (Alberdi et al 1998; Telford et al 2002; McQuiston et al 2003). *A. phagocytophilum* DNA was identified in *Haemaphysalis longicornis* from Korea but the report does not confirm that the tick can transmit the organism (Kim et al 2003). It has been suggested that although natural infection of several genera of ticks by single species of *Ehrlichia* occurs, infected species of ticks may not

necessarily be competent vectors, and each species of *Ehrlichia* is transmitted by a single genus of competent ticks only (Sumption & Scott 2004). Therefore, it seems likely that the competent vectors of *A. phagocytophilum* are *Ixodes* spp. and that *Haemaphysalis* spp. are not competent vectors.

Antibody can be detected by immunofluorescent antibody tests (Petrovec et al 2002; Zeman et al 2004). Organisms can be detected by microscopic examination of bloodsmears or by conventional or real time PCR tests (Ahrens et al 2003; Courtney & Massung 2003; Courtney et al 2004; Hulinska et al 2004).

A single case of *A. phagocytophilum* infection has been described in a llama. In this case the vector was apparently *Ixodes pacificus* (Barlough et al 1997a). Humans and domestic animals are regarded as largely accidental hosts of an organism that is maintained in wildlife hosts (McQuiston et al 2003). Therefore, this case is assumed to be a very rare incident of disease in an accidental host.

19.1.5. Hazard identification conclusion

Ehrlichiosis caused by *A. phagocytophilum* in camelids is considered to be a rare disease in an accidental host. In addition, the main vectors for the organism are *Ixodes* spp. ticks that are not present in New Zealand. Therefore, *A. phagocytophilum* is not considered to be a hazard in the commodity.

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20. *Bacillus anthracis*

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Bacillus anthracis is an aerobic, spore-forming bacillus that causes the disease anthrax.

20.1.2. OIE list

Listed as a disease of multiple species.

20.1.3. New Zealand status

The last case of anthrax occurred in 1954 (Gill 1992). It is an unwanted, notifiable organism (MAF 2009).

20.1.4. Epidemiology

Anthrax is a bacterial disease of most warm-blooded vertebrates including camelids. It is primarily a natural disease of herbivores which are most susceptible, followed by humans and pigs. Carnivores such as the dog and cat are resistant to infection (Langston 2005; Coker 2008). The disease has occurred in recent years in many countries including Australia, Canada, the EU, the USA and many South American countries (OIE 2009).

The infectious agent is a spore forming bacillus that can survive in the spore state in suitable soils for many decades. In 1999 an outbreak occurred in Australia on farms where the disease had not occurred for about 100 years. On these properties earthworks in relation to an irrigation scheme possibly resulted in disturbance of old burial sites of cattle (Turner et al 1999a; Turner et al 1999b).

Bacillus anthracis is probably an obligate pathogen that multiplies only in animals, and if an infected carcass is opened, it sporulates resulting in contamination of soil and the environment. In unopened carcasses the organism does not sporulate and is destroyed by putrefaction (De Vos & Turnbull 2004). The disease is not directly transmissible from animal to animal and infection is believed to be associated with ingestion of soil or other infected material that is contaminated with spores. Biting flies may carry the infection but they were not considered to be important in the transmission of the disease in an outbreak in Australia (Turner et al 1999a). Blowflies may be important in the spread of the disease when they have been feeding on infected carcasses (De Vos & Turnbull 2004). Infection through skin wounds and abrasions may also occur and is a common route of infection for humans (De Vos & Turnbull 2004). In some circumstances human infection can occur by inhalation (so-called woolsorter's disease) but inhalation is not likely to be of importance in camelids.

The incubation period probably ranges from 1-14 days. In the peracute form in susceptible species, animals may die without showing signs. In other cases animals may die in 1-3 days after developing subcutaneous swellings on various parts of the body (Fowler 1998). Efficient live spore vaccines are available for control of the disease. The vaccine strain developed by Sterne (Sterne 1937) is used for most animals including camelids. It is a rough strain that has lost plasmid pX02 which codes for the bacterial capsule. The vaccine is non-pathogenic in most animal species and provides good immunity for about a year (De

Vos & Turnbull 2004). However, one report attributes the death of two crias (young unweaned camelids) to the vaccine (Cartwright et al 1987).

20.1.5. Hazard identification conclusion

Anthrax is known to occur in camelids and the disease occurs in all countries relevant to this risk analysis. Therefore, *B. anthracis* is considered to be a potential hazard in the commodity.

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

Although anthrax is a rare disease it still occurs sporadically in a number of countries. The incubation period is short and camelids are susceptible, exhibiting obvious clinical signs of infection. Therefore, the likelihood of an imported animal being in the incubation period for the disease or carrying spores in its intestines is assessed to be very low but non-negligible.

20.2.2. Exposure assessment

The *Code* chapter states: "There is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs". An imported animal that is in the incubation period for the disease would be likely to die. In the extremely unlikely event that an infected camelid is imported and dies from anthrax, it is highly improbable that its carcass would extensively contaminate the environment. The original outbreaks of anthrax in New Zealand in the early 1900s resulted from the importation of thousands of tons of unsterilised animal bones that were applied to pastures as fertiliser (Barry 1954). Despite this widespread practice and several outbreaks, *B. anthracis* never became established. An imported case of anthrax would not contaminate the environment to the same extent.

However, other animals could become infected from contaminated soil or water, resulting in further spread of the organism and contamination of the environment. Therefore the risk of exposure is considered to be very low but non-negligible.

20.2.3. Consequence assessment

Control measures would be introduced in response to an outbreak. Quarantine and disinfection of infected areas and vaccination of animals would incur costs. In the long term, should the introduction lead to long term contamination of the environment, sporadic cases of anthrax could occur in humans and in animals. Therefore, the consequences of introduction are assessed as non-negligible.

20.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and *B. anthracis* is classified as a hazard in the commodity. Therefore, risk management measures may be justified.

20.3. RISK MANAGEMENT

20.3.1. Options

The following points were considered when drafting options for the effective management of *B. anthracis* in the commodity:

- Introduction of the organism would result in Government intervention to control and eradicate an outbreak.
- Rare sporadic cases of animal and human infection could occur.
- An efficient attenuated vaccine is available but there is some doubt about its safety when used in camelids.
- Both the incubation period and course of clinical disease are short. Therefore, quarantine would be an effective measure to prevent introduction of the disease agent.
- The recommendations made in the *Code* are for the safe trade in ruminants, equines and pigs, rather than camelids. Although there are no international standards that are directly applicable when trading camelids, it is reasonable to apply the *Code* chapter on anthrax for the same bacteria in other susceptible species. The *Code* Article relating to anthrax is given below:

Article 8.1.2.

Recommendations for the importation of ruminants, equines and pigs
Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of anthrax on the day of shipment;
2.were kept for the 20 days prior to shipment in an *establishment* where no *case* of anthrax was officially declared during that period; or
3.were vaccinated, not less than 20 days and not more than 6 months prior to shipment

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

1. Camelids to be imported showed no clinical sign of anthrax on the day of shipment.
2. Camelids to be imported could be quarantined for the 20 days before shipment where no case of anthrax has occurred.

   N.B. This measure more than covers the incubation period (1-14 days) and when combined with clinical freedom on the day of shipment provides an effective risk management option.

3. Camelids to be imported could be vaccinated with an attenuated live vaccine at least 20 days and not more than 6 months before shipment.

   N.B. Including a vaccination option is equivalent to those recommended in the *Code*, i.e. points 1 and 2 of Article 8.1.2., or 3. Additional measures beyond the *Code* are not necessary to suggest. However, if the vaccination option is applied to camelids, there may be associated health risks (including death) for these species.

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21. *Brucella* spp.

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

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

21.1.2. OIE list

Bovine (*B. abortus*), porcine (*B. suis*) and sheep and goat (*B. ovis* and *B. melitensis*) brucellosis are listed.

21.1.3. New Zealand status

B. abortus was eradicated from New Zealand by 1989 (Hellstrom 1991; Mackereth 2003). *B. melitensis* and *B. suis* are not present. *B. abortus*, *B. melitensis* and *B. suis* are unwanted, notifiable organisms (MAF 2009). *B. ovis* is endemic.

21.1.4. Epidemiology

Information on the global occurrence of *Brucella* species and brucellosis is available from the World Animal Health Information Database (OIE 2009). Bovine brucellosis formerly had a world-wide distribution but has now been eradicated from many developed countries. Canada and Australia are free from the disease. It still occurs, but at a low prevalence, in the USA and in some parts of the EU, Central and South America and Asia.

B. melitensis affects goats primarily but also sheep and occurs in some countries in Europe and South and Central America but not in Australia, the USA or Canada. *B. suis* occurs in some European, Asian, South and Central American countries and at a low prevalence in the USA. The last occurrence of *B. suis* in Australia was reported in 2004. The UK is free from all *Brucella* spp.

Brucellosis is a rare disease of camelids. Fowler (1998) reviewed a report in which an outbreak caused by *B. melitensis* is described. He also reviewed a paper describing the death of three llamas after exposure to camels recently imported from Russia. These animals had high antibody titres to *Brucella*. However, this report must be viewed with some scepticism since brucellosis never causes acute fatal infections in adults of other animal species. Nevertheless, Nielson (2008) reports brucellosis in camelids related to contact with large and small ruminants infected with *B. abortus* or *B. melitensis*. Llamas have been experimentally infected with *B. abortus*. The only pregnant llama in the exposed group aborted and the organism could be isolated from the placenta, foetus and various organs in the dam (Gidlewski et al 2000). Antibody was detected in llamas experimentally exposed to *B. abortus* S19 (vaccine strain) and strain 2308 (Gilsdorf et al 2001). Exposed camelids developed antibody that was detectable by a range of conventional serological tests (Gilsdorf et al 2001; Rojas et al 2004) but difficulties were experienced when using the complement fixation test due to anticomplementary activity of the sera (Rojas et al 2004).

No evidence was found of natural infection of camelids with *B. suis,* but since this organism is closely related to other *Brucella* spp. (*B. abortus* and *B. melitensis*) it is likely that it could be infectious for camelids. It is unlikely that *B. ovis* would be infectious for

camelids since it is found only in sheep and deer. In addition it occurs endemically in New Zealand.

Natural infections of camelids with *Brucella* species appear to be rare, since no descriptions of natural infections, other than those reported above could be found. In addition, antibodies were not found in serological surveys of South American camelids (Marin et al 2008; Rojas et al 2004).

There is no evidence to indicate that attenuated strain vaccines used for *B. abortus* or *B. melitensis* are safe or effective in camelids. There are no effective methods for treating brucellosis in animals.

B. abortus, *B. melitenis* and *B. suis* are zoonotic organisms that cause serious debilitating disease of humans. Humans can contract the disease by drinking unpasteurised milk or by contact with animals, or their infected discharges at parturition.

21.1.5. Hazard identification conclusion

Brucellosis is rare in camelids. However, since they are susceptible to experimental exposure and rare cases of natural infection have been reported (Nielsen 2008), it is possible for natural infections to occur. Therefore, *Brucella* spp. are considered to be potential hazards in the commodity.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Many countries from which camelids may be imported are free from brucellosis. Importation from these countries would not involve any risk. However, several relevant countries are infected with brucellosis. Since camelids are susceptible to experimental infection and natural infection has been reported, the likelihood of introducing the organism in imported camelids is non-negligible.

21.2.2. Exposure assessment

It is assumed that brucellosis in camelids is spread in a similar manner to cattle. Therefore, infected camelids could excrete the organism in vaginal discharges and milk after parturition or abortion. Animals in contact with them could become infected and this could lead to transmission of the disease to cattle (*B. abortus*) or sheep and goats (*B. melitensis*). Infection of pigs with *B. suis* is possible but less likely.

21.2.3. Consequence assessment

Once introduced, movement of animals could lead to the spread of the disease and the organism could become endemic. This would lead to production losses affecting individual farmers which could eventually translate to significant deterioration in national production. Eradication of the disease would be necessary to prevent ongoing production losses and preserve entry into our preferred markets. An eradication campaign could be expensive depending on how far the disease had spread. Further, since brucellosis is a zoonotic disease, sporadic cases of serious disease could occur in humans.

21.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and *B. abortus, B. melitensis* and *B. suis* are classified as hazards in the commodity. Therefore, risk management measures may be justified.

21.3. RISK MANAGEMENT

21.3.1. Options

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

- The incubation period for *Brucella* infections can be long and long-term carriers are known to occur, therefore isolation in quarantine is not an effective option.
- Vaccination has not been proven to be effective in camelids.
- There are no effective treatments for brucellosis in animals.
- There are several serological tests prescribed by the OIE for international trade of cattle. Since infection in camelids follows a course similar to that in cattle, the OIE *Manual* suggests that the same serological procedures may be used in camelids but they should be validated.
- Little is known about brucellosis in camelids. The recommendations made in the *Code* are for the safe trade in cattle, sheep and goats, rather than camelids. Although there are no international standards that are directly applicable when trading camelids, it is reasonable to apply recommendations in the *Code*, acknowledging that they are only broadly applicable to camelids since there are no official accreditation schemes for, or recommendations for flock freedom for camelids.
- The *Code* chapters relating to brucellosis in cattle, sheep and goats are given below:

Article 11.3.5. (Cattle)

Recommendations for the importation of cattle for breeding or rearing (except castrated males)
Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of bovine brucellosis on the day of shipment;
2.were kept in a *herd* in which no clinical sign of bovine brucellosis was officially reported during the 6 months prior to shipment;
3.were kept in a country or zone free from bovine brucellosis, or were from a *herd* officially free from bovine brucellosis and were subjected to a serological test for bovine brucellosis with negative results during the 30 days prior to shipment; or
4.were kept in a *herd* free from bovine brucellosis and were subjected to buffered *Brucella* antigen and complement fixation tests with negative results during the 30 days prior to shipment;
if the cattle come from a *herd* other than those mentioned above:
5.were isolated prior to shipment and were subjected to a serological test for bovine brucellosis with negative results on two occasions, with an interval of not less than 30 days between each test, the second test being performed during the 15 days prior to shipment. These tests are not considered valid in female animals which have calved during the past 14 days.

Article 14.1.5. (Sheep and goats)

Recommendations for the importation of sheep and goats for breeding or rearing (except castrated males) destined for flocks officially free from caprine and ovine brucellosis

Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that the animals:
1.showed no clinical sign of caprine and ovine brucellosis on the day of shipment;
2.come from a sheep or goat *flock* officially free from caprine and ovine brucellosis;
OR
3.come from a sheep or goat *flock* free from caprine and ovine brucellosis; and
4.have not been vaccinated against brucellosis, or, if vaccinated, that the last vaccination was performed at least 2 years previously; and
5.were isolated in the *establishment* of origin, and were subjected during that period to a diagnostic test for caprine and ovine brucellosis with negative results on two occasions, at an interval of not less than 6 weeks.

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

1. Animals could be imported with no restrictions since brucellosis is rare in camelids.
2. During PEI (at least 30 days) camelids could be subjected to a complement fixation test using *B. abortus* antigen or an ELISA, with a negative result.

   N.B. This measure reflects the current requirement for importing camelids from the US. The tests required in this option could be changed to include all prescribed serological tests listed in the *Manual*. Under the *Code* recommendations for cattle, this option implies that the animals were kept in a country or zone free from bovine brucellosis, or were from a herd officially free from bovine brucellosis. However, the US is not free from bovine brucellosis.
3. Animals could be isolated prior to shipment and subjected to a prescribed serological test for bovine brucellosis with negative results on two occasions, with an interval of not less than 30 days between each test, the second test being performed during the 15 days prior to shipment. These tests could be considered invalid in female animals which have given birth during the past 14 days.

   N.B. The *Manual* states that the manifestations of brucellosis in camelids are similar to those in cattle. This measure is equivalent to those recommended in the *Code* for cattle.
4. Animals could be imported only from countries that are free from *B. abortus* such as Australia and Canada.

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22. *Burkholderia pseudomallei*

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Burkholderia pseudomallei is a gram-negative bacterium.

22.1.2. OIE list

Not listed.

22.1.3. New Zealand status

The organism does not occur in New Zealand. A single introduced human case of melioidosis has been reported (Corkill & Cornere 1987).

22.1.4. Epidemiology

Melioidosis is a disease of humans and animals that occurs predominantly in tropical and subtropical regions of Asia and northern Australia and in some foci in Africa (Groves & Harrington 1994; Inglis 2004; Inglis et al 2004). It has been reported to occur in pigs as far south as south eastern Queensland (Ketterer et al 1986). The aetiological agent occurs in the environment and is widely distributed in water and soil (Sprague & Neubauer 2004). In Thailand, two thirds of paddy fields are infected with the organism and antibodies to the organisms are found in 80 % of children under 4 years old. However, avirulent cross reacting organisms are also found in the environment (Dance 2000). It has been transmitted to animals via oral mucosa, nasal mucosa, ingestion, parental inoculation, and skin scarification (Groves & Harrington 1994). Infection in natural cases is by contact with infected water and mud especially through abrasions and wounds. Water was implicated as a possible source of infections in six locations in one study (Dance 2000; Inglis et al 2004).

In animals, clinical melioidosis is most commonly seen in sheep, goats and swine. The agent may cause a wide variety of signs and lesions, varying from septicaemia and acute respiratory infections to localized abscesses. In humans, *B. pseudomallei* primarily infects hosts with impaired immunity and is believed to have a low disease-causing potential in healthy hosts. Disease does not spread from person to person (Cheng & Currie 2005). Transmission from animal to animal has not been described.

There are several reports of infection of dromedary camels in Australia (Bergin & Torenbeeck 1991; Choy et al 2000; Forbes-Faulkner et al 1992). According to Wernery and Kaaden (2002), Curasson reported in 1947 that camelids were susceptible. A case of melioidosis was reported in an alpaca in Australia (Janmaat et al 2004).

22.1.5. Hazard identification conclusion

B. pseudomallei is found very widely in the environment in tropical and subtropical areas, but has not established in temperate climates. It appears to be an opportunistic pathogen and direct transmission from animal to animal does not occur. Therefore, it is not considered to be a potential hazard in the commodity.

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23. *Chlamydophila* spp.

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Chlamydophila abortus and *Chlamydophila psittaci*. The taxonomy of the species was revised in 1999 (Everett et al 1999). The genus *Chlamydophila* was formerly *Chlamydia* and there is confusion in the older literature about the taxonomy of some species. The taxonomy of the family *Chlamydiaceae* is grouped into two genera (*Chlamydia* and *Chlamydophila*) and nine species based on DNA sequence analysis (Everett et al 1999; Longbottom 2008).

23.1.2. OIE list

Enzootic abortion of ewes is listed, and the causative agent is *C. abortus*.

23.1.3. New Zealand status

Chlamydophila abortus is exotic and is an unwanted, notifiable organism (MAF 2009).

Chlamydophila pecorum has been isolated in New Zealand (Mackereth & Stanislawek 2002).

23.1.4. Epidemiology

C. abortus is primarily a pathogen of sheep and goats (Aitken 1983), but also infects cattle. In these respective species it causes the diseases called enzootic abortion of ewes and epizootic bovine abortion.

C. abortus does not occur in Australia but is endemic in North America and the EU. Chile recently reported confirmed infection to the OIE but without clinical signs (OIE 2009b).

Transmission probably occurs by the faecal-oral and venereal routes. Persistent infections of male accessory glands and the presence of *C. abortus* in semen have been described (Storz et al 1976; Andersen 2004). Ewes that have aborted remain long-term intestinal carriers (Aitken 1983) and may also be chronically infected in their reproductive tracts (Papp et al 1994; Papp et al 1998; Andersen 2004; Teankum et al 2006). Bulls may remain carriers and excrete the organism in semen for at least 18 months (Domeika et al 1994).

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

The disease is diagnosed by demonstration or isolation of the organism in placental material. Diagnostic techniques include examination of suitably stained smears, antigen detection ELISA, PCR, demonstration of organisms in tissue section by direct staining or immunostaining, or isolation of the organism in tissue culture or embryonated eggs (Dagnall & Wilsmore 1990; Thomas et al 1990; Domeika et al 1994; Szeredi & Bacsadi 2002; Andersen 2004; Longbottom 2008; OIE 2009a). PCR tests are available for the detection of the organism in semen (Teankum et al 2006). Serological testing is unable to differentiate between the exotic *C. abortus* and endemic *C. pecorum*. They can be differentiated by sequence analysis of the 16S rRNA (Mackereth & Stanislawek 2002).

Serological tests include the complement fixation test and ELISA, but specificity is not high and cross reactions occur between *C. abortus* and *C. pecorum* and some gram-negative organisms (Longbottom 2008). Competitive ELISAs using monoclonal antibodies and tests using specific recombinant antigens that discriminate between *C. abortus* and *C. pecorum* have been developed (Longbottom 2008). The *Manual* does not prescribe a test for international trade, but the complement fixation test is suitable for use when importing/exporting animals.

There is little information on infection in camelids, however evidence was found that *C. abortus* occasionally affects alpacas (CFSPH 2009). *C. abortus* could not be demonstrated in vaginal swabs from 67 healthy llamas (Wittek 2008). Antibody to *Chlamydophila* was found in 32 % of Camelidae in 11 zoological institutions (Probst 2007). It has been stated that "leptospirosis, toxoplasmosis and chlamydiosis have been diagnosed as the major causes of abortion in llamas and alpacas" (Tibary et al 2005). Wernery and Kaaden (2002) reviewed reports of abortion and disease, characterised by eye infections and arthritis, caused by *Chlamydia* spp. Of 53 crias born over 12 years, 32 died from the infection (Wernery & Kaaden 2002). It is not clear what species of *Chlamydophila* was involved in these cases. Since there is little information about the infection in camelids extrapolation from what is known in other animal species is necessary.

23.1.5. Hazard identification conclusion

Since *C. pecorum* occurs in New Zealand it is not considered to be a hazard in the commodity. The role of *C. abortus* in causing abortion and disease in camelids is uncertain. Therefore it is considered to be a potential hazard in the commodity.

23.2. RISK ASSESSMENT

23.2.1. Entry assessment

Infections with *C. abortus* in sheep and cattle may be subclinical and chronic, and it is therefore considered possible that a similar situation could occur in camelids. Animals from any country in which the disease is endemic could be infected with the organism. Therefore the likelihood of introduction is assessed to be non-negligible.

23.2.2. Exposure assessment

Introduced camelids are likely to be mixed with New Zealand camelids and other animals. Since the organisms could be excreted in vaginal secretions after partuition, transmission to in-contact animals would be possible. Therefore, the likelihood that New Zealand animals would be infected is non-negligible.

23.2.3. Consequence assessment

Introduction of the organism could cause serious production losses, particularly in sheep in which it causes an economically important disease. The organism is pathogenic for humans and abortions have been described in pregnant women working with sheep at lambing time (Longbottom 2008). Therefore, sporadic cases of abortion could occur in women. The consequences of introduction are assessed to be non-negligible.

23.2.4. Risk estimation

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

23.3. RISK MANAGEMENT

23.3.1. Options

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

- Subclinically infected long term carriers occur. Therefore quarantine will not be effective in preventing the introduction of the organism.
- There are no effective vaccines or treatments.
- The *Manual* does not prescribe a test for international trade, but the complement fixation test is listed as a suitable alternative test for use when importing/exporting animals in general. This test is considered suitable for use in camelids.
- The recommendations made in the *Code* are for the safe trade in sheep and goats. Although there are no international standards that are directly applicable when trading camelids, it is reasonable to apply the *Code* chapter on enzootic abortion of ewes (EAE). The *Code* gives definitions for enzootic abortion-free sheep flocks but there are no equivalent recommendations for camelids, and it is doubtful whether *Chlamydia*-free flocks of camelids are available in any country. Therefore, Article 14.5.3. herds free from EAE, is unlikely to be applicable.
- The relevant *Code* Article relating to the importation of sheep and goats is given below:

Article 14.5.2.

Recommendations for the importation of sheep and/or goats for breeding
Veterinary Authorities of *importing countries* should require the presentation of an *international veterinary certificate* attesting that the animals:
1.have remained since birth, or for the previous 2 years, in *establishments* where no EAE has been diagnosed during the past 2 years;
2.showed no clinical sign of EAE on the day of shipment;
3.were subjected to a diagnostic test for EAE with negative results within the 30 days prior to shipment.

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

1. Animals could be imported without restriction.

   N.B. This measure is based on the assumption that since no evidence was found to confirm that camelids can be infected with *C. abortus*, they are not carriers of the organism.

2. Animals for importation could have remained since birth or the previous 2 years in a herd in which enzootic abortion has not been diagnosed for 2 years; *and* be tested

with negative results using the complement fixation test within 30 days of shipment.

3. Animals for importation could have remained since birth or the previous 2 years in a herd in which enzootic abortion has not been diagnosed for 2 years; *and* the herd of origin could be tested using the complement fixation test to a level that gives a high degree of confidence that the herd is free from chlamydial infections.
4. Camelids for export to New Zealand could be required to have been resident since birth in a country or zone that is free from *C. abortus* infection.

    N.B. This measure would restrict importation to animals coming from Australia and some South American countries.

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24. *Coxiella burnetii*

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

Coxiella burnetii is an obligate intracellular gram-negative bacterium that causes the disease Q fever.

24.1.2. OIE list

Q fever is listed but there is no *Code* chapter.

24.1.3. New Zealand status

Listed as an exotic and notifiable organism (MAF 2009).

24.1.4. Epidemiology

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

C. burnetii probably infects all mammalian species, birds and many arthropods (Marrie 1990; Marin & Raoult 1999). In animals the infection is of minimal economic importance and rarely causes disease, but *C. burnetii* is a zoonotic organism that sometimes causes serious disease in humans. Most human infections are asymptomatic or present as a mild flu-like condition. Acute or chronic infections sometimes occur and sometimes result in serious complications such as myocarditis, endocarditis, hepatitis and renal failure (Marin & Raoult 1999; Woldehiwet 2004). *C. burnetii* causes sporadic abortions in both humans and animals (Raoult et al 2002; Hatchette et al 2003).

Transmission frequently occurs from contact with infected uterine discharges and placentae and probably by inhalation of dust contaminated by animals and their birth products (Behymer & Riemann 1989; Marrie 1990; Hawker et al 1998; Marin & Raoult 1999; Tissot-Dupont et al 1999). Infected ticks may also play a role in spreading the disease. At least 40 species of ticks from 11 genera can be infected and their dried faeces form dust that can contaminate animal coats. Infected cattle shed the organism intermittently in their milk after successive parturitions (Kelly 2004).

Infected animals generally show few clinical signs thus making the determination of the incubation period and the interval to the development of antibodies difficult. In humans the incubation period is given as 1-3 weeks and the development of detectable antibody titres takes 2-3 weeks after the onset of symptoms (Marin & Raoult 1999). It is assumed that infected camelids will develop antibody within a similar time interval after infection.

The infection is diagnosed by serological tests or by identification or isolation of the organism. The ELISA is considered to be more sensitive than the complement fixation test (CFT) when testing cattle and sheep. However, for camelids the CFT is recommended since the ELISA has not been validated in this species (Kittelberger et al 2009). The *Manual* does not prescribe a test for international trade, but the CFT is listed as a suitable alternative test for use when importing/exporting animals.

No information could be found about the occurrence of the disease or the presence of antibodies in camelids. However, antibodies have frequently been found in camels. Wernery and Kaaden (2002) reviewed 26 articles in which the prevalence of antibodies varied from 0 to 26 % (Wernery & Kaaden 2002). In another survey the seroprevalence of antibodies in camels in Chad was 80 % (Schelling et al 2003). Therefore, it is assumed that camelids could be subclinically infected, and potential carriers of *C. burnetii*.

24.1.5. Hazard identification conclusion

C. burnetii is an exotic, notifiable and zoonotic organism and camelids are potential chronic carriers. Therefore, it is considered to be a potential hazard in the commodity.

24.2. RISK ASSESSMENT

24.2.1. Entry assessment

Since camelids are potential carriers of *C. burnetii* and the organism is globally distributed, the likelihood of entry is assessed to be non-negligible.

24.2.2. Exposure assessment

High seroprevalences of antibodies in camels indicates that they are susceptible to infection and that the organism is readily transmitted amongst them. It is reasonable to assume that *C. burnetti* could similarly be transmitted from camelids to other susceptible animals, including humans. Therefore, the likelihood of exposure of New Zealand animals and people is assessed to be non-negligible.

24.2.3. Consequence assessment

Since the organism rarely causes clinical disease in animals, establishment of *C. burnetii* would have minimal economic consequences for animal industries. However, the organism has zoonotic potential and rare sporadic cases of serious disease could occur in humans. Therefore, the consequences of introduction are assessed as non-negligible.

24.2.4. Risk estimation

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

24.3. RISK MANAGEMENT

24.3.1. Options

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

- Long term carriers of *C. burnetii* occur. Quarantine alone is therefore not a suitable measure for preventing entry of the organism.

- Although quarantine is not suitable as a means of preventing the introduction of the organism, it is useful in preventing the introduction of ticks that could be carrying the organism.
- Testing while in quarantine could be used to ensure that animals in the incubation period are not imported. The *Manual* lists the CFT as a suitable alternative test when screening animals for importation.
- There are no suitable vaccines or treatments.
- Economic impacts of introduction are likely to be minimal.
- Introduction could result in sporadic cases of severe human disease.
- There is no chapter on Q fever in the *Code*.

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

1. Animals to be imported could be tested with a serological test such as the CFT with negative results within 7 days of shipment.

   N.B. This measure would not exclude the entry of animals that are incubating the disease when tested.

2. Animals to be imported could be isolated in tick-free premises and maintained tick-free as specified in the tick section for at least 30 days and tested with a serological test with negative results within 7 days of shipment.

   N.B. This measure would ensure that animals do not become infected by ticks while in quarantine and ensures that if an animal were to be incubating the disease when introduced into quarantine it would be likely to be detected by the serological test.

3. Animals could be isolated and maintained tick-free as described above and tested twice with an interval of 3 weeks while in PEQ with a serological test with negative results.

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25. *Leptospira* serovars

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Before 1989 in the taxonomic scheme accepted at that time the species *Leptospira interrogans* contained all pathogenic serovars. Now, over 200 serovars of *Leptospira interrogans* have been re-classified serologically into at least 23 new serogroups on the basis of antigenetic relatedness. Different strains with small antigenic differences can sometimes be found within certain serovars.

For the purposes of this risk analysis serovars are written as if they were single species e.g. *Leptospira hardjo*, *L. pomona* etc.

25.1.2. OIE list

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

25.1.3. New Zealand status

Leptospira hardjo, *L. pomona, L. balcanica, L. copenhageni*, *L. ballum*, and *L. tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). Five of the species endemic in farm animals infect humans but *L. balcanica* which is associated with possums has not been diagnosed in humans (ESR 2008). A single isolation of *L. australis* has been reported from a human (Thompson 1980). In humans there were 121 cases of leptospirosis in 2008, equating to an infection rate of 2.8 per 100,000 (ESR 2009). However, leptospirosis is a rural disease and humans involved in animal husbandry are over-represented. Other *Leptospira* spp. are classified by MAF as "other exotic organisms".

25.1.4. Epidemiology

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

(Thornley et al 2002). Leptospirosis occurs world-wide and in all countries covered by this risk analysis. The endemic serotypes in each country differ.

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

In accidental hosts, the incubation period may be from 2-16 days and is followed by a period of bacteraemia. A variety of signs may be shown by diseased animals including abortion, haemolytic anaemia, icterus and nephritis. The disease can be diagnosed by the isolation of the organism, but because this is a difficult and lengthy process (taking up to 6 months) infection is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low level titre indicating resolution or a chronic infection. Serological methods are available but are difficult to interpret due to cross reactions between various serovars and problems related to selection of suitable cut-off points for interpretation and reproducibility of results. The microscopic agglutination test is still the most commonly used herd test but a number of variations of ELISAs are also available. ELISAs generally lack serovar specificity (Bolin 2008).

Leptospirosis is seldom the cause of economically serious disease in animals and is mainly of concern because it is zoonotic and occasionally causes serious disease in humans (Thornley et al 2002).

Leptospira spp. are sensitive to several antibiotics (Oie et al 1983; Gerritsen et al 1993; Gerritsen et al 1994; Alt et al 2001; Murray & Hospenthal 2004). In particular streptomycin and penicillin have been extensively used for prophylaxis and treatment of live cattle, semen and embryos in international trade.

Vaccination of animals against the main serovars occurring in New Zealand is widely practised, with the aim of developing an immune population thereby reducing the risk to humans that are in contact with the cattle.

There is little information about leptospirosis or *Leptospira* infections in camelids. Infections have been described in alpacas and a 3 month old guanaco (Wernery & Kaaden 2002). Leptospirosis has been implicated as a cause of abortion (Chenoweth 2006). In North America reported infectious causes of abortion in camelids includes leptospirosis (Tibary et al 2006). In a serological survey in Argentina, the prevalence was 36 % (Marin et al 2008). In another study in Argentina the prevalence varied from 47 to 96 % in llamas, 0 to 13 % in guanacos and 9 to 63 % in vicunas (Liorente et al 2002; Tibary et al 2006). Antibody was detected against *L. ballum*, *L. bataviae*, *L. icterohaemorrhagiae*, *L. pomona*, *L. pyogenes* (Marin et al 2008), *L. copenhageni* and *L. castellonis* (Liorente et al 2002). It can be concluded that leptospirosis is a rare disease of camelids but that subclinical infections are relatively common. Therefore, camelids can be accidentally infected and could be carriers of particular *Leptospira* serovars, but it is not known which serovars, if any, they act as maintenance hosts for.

In North America, llamas in some locations are routinely vaccinated against leptospirosis with commercially produced vaccines intended for cattle (off-label use). However, the serological response is inconsistent in vaccinated camelids. No serological response to any or, only some of the poly-valent serovars contained in the vaccine occurs. Generally the

immune response is a low titre that is short in duration (Hill & Wyeth 1991; Pugha et al 1995; Tibary et al 2006).

25.1.5. Hazard identification conclusion

Camelids may be infected with serovars that do not occur in New Zealand. Therefore exotic *Leptospira* serovars are considered to be potential hazards in the commodity.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

Since pathogenic *Leptospira* serovars are widely distributed around the world and camelids could be infected with serovars that do not occur in New Zealand, the likelihood of entry in imported camelids is assessed to be non-negligible.

25.2.2. Exposure assessment

Imported camelids will be mixed with New Zealand camelids and other livestock and new serovars of leptospira could be transmitted to New Zealand animals that contact their urine, or urine contaminated water. The likelihood of exposure is assessed to be non-negligible.

25.2.3. Consequence assessment

The consequences of introduction and establishment of new serovars of *Leptospira* are likely to be minimal for camelids and livestock since infections are usually subclinical or cause minor disease. The establishment of a new *Leptospira* serovar to which humans are susceptible could lead to sporadic occurrence of leptospirosis in humans. The number and seriousness of the cases would depend on the serovars involved and the possibility for contact with infected animals. However, the OIE Terrestrial Animal Health Standards Commission has stated that “the disease is virtually ubiquitious and international trade is not considered to increase risks to human or animal health”.

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

Since human cases could result from the introduction of new serovars of *Leptospira* associated with camelids either directly (contact with infective urine) or indirectly (establishment in rodents), the consequences for human health are assessed to be non-negligible.

25.2.4. Risk estimation

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

25.3. RISK MANAGEMENT

25.3.1. Options

The following points were considered when drafting measures for the effective management of exotic *Leptospira* serovars in the commodity:

- There are a very large number of pathogenic *Leptospira* serovars and the species in each country vary.
- Vaccines that protect against all serovars are not available. There is little information on the efficacy of vaccines in camelids, and none are registered for use in this species. Vaccination is generally unreliable; to be effective it must take place before exposure and immunity must be maintained by repeated vaccination. Vaccination is effective only against those serovars included in the particular vaccine.
- Leptospirosis is a rare disease of camelids, but subclinical infections may occur.
- The length of time that camelids may remain carriers is not known, but extrapolation from other species suggests that it may be a protracted period should they be maintenance hosts for a particular serovar(s).
- Isolation of organisms is a difficult and lengthy process taking up to 6 months and not suitable as a diagnostic method for international trade of animals.
- There are problems with the use of serological tests as a screening test particularly relating to serovar specificity. The MAT screening test for the importation of animals has limitations due to its poor sensitivity in both early and chronic infections, particularly if the relevant antigens are not represented and testing is carried out on a single sample. Serological screening for leptospirosis is of limited value because animals may be carriers without evidence of antibody production and the detection of antibody does not indicate carrier status.
- Antibiotic treatment has been used for many years as the sole method for management of the introduction of the organisms when importing animals. Antibiotic treatment is generally effective but cannot be guaranteed to clear leptospires from the kidneys in all cases.
- Recently, 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*. Leptospirosis still remains an OIE listed disease.

- The rationale for deletion was cited in the March 2009 report of the Terrestrial Animal Health Standards Commission:

  "*Leptospirosis is distributed globally; it is improbable that any country can, with any credibility, claim to be free from the disease. Further, it is unlikely that any country has an official control programme for leptospirosis. Current serological tests and culture techniques are not able, with any degree of confidence, to demonstrate that an animal is free from leptospirosis. Antibiotic treatment to clear renal carriage of leptospires is not consistently successful and has not been validated in all the species subject to international trade.*

Retention of this empty Chapter, with the words 'under study' gives the false impression that the OIE is able to formulate meaningful measures to manage the disease."

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

1. It is not known which serovars, if any, they act as maintenance hosts for. No restrictions could be placed on the introduction of camelids.

   N.B. This measure reflects the view that the disease is virtually ubiquitous and international trade is not considered to increase the risks to human or animal health (OIE 2007).

2. Camelids to be imported could be tested by serological methods that would detect antibodies to the serovars likely to occur in the exporting country.

   N.B. As discussed above, serological testing is not a reliable method for identifying infected animals and the specific serovars involved.

3. Animals to be imported could be treated with an effective course of antibiotics shortly before shipment.

   N.B. This is the method that has been most widely used for international trade to date. The treatment of choice is considered to be dihydrostreptomycin if available, otherwise oxytetracycline (these antibiotic treatments have been shown to be effective in other animals such as the pig and cattle for some serovars). It is appropriate to continue to recommend dihydrostreptomycin since this is the chemotherapy of choice and although availability is becoming restricted it may be available to exporters under permit where necessary for trade.

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26. *Mycobacterium bovis*

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

Mycobacterium bovis, an intracellular bacterium that causes bovine tuberculosis in several species of mammal, including humans.

26.1.2. OIE list

Listed.

26.1.3. New Zealand status

Mycobacterium bovis, is an endemic organism that is subject to an official control programme in New Zealand, in the form of a National Pest Management Strategy (NPMS) under the Biosecurity Act, 1993.

26.1.4. Epidemiology

Tuberculosis in camelids has been reviewed by Wernery & Kaaden (2002). It is primarily caused by *M. bovis* (Thoen et al 1977; Barlow et al 1999; Wernery & Kaaden 2002; Ryan et al 2008), and there are rare reports of *M. tuberculosis* (Wernery & Kaaden 2002) and very rare reports of *M. microti* (Oevermann et al 2004; Emmanuel et al 2007; Lyashchenko et al 2007; Zanolari et al 2009).

Camelids generally become infected with *M. bovis* by contact with other infected animal species (Twomey et al 2007). While there has been anecdotal evidence suggesting that camelids are not very susceptible to tuberculois and that transmission between camelids or to other animals from camelids is limited (Fowler 1992), in recent years tuberculosis in camelids has been recognised as a problem in a number of herds of farmed camelids in the UK, where there has been an increase in vector-related *M. bovis* infection in cattle herds (Defra 2008).

Tests available for international trade purposes are described in the *Manual*. The prescribed test is the delayed hypersensitivity (tuberculin) skin test. There are a number of blood-based tests, including interferon-gamma and ELISA. Isolation of organisms or demonstration of DNA by PCR are reliable diagnostic tests but are not generally suitable for use in live animals for export testing. Several reports indicate that the tuberculin test is unreliable, with problems associated with both specificity and sensitivity (Fowler 1992; Stevens et al 1998; Everett et al 1999; Wernery & Kaaden 2002; Lyashchenko et al 2007; Twomey et al 2007; Connolly et al 2008; Ryan et al 2008). Although serological tests show some promise (Stevens et al 1998; Lyashchenko et al 2007), only limited information is available on their use. The best approach seems to be to use a combination of tuberculin testing and serological testing.

26.1.5. Hazard identification conclusion

M. bovis can infect camelids. It is an endemic organism that is the subject of an official eradication programme (a National Pest Management Strategy under the Biosecurity Act

1993). It causes severe disease in a number of animal species including cattle and it may affect humans. Therefore, it is considered to be a potential hazard in the commodity.

26.2. RISK ASSESSMENT

26.2.1. Entry assessment

M. bovis can infect camelids and this organism is present in many countries from which camelids could be imported. In clinically healthy animals the likelihood of entry is considered to be low. However, since subclinically infected animals could be imported, if the source country had a significantly higher rate of infection in livestock than New Zealand then the likelihood of imported camelids harbouring *M. bovis* would be higher than animals from this country. Therefore the likelihood of entry is assessed as low but non-negligible.

26.2.2. Exposure assessment

There is some indication that camelids infected with tuberculosis do not show clinical signs and are not highly contagious. However, assuming that transmission by the respiratory route is possible from subclinically infected camelids, as with infected ruminants, the likelihood that imported camelids could transmit the disease to susceptible animals through contact is assessed to be low but non-negligible.

26.2.3. Consequence assessment

The introduction of infected camelids could result in a small increase in the number of cases of bovine tuberculosis in cattle and deer. However, it is unlikely that such effects would be noticed unless infected camelids were introduced directly into cattle herds that were free from bovine tuberculosis. There are no controls under the Tb NPMS to restrict the movement of camelids in this country, reflecting the low importance of these animals in the epidemiology of bovine tuberculosis in New Zealand. Nevertheless, admission of camelids into Agricultural and Pastoral (A&P) shows frequently requires a negative skin test. Therefore, although the likelihood of significant consequences arising as a result of importation of camelids is remote, for the purposes of this risk analysis the consequences are assessed to be non-negligible.

26.2.4. Risk estimation

There is a low likelihood of imported camelids harbouring *M. bovis*, and although the likelihood of transmission from infected camelids is somewhat uncertain, it appears to be lower than for cattle and deer. While the possibility of additional consequences in animals covered by the NPMS cannot be completely discounted, it is considered that the risk posed by imported camelids is unlikely to be higher than for domestically sourced animals.

Since camelids are not subjected to any control measures under the NPMS for *M. bovis*, under New Zealand's international obligations it is not possible to impose measures on imported camelids unless they originate from countries where the rate of *M. bovis* infection in livestock is higher than in this country.

As a result, the risk estimate for *M. bovis* is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures may be justified.

26.3. RISK MANAGEMENT

26.3.1. Options

The following points were considered when drafting measures for the effective management of the risk posed by *Mycobacterium bovis* in the commodity:

- Camelids appear to be not very susceptible to infection.
- Transmission from infected camelids does not appear to be common, but for the purposes of this risk analysis it has been assumed to be possible by the respiratory route from subclinically infected animals.
- New Zealand has a national control program for *M. bovis* in place under the Biosecurity Act, but this does not apply to camelids.
- Ad hoc testing of camelids for *M. bovis* is carried out in New Zealand, particularly of animals that are going to A&P shows.
- Testing protocols for camelids have been developed in the context of the New Zealand Alpaca Association's voluntary control scheme. The primary test is the single intradermal tuberculin test, and the comparative tuberculin test may be used in the case of suspicious positives.
- The ancilliary "blood tuberculosis test" (BTB), which is a combination of a lymphocyte transformation test and an ELISA, is unvalidated for camelids.
- The *Manual* discusses a lateral flow-based rapid test (TB StatPak) has been shown to be useful for detecting tuberculous animals in several species of zoo animals including South American camelids. This test is now licensed in the USA by the USDA for species such as elephants and nonhuman primates and is approved for use in the UK for badgers. However, this is not a prescribed or alternative test for international trade, and the degree to which it has been validated for camelids is uncertain.

There are no recommendations in the *Code* for tuberculosis in camelids. Article 11.6.5 has recommendations for bovine tuberculosis in cattle, water buffalo and wood bison, and Article 11.7.5 has recommendations for farmed deer.

The *Code* recommendations for Bovine tuberculosis are:

Article 11.6.5.

Recommendations for the importation of cattle, water buffalo and wood bison for breeding or rearing

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

1. showed no signs of bovine tuberculosis on the day of shipment;
2. originate from a *herd* free from bovine tuberculosis that is in a country, *zone* or *compartment* free from bovine tuberculosis; or

3.were subjected to the tuberculin test for bovine tuberculosis with negative results during the 30 days prior to shipment and come from a *herd* free from bovine tuberculosis; or
4.have been isolated for at least 90 days prior to entry into the *herd* including protection from contact with wildlife reservoirs of bovine tuberculosis and were subjected to at least two tuberculin tests carried out at a six-month interval with negative results with the second tuberculin test performed during the 30 days prior to entry into the *herd*.

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

1. No measures for camelids from any country.

NB: this reflects that infection in camelids and transmission by them is considered to be rare. It also reflects that there is no testing of camelids carried out in New Zealand under the NPMS and there are no international standards for bovine tuberculosis in camelids.

2. For countries that are free from bovine tuberculosis or countries that have an official control program that has resulted in a prevalence in cattle and deer herds as low as or lower than that in New Zealand, no measures.

N.B. This measure would be the least trade restrictive measure for countries of an equivalent or higher bovine tuberculosis status than New Zealand.

3. For countries where the prevalence of bovine tuberculosis is higher than New Zealand.

    a. Bovine tuberculosis must be a notifiable disease, and during the 3 months immediately prior to entering PEQ facilities the animals have not been on any property where *M. bovis* has been diagnosed occurred during that period.

    NB: this measure is the current New Zealand requirement in the IHS for camelids from the USA

    OR

    b. The camelids originated from establishments where no case of bovine tuberculosis occurred during the past 5 years AND
    c. The camelids were tested for bovine tuberculosis by an approved single intradermal tuberculin test at the axillary site (using 0.1ml of tuberculin PPD), with negative results (negative being no swelling or a swelling not greater than 2mm at the site of injection 72 hours after injection), within 14 days of entering PEQ but more than 90 days after any previous tuberculin test.

    NB: this measure reflects the Australian requirements for camelids from the USA, Canada and New Zealand. The NZAA voluntary scheme has a similar measure, but the test must not be carried out with 60 days of any tuberculin test.

    OR

    d. Apply the *Code* recommendations for bovine tuberculosis in cattle and farmed deer to camelids.

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27. *Mycoplasma haemolamae*

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Mycoplasma haemolamae was formerly classified as a species of *Eperythrozoon*. The organism was initially considered to most likely be *Eperythrozoon suis* (McLaughlin et al 1991). Further investigation by inoculating potential host animals led to the conclusion that the organism was not *Haemobartonella felis*, *Eperythrozoon suis* or *Eperythrozoon ovis* (Mclaughlin et al 1991). Recently, members of the *Eperythrozoon* and *Haemobartonella* genera have been reclassified as *Mycoplasma* spp. (Messick 2004; Messick et al 2002).

27.1.2. OIE list

Not listed.

27.1.3. New Zealand status

Not listed on the Unwanted Organisms Register (MAF 2009). No reference could be found of the occurrence of the organism in New Zealand. The related species *Mycoplasma* (*Eperythrozoon*) *wenyoni*, *Mycoplasma* (*Eperythrozoon*) *ovis*, *Mycoplasma* (*Eperythrozoon*) *suis*, *Mycoplasma* (*Haemobartonella*) *canis* and *Mycoplasma* (*Haemobartonella*) *felis* all occur in New Zealand (Thompson 1998).

27.1.4. Epidemiology

Eperythrozoonosis in a llama was first described in the USA (McLaughlin et al 1991). While it has been associated with wasting and anaemia in heavily infected animals, the majority of infections appear to be subclinical. For example, 70% of several hundred blood samples obtained from llamas throughout North America were positive to an ELISA (Johnson et al 1991). In another study, 24 % of llamas were found to be ELISA positive (Johnson 1989).

The mode of transmission has not been established but in line with other similar infections it is considered likely to be transmitted by arthropod parasites such as fleas, lice and ticks. It may also be transmitted *in utero* from subclinically infected dams (Almy et al 2006). Few transmission studies appear to have been carried out in camelids. Injection of infected blood into 2 splenectomised llamas and 4 cria resulted in infection in only one of the cria, although the rate was higher in animals immunosuppressed with dexamethasone (Johnson et al 1991). Available evidence in camelids and extrapolation from similar infections in other species suggests that chronic carriers may occur.

Diagnosis can be confirmed by the demonstration of the organism in blood smears but this method is likely to be insensitive for diagnosis in subclinically infected carriers. Serological tests are available including an ELISA (Johnson et al 1991). The PCR is the most sensitive test for the diagnosis of subclinical infections (Tornquist et al 2002; Almy et al 2006).

Although the organism has not been described in New Zealand, there appears to have been no active surveillance for it. It is commonly present as a subclinical infection in camelids and since thousands of camelids have been imported into New Zealand over many years

without any measures for detecting *M. haemolamae*, it is considered highly likely that this organism is already present in New Zealand. A number of other vector-transmitted mycoplasmas are present in New Zealand, indicating that suitable vectors may be present. However, for the purposes of this risk analysis it is regarded as an exotic organism.

27.1.5. Hazard identification conclusion

M. haemolamae is commonly present as a subclinical infection in camelids in the USA and probably in other countries. It has not been diagnosed in New Zealand. Therefore *M. haemolamae* is considered to be a potential hazard in the commodity.

27.2. RISK ASSESSMENT

27.2.1. Entry assessment

M. haemolamae is a common subclinical infection in camelids and is likely to be present in any country from which camelids are to be imported. Therefore, the likelihood of entry is assessed as non-negligible.

27.2.2. Exposure assessment

The high prevalence of infected animals in countries where it is known to be present indicates that the infection is easily transmitted amongst camelids. Since vectors are apparently present in New Zealand for transmission of a number of species of *Mycoplasma* found in the blood of other animal species, it is possible that competent vectors for *M. haemolamae* are present. Since imported camelids are likely to be mixed with New Zealand animals the likelihood of transmission to susceptible animals is assessed as non-negligible.

27.2.3. Consequence assessment

The establishment of the organism could lead to sporadic outbreaks of wasting and anaemia in very young animals. However, in most animals, infection can be expected to be subclinical. Therefore, in most cases it is unlikely that significant losses will be experienced. The organism does not affect humans. Domestic, feral and wild animals are not known to be infected by the organism and it is anticipated that introduction of the organism would not have any effect on any species other than camelids. There are unlikely to be any trade implications. However, since rare sporadic losses could occur in camelids the consequences are assessed to be very low but non-negligible.

27.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and *M. haemolamae* is classified as a hazard in the commodity. Therefore, risk management measures may be justified.

27.3. RISK MANAGEMENT

27.3.1. Options

The following points were considered when drafting options for the effective management of *M. haemolamae*:

- The organism is a commonly transmitted chronic infection of camelids and the only risk management measure that has applied to date is absence of diagnosis on the premises over the previous 3 months.
- Infection results in few cases of serious disease.
- Long-term carriers occur. Therefore quarantine will not be effective to prevent entry of the disease.
- There are no vaccines for the disease.
- Treatment with tetracyclines is helpful in clinical cases but is unlikely to result in elimination of the organism from carriers.
- There is no *Code* chapter relating to *Mycoplasma haemolamae*.

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

- Since the organism causes a minor disease and is likely to be present in New Zealand already, camelids could be imported provided they meet the commodity definition and are free from ectoparasites.

N.B. This recognises that a large number of camelids have been imported in the past with no measures in place to detect subclinical infections.

- Camelids could be certified as not having resided in the past 3 months on any property where *M. haemolamae* has been diagnosed.

N.B. This measure reflects the current requirement for alpacas and llamas imported from the USA.

- Animals to be imported could be kept free from external parasites for 30 days before shipment and tested by microscopic examination of a blood smear within 10 days of shipment with negative results.

N.B. This screening method lacks sensitivity because mycoplasmas quickly detach from erythrocytes once blood is taken and bacteraemia is generally low and intermittent.

- Animals to be imported could be maintained free from external parasites for the 30 days before shipment and tested using a PCR test within 10 days of shipment, with negative results.

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28. *Pasteurella multocida* types 6B and 6E

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agent

Pasteurella multocida strains are classified into five capsular antigen types (A, B, D, E and F) and 16 somatic antigen types (De Alwis 1999). Capsular typing is done by various methods described by Carter, Heddleston, Namioka and Murata. If the Namioka-Carter classification method is used the types that cause haemorrhagic septicaemia are 6.B and 6.E. In the Heddleston-Carter system the synonyms for these strains are B:2 and E:2 (Srivastava et al 2008). Other types are associated with pneumonic pasteurellosis.

28.1.2. OIE list

Haemorrhagic septicaemia is listed as a disease of bovidae (cattle and buffaloes).

28.1.3. New Zealand status

Pasteurella mutocida types B:2 and E:2 (6.B and 6.E) are listed as unwanted, notifiable organisms.

28.1.4. Epidemiology

Haemorrhagic septicaemia is predominantly a disease of cattle and buffaloes and they are the reservoir hosts. It occurs in, but is not restricted to, tropical and sub-tropical countries of Asia and Africa. It does not occur in Australia or Canada, and has been suspected but not confirmed in the USA. It occurs in Brazil and has previously occurred in Argentina and Panama (OIE 2009c). In the 27 countries that make up the EU it has occurred only in Italy and Portugal during the last 3 years (OIE 2009c). In Africa, it is caused by *Pasteurella multocida* types B and E and in Asia by type B (Carter 1998; Bastianello & Henton 2004).

The incubation period in naturally acquired infections is from 1-3 days (De Alwis et al 1990; De Alwis 1992; Carter 1998; Bastianello & Henton 2004). The course usually varies from peracute to subacute but inapparent infections also occur. Peracute infections are characterised by sudden death, while acute cases show fever, profuse salivation, nasal discharge, and rapid respiration. Firm subcutaneous swellings in the submandibular region are seen in subacute cases. Untreated cases usually end fatally (Bastianello & Henton 2004). Animals that survive infection may be active carriers for 4-6 weeks and then become latent carriers. In herds recently exposed to the infection, up to 23 % of animals may be latent carriers for at least 229 days (De Alwis et al 1990; Bastianello & Henton 2004) with the organism being harboured in the nasopharynx, retropharyngeal lymph nodes, and tonsils, from which it is periodically shed when the animal is stressed (De Alwis et al 1990; Bastianello & Henton 2004). The organism is excreted in respiratory aerosols, saliva, urine, faeces and milk. Transmission is by the respiratory route or on fomites.

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

Animals become septicaemic a few hours before death and culture from blood is possible only in this period (Srivastava et al 2008). Recovered animals and latently infected animals carry the organism in their tonsils. Repeated culturing of tonsillar swabs is recommended in the *Code* (OIE 2009b). 6.B and 6.E strains produce hyaluronidase and can be identified by various PCR methods as well as serological methods (Srivastava et al 2008). Serological tests using the indirect haemagglutination test are seldom used. High antibody titres are indicative of recent infection (Srivastava et al 2008) but are not useful for detecting latent carriers.

Both live and dead vaccines have been used. Vaccination as a means of control has been reviewed (De Alwis 1999). Although most authorities believe that vaccination reduces mortality, accurate data are not available and there is no evidence that suggests it could be used in imported camelids to effectively control the entry of the organism.

There is some doubt about the occurrence of the disease in camels. Some cases of natural infection have been described but attempts to transmit the disease with isolates have generally failed. The situation has been reviewed by Werney & Kaaden (2002). The meeting of the OIE ad hoc group on Camelidae diseases classified haemorrhagic septicaemia as a significant disease of camels. In camelids, haemorrhagic septicaemia is classified as a minor or non-significant disease and the group recommended that work be done on controversial data on the susceptibility and aetiology in camelids (OIE 2009a). No evidence was found that camelids are susceptible to haemorrhagic septicaemia.

28.1.5. Hazard identification conclusion

Since haemorrhagic septicaemia is predominantly a disease of cattle and buffalo and no evidence could be found of it occurring in camelids it is not considered to be a hazard in the commodity.

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29. *Salmonella* spp.

29.1. HAZARD IDENTIFICATION

29.1.1. 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 of serovars 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 *Salmonella* Typhimurium is used. Phage typing of *Salmonella* serovars is also commonly used to classify strains. The definitive phage type number is given after the serovar name e.g. *Salmonella* Typhimurium DT104.

29.1.2. OIE list

Only *Salmonella* Abortusovis, *Salmonella* Pullorum and *Salmonella* Gallinarum are listed.

29.1.3. New Zealand status

Salmonella Abortusovis, *Salmonella arizonae*, *Salmonella* Dublin, *Salmonella* Enteritidis DT4, *Salmonella* Gallinarum, *Salmonella* Pullorum, *Salmonella* Typhimurium DT44 and DT104 and salmonellae (exotic affecting animals), are listed as unwanted organisms (MAF 2009).

Salmonellae 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 2009). Isolations from both medical and animal health laboratories are included. In 2008 there were 1399 isolates from humans and 1349 isolates from non-human sources (mainly animals). The most common serotype isolated from both human and non-human sources was *S.*Typhimurium. *S.* Typhimurium DT104 is of particular importance because it exhibits multiple resistance to the commonly used antibiotics and is a threat to human health (Hogue et al 1997; Jones et al 2002; Plagemann 1989). In 2008 the definitive phage types DT104 and DT44 were not isolated from humans or animals.

29.1.4. Epidemiology

There is little specific information about *Salmonella* infections in camelids. In an extensive review up to 2002, Wernery and Kaaden (2002) mention only one report of salmonellosis in camelids. No salmonellae were isolated from 45 crias with diarrhoea and 268 llamas from 29 properties (Rulofson et al 2001; Cebra et al 2003).

The first report of enteric salmonellosis in New World camelids appeared in 2004, a case of suppurative hepatitis in an alpaca associated with *S.* Typhimurium (Saulez et al 2004). Other forms of salmonellosis in camelids include meningitis in a newborn alpaca caused by *S.* Newport (D'Alterio et al 2003) and two cases of septicaemic salmonellosis caused by *S.* Choleraesuis and *S.* Typhimurium (Anderson et al 1995). Salmonellosis has been suggested as a cause of neonatal mortality in crias (Tibary et al 2006).

Since salmonellosis in camelids is rarely reported, much of the information given below is extrapolated from other species, particularly cattle, in which enteric salmonellosis caused by host adapted serovars is a very common form of the disease.

In enteric salmonellosis transmission is mainly by the oral route and factors such as infecting dose, the particular strain and serovar, and various stress factors influence the outcome of infection (Fenwick & Collett 2004). The incubation period in camelids is not known, but in cattle it is as little as 15 minutes in newborn calves (Radostits et al 2007). After oral infection, salmonellae colonise the distal ileum. Initial infection may be followed by bacteraemia and dissemination to several organs. In the case of pregnant animals, abortion may occur. Animals that recover from *Salmonella* infections may become carriers for life, shedding organisms sporadically in their faeces. Excreted organisms contaminate the environment and become a source of infection (Radostits et al 2007).

Carriers of infections can be detected by culturing faeces but, because excretion is intermittent, repeated sampling and culture may be necessary. Serology may be useful but is best applied on a herd basis (Davies 2008; Veling et al 2002). No practical serological method exists for detecting individual carrier animals (Hansen et al 2006).

29.1.5. Hazard identification conclusion

Salmonellosis is very rarely reported in camels. However, assuming that they could, like other animals, become carriers of exotic *Salmonella* serovars such as *S.* Dublin or *S.* Typhimurium DT104 that could be harmful to animal industries or to humans, salmonellae are considered to be potential hazards in the commodity.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

Salmonella spp. have a world-wide distribution and the range of serovars varies from one country to the next. Assuming that subclinical carriers can occur, the likelihood that imported camelids could introduce exotic *Salmonella* serovars is assessed to be non-negligible.

29.2.2. Exposure assessment

Imported camelids will be introduced onto premises where New Zealand camelids and other animals are present. Assuming that camelids that are enteric carriers of salmonellae will excrete organisms in their faeces as other carrier animals do, the likelihood of exposure and infection of indigenous animals is assessed to be non-negligible.

29.2.3. Consequence assessment

Introduction of infected animals would be likely to result in spread of the organisms throughout the country due to movement of animals and people. The potential for spread is illustrated by the spread of *S.* Brandenberg in sheep and humans (Clark et al 2004; Clarke & Tomlinson 2004). Another example was the spread of *S.* Typhimurium DT160 following its first detection in sparrows and humans (Alley et al 2002). It spread rapidly to become the most widely isolated *Salmonella* spp. as can be seen in the data reported in the ESR database (ESR 2009). Introduction of new serovars may result in production losses in

animals and sporadic cases of salmonellosis in humans. Wild and feral animals and birds may also be susceptible to infection.The consequences of introduction are therefore considered to be non-negligible.

29.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and exotic salmonellae are classified as hazards in the commodity. Therefore, risk management measures may be justified.

29.3. RISK MANAGEMENT

29.3.1. Options

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

- Salmonellae are very rarely reported in camelids, and there is no evidence of host adapted serovars in camelids.
- When considering imports of animals that may be long term carriers, quarantine without other supporting measures is not useful to prevent the introduction of exotic salmonellae.
- Carriers of infections can be detected by culturing faeces but, because excretion is intermittent, repeated sampling and culture may be necessary.
- There are no suitable vaccines for use in camelids.
- Treatment is not a useful option since resistance of salmonellae to antibiotics is widespread.
- Many serovars of salmonellae are present in New Zealand, including most of the common and significant serovars.
- There are large numbers of horses, cats and dogs imported into New Zealand without safeguards for salmonellae.
- About 4 million people enter New Zealand annually without any safeguards being applied for salmonellae. Up to 11 % are likely to be carrying Salmonellae. Direct person-to-person spread is estimated to be around 5 % of human cases in New Zealand (MacDiarmid 2010).
- There are no recommendations in the *Code* relating to *Salmonella* serovars for species other than poultry.

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

1. Camelids could be imported without restrictions.

NB. This measure recognises that salmonellae are very rarely reported in camelids and assumes that importation of exotic salmonellae by camelids is an insignificant pathway of introduction when compared to other pathways. This option would be consistent with the fact that there are large numbers of horses, cats and dogs imported into New Zealand without safeguards.

2. Camelids could be required to have originated from farms where there have been no laboratory-confirmed cases of salmonellosis due to *Salmonella* Dublin or *Salmonella* Typhimurium DT 104 in the last 1-3 years.

N.B. This option rests on the assumption that camelids may be carriers of *S.* Dublin and *S.* Typhimurium DT104. The number of years property history required can be varied according to acceptability of this risk.

3. While in PEQ, faecal samples from camelids could be cultured for S. Dublin and S. typhimurium DT104, with negative results.

N.B. This option is partly consistent with measures in the current IHS, and rests on the assumption that camelids may be carriers of *S.* Dublin and *S.* Typhimurium DT104. Extra sensitivity could be achieved by carrying out two faecal culture tests at an interval of 10 to 14 days.

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30. *Yersinia pestis*

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

Yersinia pestis is the causative agent of plague and is a gram-negative bacterium.

30.1.2. OIE list

Not listed.

30.1.3. New Zealand status

The last case of plague in humans was reported in 1911. It is classified as an unwanted, other exotic organism (MAF 2009) and yersiniosis is a Section A disease notifiable to the Medical Officer of Health (MoH 2009).

30.1.4. Epidemiology

Plague, caused by *Yersinia pestis*, resulted in millions of human deaths in the middle ages. Modern treatment and management of vermin has reduced the threat but the World Health Organization still reports 1,000 to 3,000 cases of plague annually (CDC 2009). Plague is primarily transmitted by fleas from rodent hosts, particularly rats and wild rodents such as prairie dogs in the USA. It can also be transmitted by bites and scratches from infected animals or by the respiratory route in cases of pneumonic plague. It is carried by a large number of rodents and mammals and according to one reference about 200 species of rodents had been proved to be naturally infected (Davis et al 1975). The disease in humans presents as bubonic plague (lymph node infection at the site of infection), septicaemic or respiratory infection.

The disease in camels has been reviewed (Wernery & Kaaden 2002). It is associated with contact with infected rodents and transmission by fleas or mechanically by ticks of the genera *Hyalomma* and *Ornithodoros*. The incubation period in camels is 1-6 days and death occurs within 20 days. In a recent plague outbreak, deaths that occurred in dromedary camels were associated with transmission by fleas from jirds (desert dwelling rodents) living in the camel enclosure. The disease was transmitted to humans that ate raw liver from a camel that had died of the infection (Bin Saeed et al 2005).

The OIE ad hoc group on Camelidae diseases does not list plague as a significant disease of camelids. Only one reference was found relating to plague in a llama. This was a personal communication only with no supporting information (Orloski 2003). Camelids are not considered to be maintenance hosts for *Y. pestis*.

30.1.5. Hazard identification conclusion

Camelids are not recognised maintenance hosts for *Y. pestis*. Only one report was found of the occurrence of plague in camelids and this was a second-hand report of a personal communication. The disease in camels is an acute disease with death occurring within 20 days. Camelids that are not infested with fleas are unlikely to transmit the disease. Fleas are not mentioned as significant parasites of camelids by the OIE ad hoc group on

Camelidae diseases, or as transmitting diseases in camelids (Wernery & Kaaden 2002). Therefore, *Y. pestis* is not considered to be a hazard in the commodity.

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31. *Coccidioides immitis*

31.1. HAZARD IDENTIFICATION

31.1.1. Aetiological agent

Coccidioides immitis is a soil-borne fungus.

31.1.2. OIE list

Not listed.

31.1.3. New Zealand status

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

31.1.4. Epidemiology

Coccidioides immitis is a dimorphic soil-borne fungus confined to areas of North, Central and South America characterised by alkaline sandy soils and high environmental temperatures. In soil it is present as vegetative mycelia which consist of alternating arthroconidia separated by smaller thin walled non viable cells. When released into the atmosphere the arthroconidia can be inhaled and develop in an infected host into spherules. The organism typically resides in the soil and humans and animals are accidental hosts and do not become infectious. The disease occurs most commonly in humans and dogs (Wernery & Kaaden 2002; Greene 2006). The disease has been described in llamas (Muir & Pappagianis 1982; Fowler et al 1992).

31.1.5. Hazard identification conclusion

The disease is confined to hot dry areas of the USA and Central and South America where the fungus is found in the soil. Infected animals and humans are not infectious. Therefore the disease could not be transmitted by camelids and could not establish in New Zealand. *Coccidioides immitis* is not considered to be a hazard in the commodity.

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32. *Trypanosoma* spp.

32.1. HAZARD IDENTIFICATION

32.1.1. Aetiological agents

Trypanosoma evansi is the aetiological agent of surra in animals. Humans are not susceptible to infection.

Trypanosoma cruzi infects humans and animals and is the aetiological agent of Chagas disease in humans.

Trypanosoma vivax infects animals but not humans and is the aetiological agent of the disease nagana in animals.

Other pathogenic trypanosomes such as *Trypanosoma congolense, Trypanosoma brucei* and *Trypanosoma simiae* are typically African parasites and do not occur in any of the countries relevant to this risk analysis.

32.1.2. OIE list

Surra is listed as a disease of multiple species and trypanosomosis (tsetse-transmitted) is listed as a disease of cattle. However, there are no *Code* chapters for either.

Of the tsetse fly transmitted trypanosomes, only *Trypanosoma vivax* is relevant to this risk analysis as it is the only species that is transmitted mechanically by biting flies in South America.

32.1.3. New Zealand status

T. evansi and *Trypanosoma* spp. are classified as unwanted, notifiable organisms (MAF 2009).

32.1.4. Epidemiology

T. evansi

T. evansi is mechanically transmitted by biting flies. It occurs in Northern Africa, the Middle East, some areas of Russia, India, China, South-East Asia (as far east as Timor) and South America (DAFF 2007; Luckins 2008; Uilenberg 1998). It causes surra, most importantly in camels and horses, but also in cattle, buffaloes, sheep and other domestic and wild animals. The occurrence of the disease in camelids is controversial. Some authors list camelids as animals in which surra occurs (Australian Government 2007; Uilenberg 1998). In other authoritative reviews, llamas are not mentioned as animals in which it occurs (DAFF 2007; Luckins 2008; Uilenberg 1998). Wernery & Kaaden (2002) state that "Trypanosomosis has not been reported in South American camelids despite the presence of *T. evansi* in cattle and horses" Fowler states that "It (surra) has also been reported from Central and South America, although trypanosomiasis has not been reported from South American camelids in these areas". He also states that " However, trypanosomes have been isolated from llamas imported into the United States from Chile" giving as reference a personal communication from a reputable source (Fowler 1992). However, the species of trypanosome that was isolated is not specified. One expert has stated that llamas may be infected with *T. evansi* but specific references to cases are not given (Uilenberg 1998). No

reports were found in which *T. evansi* was positively identified in natural cases in camelids or transmitted experimentally to camelids. However, it is a major disease in camels and since a wide variety of animal species are susceptible to the disease it is likely that camelids could also be susceptible.

In camels, surra has an incubation period of 5-60 days (DAFF 2007). Acute cases may occur but the disease is more often chronic and although most cases end fatally infected animals may live for several years (DAFF 2007; Uilenberg 1998). The disease is characterised by recurrent bouts of fever and parasitaemia, loss of condition, oedema and anaemia. It can be diagnosed by the demonstration of parasites in the blood by a number of microscopic techniques, by inoculation of laboratory rodents or by PCR. However PCR has not proven to be any more sensitive than mouse inoculation (Luckins 2008). Serological tests are available. The ELISA is probably the most reliable test and a card agglutination test can be used to confirm equivocal test results (Luckins 2008). Generally, the diagnosis of surra is based on the demonstration of parasites in the blood, supplemented by serological tests.

T. cruzi

T. cruzi is the aetiological agent of Chagas disease a common and serious disease of humans. The disease is restricted to South and Central America and occurs more rarely in the southern parts of the USA. Closely related trypanosome species such as *T. rangeli* also occur in these regions. The organisms can infect large numbers of animal species.

The agent is transmitted by kissing bugs (Family: *Reduviidae*, Sub-family: *Triatomatinae*). Kissing bugs do not stay on the host but hide in cracks in walls and other places and attack humans or animals for brief periods, usually at night, during which time they take blood meals from their hosts (Krinsky 2009). Infected animals are not contagious.

T. vivax

T. vivax is an African trypanosome that is transmitted by tsetse flies. However, it can also be transmitted mechanically by biting flies and has become established in Central and South America, Mauritius and some Carribean islands (Silva & Davila 1996; Silva & Davila 2001; Connor & Van den Bossche 2004).

Trypanosoma vivax can infect camels but the position with regard to camelids remains uncertain and no specific descriptions of the disease in camelids were found. However, since the parasite infects a wide variety of animals including camels it is assumed that it could infect camelids. A diagnosis can be made by a variety of techniques for the microscopic examination of blood but these tests lack sensitivity in cases with low parasitaemia. PCR methods are available but species-specific primers are required for each *Trypanosoma* spp. By using a primer specific for *T. vivax* PCR should be highly sensitive. Antibody tests are also available and ELISAs for *T. vivax* have high sensitivity and genus specificity but species specificity is generally low (Desquesnes 2008).

32.1.5. Hazard identification conclusion

Since the vectors necessary for transmission of *T. cruzi* are not carried on animals, and do not occur in New Zealand, the likelihood that the parasite could be introduced by camelids and become established in New Zealand is negligible.

T. evansi and *T. vivax* occur in South America and *T. cruzi* occurs rarely in southern parts of the USA, but not in other countries of relevance to this risk analysis. It is concluded that *T. vivax* and *T. evansi* are not hazards in camelids imported from Australia, Canada, the European Union or most parts of the United States, but they are considered to be potential hazards in camelids from Central and South America.

32.2. RISK ASSESSMENT

32.2.1. Entry assessment

T. evansi and *T. vivax* occur in South America and may be capable of infecting camelids. Therefore, the likelihood that they could be introduced by camelids imported from South America is assessed to be non-negligible.

32.2.2. Exposure assessment

Surra is a tropical disease and the principal vectors are *Tabanus* spp. flies not present in New Zealand. However, both trypanosomes are transmitted mechanically by biting flies such as *Stomoxys* spp. *S. calcitrans* is a competent vector typically found in greater numbers in warmer parts of New Zealand. Therefore, north of Auckland would be the most likely area for establishment to occur initially. The feeding hosts for *S. calcitrans* are cattle, horses, sheep, dogs and humans. However, *S. calcitrans* is widely distributed world-wide in countries where surra does not occur. This indicates that surra is unlikely to establish in New Zealand as it has not done so in any other temperate climate despite the presence of *S. calcitrans*.

Since *Stomoxys* spp. are present in New Zealand the likelihood of transmission to susceptible New Zealand animals and the establishment of *T. evansi* and *T. vivax* is assessed to be very low but non-negligible.

32.2.3. Consequence assessment

If *T. evansi* or *T. vivax* were to establish there could be serious consequences for a number of animal industries and companion animals since the organisms have a wide host range. Horses are particularly susceptible to *T. evansi* and are also susceptible to *T. vivax* and cases of clinical disease and mortalities could cause economic losses. Ruminants are also susceptible to both trypanosomes. Wild animal species such as deer could also be infected. Humans are not susceptible and there would be no consequences for human health.

32.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible for camelids imported from South America, the risk is assessed as non-negligible and *T. evansi* and *T. vivax* are classified as hazards in the commodity. Therefore, risk management measures may be justified.

32.3. RISK MANAGEMENT

32.3.1. Options

The following points were considered when drafting options for the effective management of *T. evansi* and *T. vivax* in the commodity:

- The disease may be chronic and during periods of remission infected animals may show no signs of disease. Therefore, quarantine alone is not a reliable method to prevent entry of the organisms. However, quarantine and testing in insect-free premises could help ensure that animals are not incubating the disease when imported.
- There are no vaccines or treatments suitable for use when importing animals.
- A number of test options are available for the diagnosis of infection in both acutely and chronically infected animals.
- There are no *Code* chapters relating to surra or trypanosomosis caused by *T. vivax,* but *T. evansi* is covered in the *Manual*. Diagnosis of surra is usually based on the demonstration of the parasites in the blood, supplemented by serological tests.

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

1. Camelids could be introduced without restrictions.

N.B. This measure assumes that camelids cannot carry either of the trypanosomes and/or that they could not establish in New Zealand. This measure would be further supported by the fact that both *T. evansi* and *T. vivax* have remained confined to certain tropical and sub-tropical countries and have never occurred in temperate climates.

2. As close as practicably possible to the date of departure, animals could undergo direct examination of the blood by a concentration method recommended in the *Manual*, with no parasites observed.

3. Animals to be imported could undergo a sensitive serological test (e.g. ELISA) for *T. evansi* and *T. vivax* within 1 week of shipment.

N.B. The above two measures (2 and 3) are usually combined to diagnose infection.

4. Animals to be imported could be isolated in insect-free premises for 4 weeks and tested by a sensitive serological test (e.g. ELISA) for *T. evansi* and *T. vivax* within 1 week of shipment.

N.B. In this case animals that are incubating the infection and are serologically negative when introduced into quarantine premises would seroconvert and be detected before shipment.

5. Animals to be imported could be isolated in insect-free premises for 4 weeks and tested by a sensitive serological test (e.g. ELISA) and by PCR for *T. evansi* and *T. vivax* within 1 week of shipment.

N.B. Using a diagnostic test to detect antibody and one to detect parasite DNA would increase the sensitivity of the testing procedure.

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33. *Echinococcus granulosus*

33.1. HAZARD IDENTIFICATION

33.1.1. Aetiological agent

Echinococcus granulosus is a tapeworm (cestode) parasite.

33.1.2. OIE list

Echinococcosis/hydatidosis is a listed disease of multiple species.

33.1.3. New Zealand status

New Zealand was declared provisionally free from *Echinococcus granulosus* in 2002 (Pharo 2002) with no cases having been found since. *Echinococcus* spp. are listed on the Unwanted Organisms Register as notifiable organisms and hydatid disease is notifiable to the Medical Officer of Health (MoH 2009).

33.1.4. Epidemiology

The adult parasite is a small tapeworm found in the intestines of dogs that have eaten offal from sheep infested with hydatid cysts. Hydatid cysts, the cystic form of *Echinococcus granulosus,* occur particularly in the lungs, liver and occasionally in other organs of sheep, goats and other susceptible host animals that have ingested tapeworm eggs that have been voided with dog faeces. Humans are accidental hosts infested by ingesting tapeworm eggs that rarely go on to develop into hydatid cysts. *Echinococcus granulosu*s can cause a severe (potentially fatal) disease in humans when the cyst stage develops in vital organs. Humans are considered to be dead-end hosts. Dogs are infested by eating hydatid cyst infested offal from sheep or other intermediate hosts.

Hydatidosis has been described in camelids and camels. The advent of molecular typing techniques has resulted in the identification of at least 10 genotypes of *Echinococcus granulosus*. Type G1 is the common sheep type, G2 is the Tasmanian sheep variant, G4 is the horse type, G5 is the cattle type, G6 is the camel type, G7 is the pig type, G8 is a cervid type and G10 a reindeer type (Lavikainen et al 2006). It has been suggested that types G6-10 may represent a separate species (Lavikainen et al 2006). Most human infestations are caused by the G1 type.

There is little information about hydatidosis in camelids. However, a large number of human infestations in Argentina are caused by G6 (the camel type) and therefore it seems probable that camelids are infected with this type (Guarnera et al 2004). Evidence from several papers suggests that sheep type G1 and the camel type G6 exist in epidemiologically distinct sheep/carnivore and camels/carnivore cycles respectively (Ahmadi & Dalimi 2006; Oudni-M'rad et al 2006).

New Zealand is free from the parasite but it could be re-introduced through the importation of animals. These could be dogs, the definitive host, or any imported intermediate host animal that is infested with hydatid cysts and subsequently fed to dogs.

Diagnosis of hydatids in live animals is difficult since they show no signs of infestation. Serological diagnosis in intermediate hosts has proved to be specific but insensitive

(Kittelberger et al 2002) and is therefore unreliable in individual animals although it may have application as a flock test. Ultrasound scanning has been suggested as a means of diagnosis in live animals but it is in principle not suitable for diagnosis of recently infested cases with small lesions. There is no literature indicating that ultrasound scanning is reliable or has been validated.

Immunisation with a recombinant vaccine has proved to be highly effective in lambs vaccinated at a young age according to the recommended regimen. It is not effective in sheep that are already infested and therefore, for control of infestation vaccination of young lambs is recommended (Heath & Holcman 1997; Heath et al 2003; Gauci et al 2005). However, the vaccine is not yet produced commercially, is not registered for use and has not been used in camelids. Therefore, it is not an option at the present time.

Legal requirements that apply domestically that relate directly to the control of *Echinococcus granulosus*/hydatidosis are:

Biosecurity Declaration of a Controlled Area Notice- *Echinococcus granulosus* (Hydatids) declares the whole of New Zealand to be a controlled area in which raw offal from livestock[C] shall not be accessible by dogs:

i. Slaughter and dressing of livestock shall take place in a dog-proof enclosure.
ii. Owners shall control their dogs at all times in such a manner as to prevent them from having access to raw offal of livestock.
iii. Offal shall be cooked by boiling for a minimum of 30 minutes before feeding to dogs.

33.1.5. Hazard identification conclusion

Echinococcus granulosus is known to affect camelids and is present in all countries relevant to this risk analysis. Hydatidosis cannot be reliably diagnosed in live animals. Therefore *E. granulosus* is considered to be a potential hazard in the commodity.

33.2. RISK ASSESSMENT

33.2.1. Entry assessment

Echinococcus has a global distribution and cannot be reliably diagnosed in live animals. Therefore, the likelihood of introducing the parasite in imported camelids is assessed to be non-negligible.

33.2.2. Exposure assessment

Introduced camelids will be kept on farms and may have contact with dogs. The slaughter of camelids at home kill facilities is probably an uncommon practice when compared to sheep. However, if camelid offal is fed to dogs, or if dogs eat offal from a dead infested camelid they could become infested and in turn infest herbivourous intermediate hosts. Although there is some evidence that the camelid/carnivore cycle may be epidemiologically distinct from the sheep/carnivore cycle this has not been definitively

[C] The Notice interprets livestock to mean: animals kept for use or profit and includes, but are not limited to, sheep, goats, cattle, deer, horses, llamas, alpacas and camels.

proven. It is assumed that introducing infested camelids could reintroduce the disease into the sheep population. Therefore, the likelihood of exposure is assessed to be low but non-negligible.

33.2.3. Consequence assessment

E. granulosus can infest cattle, sheep and goats, pigs, camelids, wild and feral ruminants, and macropods such as wallabies that occur in New Zealand. Wild and feral animals could be involved in maintaining and disseminating the parasite to dogs. The presence of the parasite in animals other than sheep could result in transmission to sheep and the re-establishment of a sheep/dog cycle and sporadic cases of human disease.

Re-establishment of the parasite in a dog/sheep cycle in New Zealand would have consequences for human health. Neither dogs nor intermediate hosts develop clinical signs of infestation, and control or re-eradication programmes would be implemented on human health grounds (Pharo 2002). This could be a lengthy and expensive process dependent on the extent to which the parasite has become dispersed.

In view of the above, the consequence assessment is assessed to be non-negligible.

33.2.4. Risk Estimation

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

33.3. RISK MANAGEMENT

33.3.1. Options

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

- New Zealand has eradicated *E. granulosus*.
- Hydatid-free flocks or herds are not defined in the *Code* and it is doubtful if the disease is reportable in most countries.
- No reliable tests are available for the ante-mortem diagnosis of hydatid cysts in intermediate hosts. Serological tests have low sensitivity and are not generally available.
- Premise freedom is not a practical option because hydatids can be diagnosed in intermediate hosts only by post-mortem inspection of offal. Therefore, the only properties that could claim historical freedom would be those that regularly sent a significant number of animals to slaughter at a plant where hydatids was recorded if found. This is unlikely to be feasible, particularly for properties that have only camelids and where the disease is not notifiable.
- Newly developed vaccines for ruminants show promise but are not yet available for general use.

- The role of an IHS is to specify measures that must be complied with up to the point of biosecurity clearance for imported risk goods. An IHS cannot include obligations that apply post-clearance.
- The parasite could not establish in New Zealand unless cysts within carcasses were eaten by dogs. A Controlled Area Notice under s131 of the Biosecurity Act (issued by MAF on 5th May 2010) prohibits the feeding of uncooked offal of camelids to dogs. If the provisions of that notice are followed by owners of camelids, the likelihood of hydatids transmission from imported camelids to dogs is negligible.

There are no practical measures to effectively manage the risk of hydatids in camelids that are suitable for inclusion in an IHS on which to base a biosecurity clearance.

However, there are several options for the effective management of the risk in a post-border setting. One or a combination of the following options could be considered:

1. At the time of importation, importers could be provided with information to inform them of their obligations under the Controlled Area notice in regard to cooking offal of livestock prior to feeding it to dogs, and in regard to controlling dogs so that they cannot gain access to offal of animals that died on the premises. This information could be contained in the Guidance Document accompanying the IHS, or could be in another form.
2. Obligations could be imposed on importers and subsequent owners of imported camelids (either by amending the current imported animals identification regulations[D] or by some other regulatory mechanism) to report imported animal deaths, thereby enabling oversight of appropriate carcass disposal.

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34. Internal parasites

34.1. HAZARD IDENTIFICATION

34.1.1. Aetiological agent

All nematode, trematode and cestode parasites (except *Echinococcus granulosus*) of camelids (see Section 34.1.4 for details).

34.1.2. OIE list

Internal parasites are not listed in the *Code*, except for *E. granulosus* which is considered in the previous section of this risk analysis.

34.1.3. New Zealand status

The parasites known to occur in New Zealand have recently been compiled into an updated check-list (McKenna 2009). This check-list is used in this section to determine which parasites are present in New Zealand.

34.1.4. Epidemiology

34.1.4.1. Nematodes

A large number of nematode parasites have been identified in camelids and undoubtedly more will be identified in the future. Three sources were consulted to make a list of parasites for consideration. Taylor et al (2007) list 18 species of parasites identified to species or genus level (Taylor et al 2007j), Wernery and Kaaden list 21 (Wernery & Kaaden 2002) and Fowler lists 25 (Fowler 1992). Some are parasites of camelids but many are also parasites of cattle, sheep and other animals. The parasites listed in the above sources were compared to the list of those identified in New Zealand (McKenna 2009) to determine which are exotic. It was concluded that the following have not been identified in New Zealand:

Angiostrongylus cantonensis

Graphinema aucheniae

Marshallagia marshalli

Nematodirus lamae

Spiculopteragia peruvianus

Thelazia californiensis

Parelaphostrongylus tenuis

Angiostrongylus cantonensis has been found in the lungs of an alpaca. It is described as a lung parasite of rats and sometimes humans. It has an indirect lifecycle requiring development in a snail and ingestion of a snail by the final host. Diagnosis of infection can be made by demonstration of eggs in faeces (Taylor et al 2007a). It is probably rare in camelids and of little importance since it is primarily a parasite of rats and only incidentally of other animals.

Graphinema aucheniae is a parasite of the abomasum of alpacas. It has been reported only from South America and has a direct lifecycle similar to other Trichostrongyles (Fowler 1992).

Marshallagia marshalli is a common abomasal parasite of sheep, goats, deer and wild small ruminants and occurs in South America. It is not considered to be an important pathogen. Diagnosis is possible from faeces examination (Taylor et al 2007f).

Nematodirus lamae is a parasite of the small intestine of camelids. It is found in South America and presumed to have a direct lifecycle and has not been reported as being pathogenic. Other members of the genus are pathogenic in young ruminants (Fowler 1992; Taylor et al 2007h).

Spiculopteragia peruvianus is found in camelids in Peru. Little is known about the parasite and it must be assumed that it has a direct life cycle similar to other trichostrongyles (Fowler 1992). Since it has not been described as a significant pathogen it is probably of minor importance.

Thelazia californiensis occurs in the eyes of a variety of animals including llamas. It is is transmitted by face flies that feed on the excretions of animals' eyes. It may cause excessive lacrimation and mild conjunctivitis (Fowler 1992). Diagnosis is by careful direct examination of the eyes.

Parelaphostrongylus tenuis is a nematode found commonly in the venous sinuses and subdural space in the brain of white-tailed deer in eastern North America (Duffy et al 2002). The nematode is not zoonotic.

The infestation is subclinical in white-tailed deer that are the natural definitive host. However, many other animal species are susceptible to infestation, including all other North American cervids, cattle, sheep, goats, horses, llamas and alpacas (Tanabe et al 2007; Reinstein et al 2010). All are aberrant hosts, exposed by living in close proximity to white-tailed deer.

Infestation of abberant hosts may cause severe neurological signs and death. Llamas particularly appear to be sensitive to the development of severe clinical signs (ADDL 2008).

In natural infestations, infective larvae have never been observed in the faeces of camelids (Tanabe et al 2007). Further, experimentally infested llamas did not produce patent infestations. The authors concluded that the risk of llamas transporting patent infestations to non-endemic areas was slight since it is highly unlikely that the life cycle is completed in a llama host (Rickard et al 1994). Camelids are therefore concluded to be aberrant dead-end hosts and *P. tenuis* is not a hazard in the commodity.

With the exception of *Thelazia*, all the parasites of concern can be diagnosed by demonstration of parasite eggs in faeces. All species can be treated with standard anthelmintic drenches. Although other nematodes will be identified in camelids in the future, diagnosis and treatments for these parasites will probably follow the standard methods used for nematodes (Taylor et al 2007e).

34.1.4.2. Trematodes

The following trematodes do not occur in New Zealand, but could be associated with camelids:

Dicrocoelium dendriticum

Eurytrema pancreaticum

Fasciola gigantica

Fascioloides magna

Dicrocoelium dendriticum is a small fluke found in the bile ducts of sheep goats, cattle deer and rabbits and, occasionally, horses and pigs. Two intermediate hosts are required; a snail and an ant. Infestation may be subclinical but if severe, anaemia emaciation and oedema may be seen. Diagnosis is confirmed by identification of eggs in faeces. A number of effective drugs are available for treatment, these include netobimin, albendazole and praziquantel (Taylor et al 2007b).

Eurytrema pancreaticum is a small fluke found in pancreatic ducts and sometimes in bile ducts. It is found in cattle, buffaloes, sheep, goats, pigs, camels, and humans in South America, Asia and Europe. Snails and grasshoppers are required for completion of the lifecycle. Low and moderate infestations produce little effect on the host and diagnosis is usually made as an incidental finding at a post-mortem examination. There is no specific treatment for the parasite (Taylor et al 2007c).

Fasciola gigantica is a liver parasite found in tropical and subtropical countries only. It has a lifecycle similar to that of the common liver fluke *Fasciola hepatica* and requires a snail intermediate host to complete its lifecycle. An important snail intermediate host for *F. gigantica* is *Lymnae radix auricularia* which is present in New Zealand (Spencer 2009). However, the trematode is confined to tropical and subtropical countries with distinct wet and dry seasons (Asia, Africa, Southern Europe and the USA) and it seems improbable that it could establish in temperate climate countries such as New Zealand. Diagnosis can be made by identification of eggs in faeces samples. Treatment is similar to that for *F. hepatica* using triclabendazole (Taylor et al 2007a).

Fascioloides magna is a liver fluke of a variety of domestic farm animals. It is predominantly found in Canada, the USA and Mexico, southwestern Europe and South Africa. A number of freshwater snails act as intermediate hosts. Diagnosis is made from clinical signs and faeces examination. In Canada and the Great Lakes area, the parasite is commonly carried by deer. Cattle become infested when grazing the same pastures as deer. Treatment and diagnosis are similar to those used for other liver flukes (Taylor et al 2007d).

34.1.4.3. Cestodes

Cestodes of camelids (except *Echinococcus granulosus*) that do not occur in New Zealand are:

Monezia benedeni

Thysaniezia spp.

Monezia benedeni is not included in the list of parasites found in New Zealand but *Monezia* sp. is listed as occurring in cattle, goats, red deer, fallow deer, alpaca and llama

(McKenna 2009). Oribatid mites are the intermediate hosts for *M. benedeni*. The species is considered to be apathogenic. Diagnosis is based on the presence of proglotids and demonstration of characteristic eggs voided with faeces. A number of drugs are available for treatment (Taylor et al 2007g).

Thysaniezia spp. have been reported in llamas but little is known about them and they are not considered to be of clinical significance (Fowler 1992). *Thysaniezia ovilla* has been described in camels. Oribatid mites are intermediate hosts and the parasites are not considered to be pathogenic. Readily identifiable mature tapeworm segments can be found voided with faeces (Taylor et al 2007i).

The parasites described above are unlikely to constitute a complete listing since the amount of literature on parasitology in camelids is limited. However, infestation with the vast majority of internal parasites can be diagnosed by examination of faeces. To provide the highest likelihood of identifying parasites, faeces should be examined carefully to find tapeworm segments or whole parasites and should also be examined by flotation and sedimentation techniques and larval culture (Taylor et al 2007i).

34.1.5. Hazard identification conclusion

Full risk assessment for all species of parasites is not necessary. Instead general diagnostic methods should be adopted to identify parasites, larvae or their eggs in faeces. Since there are several parasites of camelids that do not occur in New Zealand parasites are considered to be potential hazards in the commodity.

34.2. RISK ASSESSMENT

34.2.1. Entry assessment

There are a large number of internal parasites that may be present in camelids. Since the animals for importation may not show any obvious clinical signs of infestation, the likelihood of entry is assessed to be non-negligible.

34.2.2. Exposure assessment

Imported animals infested with internal parasites will void parasite eggs in their faeces and thus contaminate the environment resulting in infestation of other animals including a variety of farm animal species. Therefore, the likelihood of exposure is assessed to be non-negligible.

34.2.3. Consequence assessment

Introduction of new parasite species could result in infestations of animals that leads to reduced animal growth and production losses. Wild animals could also become infested with some parasite species. *Fasciola gigantica* is the only known parasite of camelids able to infest humans. Therefore, there would not be any consequences for human health since this trematode would not be able to establish here.

34.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and internal parasites are classified as hazards in the commodity. Therefore, risk management measures may be justified.

34.3. RISK MANAGEMENT

34.3.1. Options

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

- Methods of diagnosis are similar for almost all internal parasites.
- Parasites found in imported animals should be identified to species level, where possible.
- Treatments are available for all parasites and the best possible drugs should be selected for each situation.
- The only *Code* chapter relating to internal parasites is for *Echinococcus granulosus,* which is covered in a separate chapter in this risk analysis.

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

1. No restrictions could be placed on the importation of camelids provided they meet the commodity definition.

N.B. This measure implies that internal parasites of camelids that are not already endemic are rare and are generally of minimal importance.

2. Camelids could be held in quarantine for 4 weeks and treated twice, for nematode, trematode and cestode parasites shortly before introduction into quarantine and again 2 weeks after introduction into quarantine. At the second treatment different anthelmintics to those used at the first treatment, could be used. Anthelmintics used could be known to be broadly effective against all classes of internal parasites.

N.B. This measure relies solely on treatment without testing to determine whether it was efficacious.

3. Camelids could be treated 2 weeks before entry into quarantine.
4. Immediately after entry animals could be treated using different anthelmintics to those used at the first treatment.
5. Two weeks after the second treatment faeces samples could be re-tested. Testing should include macrosopic examination of faeces and flotation, sedimentation and larval culture methods.
6. If any animals are still infected with parasites the procedure could be repeated until they are found to be parasite free.

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35. Mites, lice and fleas

35.1. HAZARD IDENTIFICATION

35.1.1. Aetiological agents

The following from Table 1 *Organisms of potential concern* have been identified as preliminary hazards:

Psoroptes ovis (mite)
Microthoracius mazzai, *Microthoracius minor*, *M. praelongiceps* (sucking lice)
Vermipsylla spp. (flea)

35.1.2. OIE list

None of these parasites are listed in the *Code*.

35.1.3. New Zealand status

The mite *Psoroptes ovis* is the causative agent of the condition 'sheep scab' that was eradicated from New Zealand many years ago (officially declared free in 1893). It is listed as a notifiable organism (MAF 2009).

No record was found that sucking lice of the genus *Microthoracius* or fleas of the genus *Vermipsylla* occur in New Zealand.

35.1.4. Epidemiology

The mite *Psoroptes ovis* infests a wide range of host animals including camelids, sheep, cattle, goats, horses and rabbit. It occurs world-wide and particularly in South America and Europe. The notable exceptions are Australia and New Zealand that are both officially free.

The lifecycle of *Psoroptes ovis* from egg to adult takes about 10 days to complete. Larvae hatch from the egg, moult through two nymphal stages and finally moult to become the adult form. The mites are non-burrowing and feed superficially on the skin surface. A hypersensitivity reaction occurs in the host to the presence of the mite. This results in inflammation and scratching that causes self-trauma to the host and damage to the fleece. The constant irritation leads to weight-loss and in some cases death of the host (Taylor et al 2007b).

Populations of *Psoroptes* mites on the host animal decline in warm weather, leaving residual populations in sites such as the axilla, groin, infra-orbital fossa and inner surface of the pinna and auditory canal where mites localise (Taylor et al 2007b).

Transmission is primarily through physical contact. However, transmission can occur via the environment. Survival off the host may be up to 18 days in favourable environmental conditions of ambient temperatures and high humidity. The potential re-introduction of the mite into New Zealand could occur through the importation of animals (Taylor et al 2007b).

Treatment of animals infested with *Psoroptes ovis* is available and effective. These treatments include pour-on application of organophosphates, synthetic pyrethroids, or the use of injectable ivermectins (Taylor et al 2007b).

Microthoracius mazzai (González-Acuña et al 2007), *Microthoracius minor* and *Microthoracius praelongiceps* (González-Acuña et al 2007) have been described in camelids. *Microthoracius cameli* is found in camels (Wernery & Kaaden 2002a). Light infestations are said to have no obvious effects, heavier infestations may cause pruritus, dermatitis and hair loss. In young animals, heavy infestations may cause anaemia (Taylor et al 2007a). The lifecycle is typical of lice. Adults lay eggs which hatch and give rise to three nymphal stages before developing into adults. The whole cycle may be completed in as little as 3 weeks. Because the eggs are resistant to insecticides treatments should be repeated at 7-10 day intervals to kill newly hatched larvae before they reach maturity and lay eggs. A large number of insecticides can be used for treatment including macrocyclic lactones, permethrin, chlorinated hydrocarbons, carbamates and organophosphates (Fowler 1992; Taylor et al 2007a). Ivermectin is not effective against biting lice but effective against sucking lice. Pour-on organophosphates are considered to give poor results (Fowler 1992). Effective penetration of the wool is necessary and treatment should preferably follow recent shearing.

Information on the infestation of camelids by fleas is sparse. Wernery and Kaaden (2002a) states that *Vermipsylla alacurt* and *V. ioffi* infest camels and camelids and quotes Fowler as the source of the information regarding camelids. However, Fowler (1998) only states that "Llama owners have described flea infestation to the author" and does not identify the genus or species of the fleas involved. Wernery and Kaaden (2002b) also states that other species such as *Ctenocephalides felis* may infest camelids. *Vermipsylla alakurt* is listed as a species of flea in one textbook (Mullen & Durden 2009). Searching electronic data-bases yielded one reference to three species of *Vermipsylla* occurring in ungulates, including camels (Zedev 1976).

Infestation of camelids by *Vermipsylla* spp. has not been well studied and is likely to be of limited significance. Infestations with other fleas such as *Ctenocephalides* spp. are not of significance to biosecurity since they are already present in New Zealand.

Typically fleas have lifecycles that can be completed in about 18 days or may take up to 6-12 months depending on temperature and humidity. Pupae can remain dormant in the environment for months. Adults can lay hundreds of eggs while on the host. Three larval and a single pupal stage occur off the host, often in the bedding material where the host lies. Treatments for fleas are similar to those for lice with the exception that since pupae may live for extended periods in the environment, elimination of infestations requires either treatment of both infested animals and their environment or treatment and removal of animals from an infected environment. It is assumed that effective control of fleas of the *Vermipsylla* genus will be similar to control measures for fleas of other genera and that they will be susceptible to the same insecticides.

35.1.5. Hazard identification conclusion

Psoroptes ovis, *Microthoracius* spp. and *Vermipsylla* spp. could infest camelids and are considered to be potential hazards on the commodity.

35.2. RISK ASSESSMENT

35.2.1. Entry assessment

Since *Psoroptes ovis*, *Microthoracius* spp. and *Vermipsylla* spp. of mites, lice and fleas respectively are known to infest camelids the likelihood that they could be introduced on the commodity is assessed to be non-negligible.

35.2.2. Exposure assessment

Introduced camelids would be mixed with New Zealand camelids and other potential host animals. Mites, fleas and lice could be transferred to other camelids and other species by close contact, in bedding and from fomites such as grooming and shearing equipment. Therefore, the likelihood of exposure is assessed to be non-negligible.

35.2.3. Consequence assessment

Re-introduction of the mite *Psoroptes ovis* could result in 'sheep scab' affecting llama, alpaca and sheep flocks with the resultant economic losses incurred from self trauma and damaged fleeces. Should control and eradication be initiated, active surveillance, movement control and compulsory treatment regimes including the destruction of flocks may be necessary to stamp-out the mite.

Lice are likely to be broadly host specific and unlikely to transfer to humans or animals other than camelids. In camelids they could be responsible for damaged fibre, skin irritation and dermatitis. Heavy infestations of young animals could cause anaemia and poor growth. Louse infestation could be spread to other camelids by movement of animals.

Vermipsylla spp. have been found on a variety of wild and domestic animals (Zedev 1976) and introduction of infested animals could lead to infestations of wild and domestic animals but it is unlikely to affect humans. Infestation of livestock could result in skin irritation and loss of condition but fleas of camelids have not been shown to be vectors of any disease agent (Wernery & Kaaden 2002b).

Since new species of mite, lice and fleas could be introduced by camelids the consequences of introduction are assessed to be non-negligible.

35.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is assessed as non-negligible and mites, fleas and lice are classified as hazards in the commodity. Therefore, risk management measures may be justified.

35.3. RISK MANAGEMENT

35.3.1. Options

The following points were considered when drafting options for the effective management of mites, fleas and lice associated with the commodity:

- New Zealand has eradicated the mite *P. ovis* that causes the condition "sheep scab" and should seek to preserve this status.

- Mites, fleas and lice of camelids cause skin irritation and damage to fibre but are not known to cause serious disease or act as vectors for disease agents.
- Steps should be taken to ensure that camelids do not introduce mites, lice and fleas into the quarantine station in which they will be isolated. Introduction of parasites into the facility could result in re-infestation of animals after successful treatment.
- There are several efficacious insecticides that can be used for the elimination of the parasites.
- There is no *Code* chapter relating to mites, fleas and lice.

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

1. Animals to be imported could be quarantined in isolation from other animals for at least 30 days before shipment; and
2. Within the 4 days before entry into the quarantine station animals could be treated with an insecticide known to be effective against mites, fleas and lice. Animals that have not been recently shorn could be shorn before treatment; and
3. Animals could again be treated with an effective insecticide 7-10 days after entry into isolation and again 10-14 days later. Different insecticides could be used at each treatment.

N.B. These measures allow for treatment with insecticides thought to be effective against mites, lice and fleas but do not provide for confirmation of the effectiveness of the treatment undertaken.

4. Animals for importation could undergo treatments as above. In addition they could be carefully inspected at the time of the second and third treatments to ensure that the previous treatments have been effective. If mites, lice or fleas are detected at the last inspection, treatments could be repeated until no parasites are found. Since populations of *Psoroptes* mites are known to leave residual populations in sites such as the axilla, groin, infra-orbital fossa and inner surface of the pinna and auditory canal, these areas could require special attention when inspecting the animal.

N.B. These measures allows for inspection as well as treatment.

5. Treatment with 5ml of diluted ivermectin solution into each ear canal and microscopic examination of saline flushings of both ear canals with negative results for *Psoroptes ovis* ear mites.

N.B. This measure is specific for residual populations of mites in the ear canals of animals and reflects Australia's level of protection for mitigating the risk of introducing *Psoroptes ovis* when importing camelids from Chile.

6. Ten days after entering pre-export isolation saline flushings of both ear canals of each animal should be examined and found to be free of evidence of *P. ovis* mites. If found then the animal should be treated with an ectoparasiticide effective against ear mites and re-examined 10 days later.

N.B. This measure reflects the current IHS conditions required when importing camelids from the USA.

7. Animals for importation could undergo all treatments and inspections as in the measure above. In addition, all hygiene measures regarding removal of bedding and cleaning of premises in the measures presented for the management of ticks could be carried out.

N.B. This measure would reduce the possibility that eggs, larvae or pupae of mites, lice and fleas surviving in bedding or on fomites that could re-infest animals that are to be imported.

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36. Ticks

36.1. HAZARD IDENTIFICATION

36.1.1. Aetiological agent

World-wide there are around 170 species of Argasidae or soft ticks and 650 species of Ixodidae or hard ticks (Allan 2001). Species known to infest camelids are discussed in Section 36.1.4.

36.1.2. OIE list

Ticks are not listed in the *Code*. However, several tick species are vectors of diseases included in the OIE list.

36.1.3. New Zealand status

There are nine species of tick in New Zealand, most of which are found on wild birds. Only one species of cattle tick, *Haemaphysalis longicornis*, is of economic importance to livestock. Five genera of ticks are listed as unwanted notifiable organisms on the Unwanted Organisms Register:

Amblyomma spp.
Boophilus spp.
Dermacentor spp.
Ixodes spp.
Rhipicephalus spp.

36.1.4. Epidemiology

Ticks cause serious economic losses. Losses are worse in hot tropical climates but also occur in countries with temperate climates. Ticks are vectors for a large number of diseases and tick toxicoses. Norval and Horak list 33 diseases and toxicoses of livestock that occur in Southern Africa (Norval & Horak 2004). The list is not complete even for Africa and does not include diseases of cats, dogs, wildlife species, and humans. Allan lists nine diseases that occur in North America (Allan 2001). Many other diseases occur in other countries. The livestock diseases carried by ticks include economically important diseases such as heartwater, babesiosis, anaplasmosis, theileriosis, and African swine fever.

World-wide losses due to tick-borne diseases and tick control have been estimated to cost several billion dollars annually (Jongejan & Uilenberg 1994). Apart from losses due to diseases carried by ticks, infestations with ticks also cause significant production losses and losses for tick control (Jonsson et al 2000; Jonsson et al 2001).

New Zealand has only one livestock tick, *Haemaphysalis longicornis*, and no significant tick-borne diseases. Many important ticks such as *Amblyomma* spp. might not be able to establish themselves in the New Zealand environment, but it is considered that New Zealand's mainly moist-temperate climate provides an ideal environment for all but the most strictly tropical or arid region tick species (Heath 2001).

Consideration of the lifecycles of ticks is important when designing programmes to prevent the entry of ticks into New Zealand.

Hard ticks (Ixodidae) have a lifecycle that is divided into four stages: egg; larva with six legs; nymphs with eight legs and no genital pore; adults with eight legs and a genital pore. Adults lay batches of several thousand eggs that hatch and the larvae climb up grass stems or other vegetation and await a passing host animal. Larvae are only pin head sized and not easily seen in grass or on an animal's body. Once they have found a host animal they move to a suitable site on the animal, attach and start ingesting blood. They are wasteful feeders and may ingest more than 100 times their own starting weight of blood (Allan 2001). Three-host tick larvae can be fully engorged within 3 days. When fully engorged the larvae moult to develop to the next stage. Three host ticks leave the host and moult off the host. Two- and one-host ticks moult on the host and then continue to feed on the same host. Mature nymphs of two-host ticks leave the host when engorged and moult off the host before finding a new host on which to develop to the adult stage. One-host ticks remain on the same host throughout larval, nymph, and adult feeding periods. Finally when the adult females are engorged they mate with a male tick while still on the host. Male ticks remain on the host and may mate repeatedly. Females are soft skinned and engorge till they are bloated, mature females of the larger species may weigh 4 grams. Male ticks have a hard dorsal shield and are much smaller. Three-host ticks such as *Rhipicephalus appendiculatus* may remain on the host animal for only 3 days while one host ticks such as *Boophilus microplus* may be on the host for about 3 weeks (Norval & Horak 2004).

Soft ticks (Argasidae) are economically less important than hard ticks but there are still several undesirable species such as *Otobius megnini* (the spinous ear tick) and *Ornithodorus savigni*. Many of the soft ticks live off the host in cracks, burrows or nests, or buried in the sand and take repeated short meals from a resting host. Therefore, soft ticks are less likely to be imported on live animals.

Many species of ticks in several countries have developed resistance to acaricides used to control them (Jongejan & Uilenberg 1994; Jonsson et al 2000; Li et al 2003; Li et al 2004; Mekonnen et al 2002).

There is not a lot of information on soft ticks that infest camelids. The spinous ear tick *Otobius megnini* is listed in two book sources (Fowler 1992; Wernery & Kaaden 2002a) and a case that resulted in brain abscessation has been recorded (Chigerwe et al 2005). Wernery and Kaaden 2002, lists *Dermacentor* spp. as infesting camelids and Fowler gives "various species of ixodid ticks". Other ticks reported from llamas include *Haemaphysalis juxtakochi* (Guglielmone et al 2005), *Amblyomma parvitarsam* (Guglielmone et al 2005; Peralta et al 1994), *Ixodes pacificus* (Barlough et al 1997), *Dermacentor accidentalis*, *D. variabilis*, *Dermacentor* spp. (Cebra et al 1996). Llamas were shown to be competent hosts for the important cattle tick *Boophilus microplus* (Aguirre et al 2000). In addition many species of ticks have been reported from camels (James-Rugu & Jidayi 2004; Lawai et al 2007; Loftis et al 2006; Wernery & Kaaden 2002b). Although extensive literature is not available on the subject it is apparent that many species of ticks can infest camelids.

36.1.5. Hazard identification conclusion

Many species of ticks that can infest camelids are competent vectors for serious tick-borne diseases. Even if they are not infected with disease agents when introduced, they would represent a threat to biosecurity. An established population of ticks would be a source of competent vectors if disease agents should be introduced at a later stage. Since a large number of tick species could be carried by camelids and tick species are widely distributed in the world they are considered to be potential hazards in the commodity.

36.2. RISK ASSESSMENT

36.2.1. Entry assessment

A large number of tick species are known to be, or are potential parasites of camelids. Tick species are widely distributed in most countries of the world and even careful inspection may fail to detect tick larvae infested camelids. Therefore, the likelihood of entry is assessed to be non-negligible.

36.2.2. Exposure assessment

Once introduced, female ticks are likely to lay large numbers of eggs that could hatch and infest animals of various species including humans. Therefore, the risk of exposure is assessed to be non-negligible.

36.2.3. Consequence assessment

If ticks establish they could be vectors or potential vectors for many diseases of domestic, wild and feral animals and humans. Apart from vector potential, there are also the direct effects of parasitism (anaemia) and toxicity (paralysis and death). The effects on human and animal health can be severe. If an exotic tick were to establish, eradication would be difficult and expensive. Therefore, the consequences of introducing ticks are assessed as non-negligible.

36.2.4. Risk estimation

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

36.3. RISK MANAGEMENT

36.3.1. Options

The following points were considered when drafting options for the effective management of ticks associated with the commodity:

1. Ticks are vectors of many disease agents.
2. Ticks can cause infested animals to lose condition resulting in production losses.
3. Once ticks have been introduced, control measures are expensive and an ongoing cost to producers.
4. Ticks have developed resistance to many acaricides. Measures to prevent introduction should not rely on acaricide treatments. Suitable management and hygiene measures can also be used to prevent introduction.
5. There is no *Code* chapter relating to ticks.

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

1. Camelids could be treated with pour-on acaricides, 7-10 days prior to entering quarantine where they could be held for 30 days; *and*
2. Animals for importation could be treated within 48 hours of entering quarantine with an insecticide/acaricide solution that is effective against ticks, applied to the animals by thoroughly wetting the entire animal including under the tail, ears, the axillary region, between the hind legs, and the interdigital spaces (e.g. using a back pack spray unit). A pour-on treatment should not be used. If unshorn, animals should be shorn before commencing treatment; and
3. Animals for importation could be treated with an acaricide within the 3 days prior to shipment.

N.B. The above three combined measures rely on treatment alone without verification of its efficacy.

4. Camelids could be quarantined and treated as above, and the animals for importation could be meticulously inspected for ticks and other ectoparasites, at least 10 days after entering PEI. If still infested, the treatment could be repeated and animals inspected again at least 10 days later. Treatments and testing could be repeated until the animals are found to be free from evidence of ticks. The ectoparasiticide could be altered if the previously used treatment has not been effective: and
5. Animals for importation could be treated with an acaricide within the 3 days prior to shipment.

N.B. The above two combined measures include visual inspection to verify that treatment has been effective.

6. Treatments and inspections could be applied as above and
7. The quarantine premises could have impervious washable floor and walls or be fenced, impervious pad without walls and surrounded by a cleared area free from vegetation. Bedding should not be straw or plant material that could contain tick eggs and larvae. Inert materials such as wood shavings or sterilised peat could be considered suitable. The animals could be fed rations that are free from potential contamination with ticks, tick eggs, larvae or nymphs. Pelleted rations could be preferred; and
8. All bedding could be removed every ten days during the quarantine period and, at this time, the walls and floor could be thoroughly cleaned, steam cleaning could be recommended, and sprayed with an acaricide.

N.B. The above three combined measures are designed to ensure that quarantine premises are free from ticks including eggs and larvae and that new parasites are not introduced in bedding or feed. Ticks on the animals will engorge and leave the hosts before the end of quarantine and be removed in bedding or destroyed by cleaning and acaricide treatment before eggs have been laid and hatched.

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37. Screwworm and other myiasis infestations

37.1. HAZARD IDENTIFICATION

37.1.1. Aetiological agent

Myiasis is a disease caused by the invasion of the tissues or open cavities (e.g. external ears, mouth, nares) of animals by dipteran larvae (Acha & Szyfres 1987). There are many species of fly that cause myiasis and camelids are susceptible to attacks from these.

The fly and blowfly species that (Wernery & Kaaden 2002) list as infesting camelids are: *Cochliomyia hominivorax, Phormia* spp., *Calliphora* spp., *Phaenicia* and *Lucilia* spp.

The nasal bot fly usually associated with cervids, *Cephenemyia* spp. (family *Oestridae*) is also listed (Wernery & Kaaden 2002).

Other flies that could cause opportunistic myiasis infestations in camelids include: *Dermatobia hominis*, (Acha & Szyfres 1987) and *Wohlfahrtia* spp.

37.1.2. OIE list

New World screwworm (*Cochliomyia hominivorax*) is listed under diseases of multiple species (OIE 2009).

37.1.3. New Zealand status

Cochliomyia hominivorax is listed as an unwanted, notifiable organism.

The following species are listed as exotic: *Calliphora albifrontalis, C. auger, C. imperialis,* and *C. nociva*.

Atherigona poecilopoda, A. excisa and *A. orientalis* are listed as unwanted (Ministry of Agriculture & Forestry 2009).

Cephenemyia spp. (cervid nasal bot fly), *Dermatobia* spp. and *Wohlfahrtia* spp. are not listed, but are considered to be exotic.

37.1.4. Epidemiology

The New World screwworm fly (NWS) *Cochliomyia homnivorax* is an obligate parasite of warm-blooded animals, including humans and rarely birds. It is a blowfly of the family *Calliphoridae*, but unlike most other species of blowfly, screwworms lay their eggs at the edges of wounds on living mammals or at their body cavities. The eggs hatch within 24 hours and the larvae (maggots) that are screw-shaped burrow into the wound in a characteristic screwworm fashion. This results in severe tissue destruction and infested wounds emit an odour that is highly attractive to other gravid female flies (Acha & Szyfres 1987). If untreated, the destructive activity of the larvae may lead to the death of the animal within a very short time.

The larvae reach maturity about 4-8 days after hatching from the egg and leave the wound, falling to the ground into which they burrow and pupate. Adult flies emerge from the pupae in 1 week (at 28°C) to 2 months time dependent on temperature and humidity (Acha & Szyfres 1987; Ausvetplan 1996). Freezing or sustained soil temperatures of 8°C or less kill the pupae (Merck 2006). The optimal temperature range for the fly is 20-30°C and this has had a major influence on their distribution. Flies will not move at temperatures below 10°C, and in the range 10-16°C they are very sluggish and probably will not mate. At no stage in the fly's lifecycle is it resistant to freezing and over-wintering in frost areas does not occur (Ausvetplan 1996).

The NWS fly is endemic in parts of Central and South America as far south as Argentina. It has been eliminated from the USA, Mexico and several Central American countries, where it was previously endemic, by use of the sterile insect technique. NWS has never established in Australia.

Dermatobia hominis, the tropical warble fly, lives in humid forested areas and is one of the most important parasites of cattle in Latin America, where it is distributed between southern Mexico and northern Argentina (Acha & Szyfres 1987). Larval stages are found in many hosts, including humans (Soulsby 1968). The adult fly fastens its eggs to different types of insects of which 49 (mostly mosquitoes and muscoid flies) have been described as vectors of *D. hominis* in Latin America. These vectors then transport the eggs to warm-blooded hosts where they hatch as the insect vector feeds. The warble fly larvae penetrate the skin of the animal within a few minutes of hatching and remain in the subcutaneous tissue for 4-18 weeks (Acha & Szyfres 1987) where they form ‘warbles’ which are connected by breathing holes through the skin to the air. When mature, the larvae leave the host and drop to the ground, burrow, and pupate (Soulsby 1968).

The gray flesh fly, *Wohlfahrtia vigil* causes cutaneous myiasis in North America. Larval stages are maggot-like in appearance and are adapted to maintain an attachment to living tissues with strongly developed oral hooks. *Wohlfahrtia vigil* is larviparous i.e. it deposits larvae (not eggs) on healthy, uninjured skin of suitable hosts. Larvae penetrate the unbroken skin and form a boil-like swelling. Development to the infective third-larval stage is usually completed in 9-14 days. The parasites then drop to the ground and pupate, approximately 11-18 days later, depending on temperature.

W. magnifica occurs in the European and African Mediterranean area, the Middle East, Russia and China. The fly is attracted to open wounds and, being larviparous, deposits larvae in these wounds. It is an important disease of sheep in southern parts of Russia (Acha & Szyfres 1987).

The following larval dipterans are often referred to as facultative myiasis-producing flies: *Musca domestica* (the house flies), *Calliphora*, *Phaenicia*, *Lucilia*, and *Phormia* spp. (the blowflies or bottleflies) and *Sarcophaga* spp. (the flesh flies). Their adult stages are synanthropic flies, i.e. they are often associated with human dwellings and readily fly from faeces to food. Larval stages are usually associated with skin wounds of any animal that has become contaminated with bacteria or with a matted hair coat contaminated with faeces. In facultative myiasis, the adult flies are attracted to a moist wound, skin lesion, or soiled hair coat. As adult female flies feed in these sites, they lay eggs. The eggs hatch,

producing larvae that move independently about the wound surface, ingesting dead cells, exudate, secretions, and debris, but not live tissue. This condition is known as fly strike. Unless appropriate therapy is administered, the infested animal may die, generally from shock, intoxication, or infection. A distinct, pungent odour permeates the infested tissue and the affected animal (Merck 2006).

Cephenemyia spp. found in North America are nasal bots of cervids belonging to the family *Oestridae*. Reported infestations in camelids are very rare and they are considered abberant hosts. It is not known if *Cephenemyia* spp. can complete their lifecycle in camelids (Wernery & Kaaden 2002).

In New Zealand, *Cephenemyia trompe* was reported from the nasopharynx of an imported Canadian wapiti in 1982. This parasite had not been found previously and presumably it was introduced from Canada, with the wapiti (Mason 1982). Other wapiti from the same shipment were treated with ivermectin, and establishment of the parasite did not occur. There have been no further reports of *Cephenemyia* spp. occurring in New Zealand.

37.1.4.1. Diagnosis

Myiasis is easily diagnosed from a careful clinical examination of the skin, any open wounds and around body cavities.

Cochliomyia homnivorax (NWS) produces a particularly vile myiasis. Female flies are attracted to open wounds, and larvae burrow deep into the wound which results in severe tissue destruction. Infested wounds emit an odour that attracts more flies.

The presence of a superficially situated dermal swelling with a central opening, especially if more than one is present, may lead to a tentative diagnosis of myiasis due to *D. hominis* (Soulsby 1968).

The first indication that an animal is infested with *Wohlfahrtia vigil* is exudation of serum and matting of the hair coat over the site of penetration. The presence of a dermal swelling with a central opening may lead to a tentative diagnosis of myiasis due to *W. vigil*. On the third or fourth day, the larvae produce abscess-like lesions. The hair coat often becomes parted over the summit of the lesions and reveals an opening 2-3 mm in diameter. The posterior aspect of the larva is visible in these openings, through which it breathes. The penetration of the skin by the larvae, their development in the subcutaneous tissues, and secondary bacterial infection produce intense irritation and inflammation (Merck 2006).

Camelids infested with *Cephenemyia* spp. show clinical signs of head shaking, sneezing and coughing with or without a nasal discharge. Granulomatous swellings may occur in the nasopharynx and nasal cavities in chronic cases forcing the animal to breathe through an open mouth (Wernery & Kaaden 2002).

37.1.4.2. Treatment

Treatment and control measures for myiasis in camelids are limited. With most myiasis infestations, removing maggots from existing deep tissue pockets may need surgical

exploration, debriding and flushing. This would involve sedating or anaesthetising the animal (Merck 2006).

Larvae of *Wohlfahrtia* species can be removed by coating the breathing pore with a thick, viscous compound, such as heavy oil, or liquid paraffin. Clogging the pore causes the larva to become hypoxic and leave the cavity in search of oxygen (Merck 2006).

Camelids infested with *Cephenemyia* spp. have been treated with ivermectin administered subcutaneously, or with rafoxanide or trichlorfon administered as a drench which effectively removes the larvae (Wernery & Kaaden 2002).

37.1.5. Hazard identification conclusion

Myiasis is a debilitating, serious disease of warm-blooded animals. *Cochliomyia hominivorax* (NWS) is listed as an unwanted, notifiable organism. All the listed agents that cause myiasis are considered to be potential hazards.

37.2. RISK ASSESSMENT

37.2.1. Entry assessment

Camelids coming from endemically affected countries could be infested with myiasis. Myiasis is generally clinically evident on careful examination of the skin, particularly under any matted fleece, open wounds and around body cavities. Some infested camelids may display clinical signs of respiratory disease, such as sneezing or nasal discharges indicating possible *Cephenemyia* infestation.

Pre-export veterinary examination on the day of travel that certifies the animal is clinically healthy should exclude such infested animals from travel. However, the animal may be infested immediately prior to departure, or en-route to New Zealand with clinically undetectable larvae. The likelihood that infested animals will be imported with myiasis is therefore assessed to be extremely low but non-negligible.

37.2.2. Exposure assessment

New Zealand animals could become infested if larvae in infested imported animals were able to complete their lifecycle and the resulting adult flies mated sucessfully. However, it is unlikely larvae in imported camelids would leave their hosts naturally since infestation is clinically obvious, and veterinary treatment would most likely be sought. If veterinary intervention did not occur, New Zealand's climate is probably not suitable for the pupal development of the tropical myiasis fly species. Facultative myiasis-producing flies such as *Lucilia* spp, of which some are already present in New Zealand, are more likely to establish. The likelihood that New Zealand animals will be exposed to exotic myiasis is therefore assessed to be non-negligible.

37.2.3. Consequence assessment

If the parasites were to establish it would have severe economic effects on New Zealand's primary industries due to production losses and treatment costs.

Occasional infestations of humans would require medical treatment. The consequences for feral and wild animals are likely to be non-negligible since parasites are not host specific, generally affecting any warm-blooded mammal and birds.

Since there could be severe negative effects on animal production and cases of myiasis in many animal species, including humans, the consequences are assessed to be non-negligible.

37.2.4. Risk estimation

Since entry, exposure and consequence assessments are all assessed to be non-negligible, risk is estimated to be non-negligible and myiasis infestations are classified as hazards in the commodity. Therefore, risk management measures may be justified.

37.3. RISK MANAGEMENT

37.3.1. Options

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

- The establishment of flies that cause myiasis could affect many livestock species and result in production losses from loss of condition and in severe cases, death.
- NWS is an OIE listed disease, and the *Code* makes recommendations for the safe importation of animals. Therefore, all camelids introduced from countries that are infested with screwworm could be subjected to measures that are based on those international recommendations.
- The OIE recommendations would also mitigate the risks from other dipteran larval infestations. The *Code* recommendations are:

Article 8.8.1.

Recommendations for importation from countries considered infested with new world or old world screwworm

for domestic and wild mammals

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

1. immediately prior to loading, the animals to be exported have been inspected, on the premises of origin, by an *Official Veterinarian*. After inspection for wounds with egg masses or larvae of new world or old world screwworm, any infested animal has been rejected for export;
2. immediately prior to entering the quarantine pens in the *exporting country*:
   a. each animal has been thoroughly examined for infested wounds, under the direct supervision of an *Official Veterinarian*, and that no infestation has been found in any animal; and
   b. any wounds have been treated prophylactically with an officially approved oily larvicide at the recommended dose; and
   c. all animals have been dipped, sprayed, or otherwise treated, immediately after inspection, with a product officially approved by the *importing* and *exporting countries* for the control of new world or old world screwworm, under the supervision of an *Official Veterinarian* and in conformity with the manufacturer's recommendations;
3. at the end of the quarantine and immediately prior to shipment for export:

a. all animals have been re-examined for the presence of infestation and all animals have been found free of infestation;
b. all wounds have been prophylactically treated with an approved oily larvicide under the supervision of an *Official Veterinarian*;
c. all animals have been prophylactically treated again by dipping or spraying as in point 2 above.

Article 8.8.2.

Quarantine and transportation recommendations

1.The floor of the quarantine area and the *vehicles* must be thoroughly sprayed with an officially approved larvicide before and after each use.
2.The transit route must be the most direct, with no stopover without prior permission of the *importing country.*

Article 8.8.3.

Post importation inspection

1.On arrival at the importation point, all animals must be thoroughly inspected for wounds and possible new world or old world screwworm infestation under the supervision of an *Official Veterinarian*.
2.The bedding material of the *vehicle* and the quarantine area should immediately be gathered and burned following each consignment.

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

1. Animals for export could be subjected to a close inspection of the skin for wounds with egg masses or larvae immediately prior to shipment. Where necessary animals should be shorn. Only animals that are free from infestation and that have a dry, unsoiled and unmatted fleece would be eligible for shipment; *and*
2. The inspection could be repeated at the arrival point in New Zealand. This inspection could identify any infestation acquired en route and be integrated with tick inspections.
3. Camelids could be quarantined for a period of 30 days pre-export and treated in accordance with OIE recommendations (Articles 8.8.1. and 8.8.2.) followed by a post-importation inspection as recommended (Article 8.8.3.).

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38. Weeds and seeds

38.1. HAZARD IDENTIFICATION

38.1.1. Aetiological agent

All plant material including seeds.

38.1.2. OIE list

Not listed.

38.1.3. New Zealand status

Organisms of concern are exotic plants and seeds.

38.1.4. General considerations

Weeds and seeds could be found attached to the fibre and hair of camelids. Large seed heads and pieces of plant material would be easily visible and could be removed before shipment but small seeds would be difficult to detect.

Seeds are specifically adapted to survive unfavourable environmental conditions and most will at least survive from one growing season to another. Many will survive for several years and germinate when favourable conditions occur. Most seeds are highly resistant to dehydration, particularly those from plants adapted to survival in desert or hot dry climates and most seeds retain viability better in dry conditions but some are specifically adapted to remain viable in water. *Mimosa glomerata* seeds survived 221 years in the herbarium of the Museum National d'Histoire Naturelle in Paris. *Lupinus arcticus* seeds frozen in a lemming's burrow that was dated as 10,000 years old germinated within 48 hours when placed in favourable conditions (Encyclopædia Britannica 2008). Some seeds are adapted to environments subjected to periodic fires and survive or are activated by fires. Others are adapted to be dispersed by water including those that are adapted to salt water.

Weed seeds can survive passage through an animal's digestive system and be passed out in faeces (Katovich et al undated). A review of passage times for weed seeds in the digestive tract of herbivores (Barton and Williams 2001) concluded that, to avoid the importation of most unwanted seeds in the digestive tracts of herbivorous animals destined for New Zealand, they should be fed a seed-free diet for at least 10 days prior to their arrival in New Zealand. Cattle passed about half the seeds ingested by 2.5 days and most of them by 7 days. A few seeds were retained for up to 1 month in cattle. It is expected that passage times for weed seeds in the digestive tracts of camelids would not be longer than those for cattle. The wide variation around the mean seed-passage times was attributed to many factors such as individual animal effects, whether or not the animal was pregnant, and food intake. The most widely reported factor with potential applicability to quarantine protocol was faster seed-passage time in animals fed a high-quality diet.

An import risk analysis of the importation of weed species by live animals recommended that animals should be held, pre-shipment, in areas free of weed species and fed on clean pasture or high quality feed (MAF 1999). During transport, provision of high quality feed with little or no weed species contamination, or feed that has been treated in such a way as to render seeds non-viable, would mitigate the risks associated with the importation of live

animals. Faeces produced during transport should be safely disposed of, either en route or on arrival in New Zealand.

38.1.5. Hazard identification conclusion

It is concluded that weed seeds or plant material could be introduced attached to animal's fibres/coats or in their faeces. Therefore weed seeds and plant material are considered to be potential hazards in the commodity.

38.2. RISK ASSESSMENT

38.2.1. Entry assessment

Seeds and plant material could be introduced attached to the animal itself or within its digestive tract. The entry assessment is therefore considered to be non-negligible.

38.2.2. Exposure assessment

Weed seeds could become detached from hair or voided in faeces. They are generally resistant to most environmental conditions and may remain dormant until conditions are favourable for germination. Therefore, the likelihood that seeds could germinate and grow if released into a suitable environment is considered to be non-negligible.

38.2.3. Consequence assessment

As a result of the entry of seeds or plant material, exotic noxious weeds could be introduced and become established with subsequent deleterious effects on the environment and the economy. The consequence assessment is therefore considered to be non-negligible.

38.2.4. Risk estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and weed seeds, plants, and plant material associated with the commodity are classified as hazards. Therefore, risk management measures may be justified.

38.3. RISK MANAGEMENT

38.3.1. Options

The following points were considered when drafting options for the effective management of weeds and seeds associated with the commodity:

- The risks of introducing seeds and plant material attached to camelids and hair could be greatly reduced if they have been closely shorn and/or groomed and kept free from visible contaminating plant material.
- The measures suggested to control the introduction of ticks could greatly reduce the likelihood of introducing weed seeds. Housing the animals for a period of 30 days in facilities with clean impervious flooring on bedding that is not made up of grass hay or straw will reduce the risk of contamination with weed seeds. Suitable

bedding materials include wood shavings, sawdust or sterilised peat. During the 30 days in quarantine the plant material eaten by the animals before they were introduced into the quarantine facilities, will have been either digested or passed out in the faeces. Regular removal of faeces and soiled bedding will reduce the likelihood that weed seeds will be present in faeces that could contaminate animal coats.

- Feeding of processed pellets that are essentially free of weed seeds could ensure that the animals do not ingest new burdens of weed seeds. Heat treatment used in the production of pellets will reduce the number of viable seeds.
- There is nothing in the *Code* relating to hitch hiker weeds and plants associated with animals.

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

1. Animals that are presented for loading could be required to be short shorn and well groomed and free from any visible weeds, seeds or plant material.
2. To ensure minimal risk of introducing weed species of concern to New Zealand, animals could be held, pre-shipment, in areas free of weed species and fed on clean pasture or high quality feed. During transport, provision of high quality feed with little or or no weed species contamination or feed that has been treated in such a way as to render seeds non-viable will mitigate the risks associated with the importation of live animals. Dung produced during transport should be safely disposed of, either enroute or on arrival in New Zealand.

N.B. This reflects the risk management section of the weed seeds import risk analysis (MAF 1999).

3. Animals could be fed a high quality, seed-free diet to speed passage time in the digestive tract, for at least 10 days prior to their arrival in New Zealand.

N.B. There may be deleterious health effects of such a diet in llamas and alpacas. They do not do well on pelleted rations and may lose weight and develop diarrhoea.

4. Measures suggested in the Tick section for the management of risk associated with ticks could also be considered for the control of weeds, seeds, and plant material.

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