

Import Risk Analysis: Live sheep and goats from Australia

FINAL



ISBN 978-0-478-33828-7 (Print)
ISBN 978-0-478-33829-4 (Online)

13 February 2009

This page is intentionally blank

MAF Biosecurity New Zealand
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand

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

Policy and Risk
MAF Biosecurity New Zealand



Import Risk Analysis: Live sheep and goats from Australia

FINAL

ISBN 978-0-478-33828-7 (Print)
ISBN 978-0-478-33829-4 (Online)

13 February 2009

Approved for general release

A handwritten signature in black ink that reads 'Christine Reed'.

Christine Reed
Manager, Risk Analysis
MAF Biosecurity New Zealand

This page is intentionally blank

Contents

Executive Summary	1
1. Introduction	2
2. Commodity Definition	2
3. Scope	2
4. Risk Analysis Methodology	2
5. Akabane Disease	13
6. Bluetongue	16
7. Palyam Virus Infections	20
8. Ross River and Barmah Forest Virus Infections	23
9. Anthrax	26
10. Melioidosis	30
11. Infections with Mycoplasmas and related Mollicutes	32
12. Salmonellosis	41
13. Leptospirosis	47
14. Q Fever	51
15. Theileriosis	56
16. Ticks	57
17. Lice	63
18. Mites	66
19. Internal Parasites	68
20. Hydatidosis	72
21. Weed Seeds, Plants, and Plant Materials	77

Contributors to this risk analysis

1. Authors

Bob Worthington	Contractor to Biosecurity New Zealand	Biosecurity New Zealand, Wellington
Stephen Cobb	Senior Adviser, Risk analysis (Animals)	Biosecurity New Zealand, Wellington

2. Internal peer review

Howard Pharo	Team Leader, Risk analysis (Animals)	Biosecurity New Zealand, Wellington
Lincoln Broad	Senior Adviser, Risk analysis (Animals)	Biosecurity New Zealand, Wellington
Gillian Mylrea	Team Manager, Import health standards (Animals)	Biosecurity New Zealand, Wellington
Sandy Toy	Senior Adviser, Risk analysis (Indigenous fauna)	Biosecurity New Zealand, Wellington
José Derraik	Senior Adviser, Risk analysis (Human health)	Biosecurity New Zealand, Wellington

3. External scientific review

Geoff Ryan	Ruminant Section Manager	Department of Agriculture, Fisheries and Forestry – Australia
Dave West	Professor, Sheep Health and Production	Massey University, New Zealand

Executive Summary

The risk of introduction of disease-causing organisms through the importation of live sheep and goats from Australia is considered in this risk analysis. Options are presented for sanitary measures to manage the risk associated with the following hazards:

- Bacillus anthracis*
- Exotic *Mycoplasma* spp.
- Exotic *Salmonella* spp.
- Leptospira* spp.
- Coxiella burnetii*
- Ticks
- Lice
- Internal parasites
- Echinococcus granulosus*
- Weed seeds, plants, and plant material

Risk management options include quarantine, sourcing of animals from trustworthy sources, treatment, vaccination, diagnostic testing, implementation of legislative principles to prevent the establishment of an agent, and a prohibition on importation of live animals as appropriate for each case. A range of options of varying stringency has been suggested for each hazard.

To prevent the re-introduction of *Echinococcus granulosus* it may be appropriate not to allow the importation of live animals and rely solely upon the importation of germplasm to ensure New Zealand's access to improved ovine genetics. This measure would also effectively manage the risks associated with *Bacillus anthracis*, internal parasites, ticks, lice, and weed seeds, plants and plant material.

1. Introduction

Scrapie is the disease of major concern to the sheep industry when considering importation of sheep and goats. Hence importation of live sheep has for many years been restricted to importations from Australia which is considered to be free from this disease. The importation of germplasm has been permitted under very tightly controlled conditions (MacDiarmid 1993).

The purpose of this risk analysis is to re-assess the risks involved in importing live sheep from Australia and to present options for the effective management of the identified risks.

2. Commodity Definition

The commodities under consideration are sheep (*Ovis aries*) and goats (*Capra hircus*) from Australia.

3. Scope

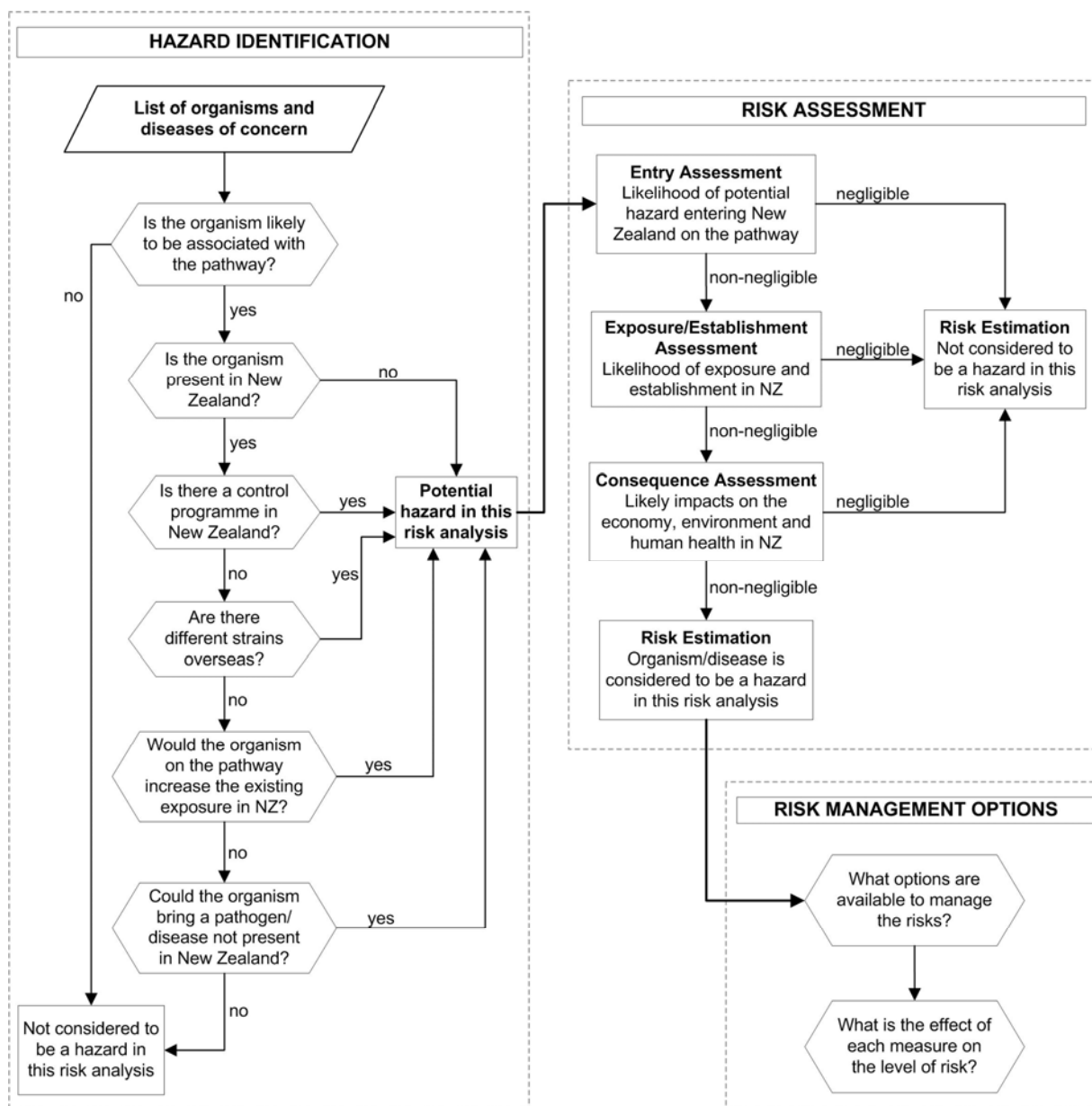
The analysis is carried out in accordance with the MAF Biosecurity New Zealand policy that risk analyses should provide the relevant technical data on which Import Health Standards (IHSs) will be based. IHSs may be required for any commodity at the discretion of the Director General as defined in Section 22 of the Biosecurity Act of 1993. The risk analyses considers organisms that may cause unwanted harm to people, the environment, and the economy as defined in the Biosecurity Act. Specifically this risk analysis covers the infectious or parasitic pathogens of sheep and goats and any weeds, plants, plant seeds or parts of plants that may be associated with them. Genetic diseases and other risk factors that may be of commercial importance to importers have not been considered. The risk analysis is qualitative and applies only to sheep and goats to be imported from Australia.

4. Risk Analysis Methodology

The methodology used in this risk analysis is described in MAF Biosecurity New Zealand's *Risk Analysis Procedures – Version 1* (Biosecurity New Zealand 2006) and is consistent with the guidelines in Section 1.3 of the OIE *Terrestrial Animal Health Code* (OIE 2007a).

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

Figure 1. The risk analysis process.



4.1. PRELIMINARY HAZARD LIST

The hazard identification process begins with the collation of a list of organisms likely to be associated with the commodities. Table 1 shows these organisms, together with some of the key information considered. For this risk analysis a list was made comprising all the diseases of sheep and goats that were listed by OIE in the *Code*, as well as other diseases mentioned in the following sources:

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

Veterinary Medicine. Radostits OM, Gay CC, Hinchcliff KW, and Constable PD, 10th edition (2007). Bailliere Tindall, ISBN 1: 0-7020 2777 4.

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

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

Diseases included in the risk analysis for the importation of germplasm from sheep and goats¹.

Weeds and plants, their seeds, and parts of plants were included at the request of the Department of Conservation.

¹ See: www.biosecurity.govt.nz/files/pests-diseases/animals/risk/risk-analysis-sheep-goat-genetic-material.pdf

Table 1. Preliminary hazard list for live sheep and goats from Australia

Organism	OIE List?	Zoonotic?	New Zealand Status	Australian status	Of concern?
Viruses					
Akabane and related simbu viruses	No	No	Exotic	Endemic (Cybinski et al 1978)	Yes
Aujeszky's disease virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Bluetongue virus	Yes	No	Exotic (OIE 2007b)	Endemic(OIE 2007b)	Yes
Border disease virus	No	No	Endemic*	Endemic (Lim and Carnegie 1984)	No
Borna disease virus (unclassified)	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic (Geering et al 1995)	No
Caprine arthritis encephalitis virus	Yes	No	Endemic (OIE 2007b)	Endemic (OIE 2007b)	No
Sheep/goat pox virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Coronavirus	No	No	Endemic (Durham et al 1979; Vermunt and Parkinson 2000b)	Presumed endemic	No
Crimean-Congo haemorrhagic fever virus	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic (Swanepoel and Burt 2004)	No
Foot and mouth disease virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Ovine pulmonary adenocarcinoma virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Louping ill and related viruses	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic (Geering et al 1995)	No
Maedi-visna lentivirus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Nairobi sheep disease virus and related viruses	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Ovine and caprine papillomaviruses	No	No	Endemic (Shortridge and Cordes 1971)	Presumed endemic	No
Palyam serogroup viruses	No	No	Exotic**	Endemic	Yes
Parainfluenza virus 3	No	No	Endemic*	Presumed endemic	No
Peste des petits ruminants virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Rabies	Yes	Yes	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Rift Valley fever virus	Yes	Yes (Swanepoel and Coetzer 2004)	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Rinderpest virus	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Ross River virus	No	Yes	Exotic	Endemic	Yes
Rotavirus	No	No	Endemic (Vermunt and Parkinson 2000a)	Presumed endemic	No
Sheep/goat pox (Capripoxvirus)	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No

Table 1. (continued)

Organism	OIE List?	Zoonotic?	New Zealand Status	Australian status	Of concern?
Viruses (continued)					
Vesicular stomatitis virus	Yes	Yes	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Wesselsbron disease virus	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic (Geering et al 1995)	No
Bacteria including <i>Mycoplasma</i> spp.					
<i>Acholeplasma laidlawii</i>	No	No	Endemic (Belton 1990; Belton 1996)	Presumed endemic	No
<i>Acholeplasma oculi</i>	-	No	Exotic**	Presumed endemic	Yes
<i>Actinobacillus lignieresii</i>	No	No	Endemic *	Presumed endemic	No
<i>Actinobacillus seminis/Histophilus ovis</i>	No	No	Endemic*	Endemic (Swanepoel and Coetzer 2004)	No
<i>Arcanobacter pyogenes</i>	No	No	Endemic*	Presumed endemic	No
<i>Bacillus anthracis</i>	Yes	Yes	Exotic (OIE 2007b)	Endemic (OIE 2007b)	Yes
<i>Brucella melitensis</i>	Yes	Yes	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Brucella ovis</i>	Yes	No	Endemic*	Endemic (Burgess 1982)	No
<i>Bordetella parapertusis</i>	No	No	Endemic (Anonymous 1975a; Shrubbs 1998)	Presumed endemic	No
<i>Branhamella ovis</i>	No	No	Endemic (Shrubbs 1998)	Presumed endemic	No
<i>Burkholderia pseudomallei</i>	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Endemic	Yes
<i>Campylobacter fetus</i> subsp. <i>intestinalis</i>	No	No	Endemic*	Endemic (Broadbent 1975)	No
<i>Campylobacter fetus</i> subsp. <i>jejuni</i>	No	No	Endemic*	Endemic (Shanker et al 1982)	No
<i>Clostridium tetani</i>	No	Yes	Endemic*	Endemic	No
<i>Clostridium botulinum</i>	No	Yes	Endemic*	Endemic	No
<i>Corynebacterium ovis</i>	No	No	Endemic *	Presumed endemic	No
<i>Corynebacterium renale</i>	No	No	Endemic (Anonymous 1975a; Anonymous 1975b)	Presumed endemic	No
<i>Dermatophilus congolense</i>	No	No	Endemic*	Presumed endemic	No
<i>Dichelobacter nodosus</i>	No	No	Endemic*	Endemic	No
<i>Erysipelothrix rhusiopathiae</i>	No	No	Endemic*	Presumed endemic	No
<i>Escherichia coli</i> (virulence plasmids)	No	Variable	Endemic*	Endemic	No
<i>Fusobacterium necrophorum</i>	No	No	Endemic*	Presumed endemic	No
<i>Haemophilus somni</i>	No	No	Endemic*	Presumed endemic	No
<i>Listeria monocytogenes</i>	No	No	Endemic*	Presumed endemic	No
<i>Moraxella bovis</i>	No	No	Endemic*	Endemic	No
<i>Mycobacterium avium</i> subsp. <i>paratuberculosis</i>	Yes	No?	Endemic*	Endemic (OIE 2007b)	No

Table 1. (continued)

Organism	OIE List?	Zoonotic?	New Zealand Status	Australian status	Of concern?
Bacteria including <i>Mycoplasma</i> spp. (continued)					
<i>Mycobacterium bovis</i>	Yes	Yes	Endemic (Control programme) (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Mycoplasma agalactiae</i>	Yes	No	Exotic (OIE 2007e)	Endemic***	Yes
<i>Mycoplasma arginini</i>	No	No	Endemic (Belton 1990; Belton 1996)	Presumed endemic	No
<i>Mycoplasma capricolum</i> subsp. <i>capripneumoniae</i>	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Mycoplasma conjunctivae</i>	No	No	Endemic (Motha 2003)	Presumed endemic	No
<i>Mycoplasma mycoides</i> subsp. <i>mycoides LC</i>	No	No	Endemic (Jackson and King 2002)	Endemic	No
<i>Mycoplasma ovipneumoniae</i>	No	No	Endemic (Belton 1990; Belton 1996)	Presumed endemic	No
Other Mollicutes	No	various	Exotic	Unknown	Yes
<i>Pasteurella haemolytica</i>	No	No	Endemic*	Presumed endemic	No
<i>Pasteurella multocida</i> B and E	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Pasteurella multocida</i> other than B and E	No	No	Endemic*	Presumed endemic	No
<i>Pseudomonas pyocaena</i>	No	Variable	Endemic*	Presumed endemic	No
<i>Salmonella abortus ovis</i>	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Salmonella Dublin</i>	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Endemic	Yes
<i>S. typhimurium</i> DT 104	No	Yes	Exotic, rare imported cases (Ministry of Agriculture and Forestry 2007)	Rare cases	Yes
Exotic <i>Salmonella</i> spp.	No	Yes	Exotic	Some endemic	Yes
<i>Staphylococcus</i> spp.	No	Variable	Endemic*	Presumed endemic	No
<i>Streptococcus</i> spp.	No	Variable	Endemic*	Presumed endemic	No
Spirochaetes					
<i>Borrelia burgdorferi</i>	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic	No
<i>Leptospira</i> spp.	Yes	Yes	Exotic, 6 species endemic	Endemic	Yes
Protozoal parasites					
<i>Babesia ovis</i>	No	No	Exotic (Ministry of Agriculture and Forestry 2007)	Not reported****	No
<i>Cryptosporidium</i> spp.	No	Yes?	Endemic*	Presumed endemic	No
<i>Eimeria</i> spp.	No	No	Endemic*	Presumed endemic	No
<i>Toxoplasma gondii</i>	No	Yes	Endemic*	Presumed endemic	No
<i>Theileria</i> spp. (sheep species)	No	No	Exotic (Ministry of Agriculture and Forestry 2007)	Not reported****	No

Table 1. (continued)

Organism	OIE List?	Zoonotic?	New Zealand Status	Australian status	Of concern?
Protozoal parasites (continued)					
<i>Trypanosoma</i> spp. (Tsetse transmitted)	Yes	No	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic (OIE 2007b)	No
<i>Besnoitia caprae</i>	No	No	Exotic	Exotic	No
Rickettsial and Chlamydial organisms					
<i>Anaplasma ovis</i>	No	No	Exotic (Ministry of Agriculture and Forestry 2007)	Not reported****	No
<i>Anaplasma mesaeterum</i> (Sheep species)					
<i>Chlamydophila abortus</i>	Yes	Yes	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Coxiella burnetii</i>	Yes	Yes	Exotic (OIE 2007b)	Endemic (OIE 2007b)	Yes
<i>Ehrlichia ruminatum</i>	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
<i>Eperythrozoon ovis</i>	No	No	Endemic (Gill 1990)	Presumed endemic	No
Other <i>Ehrlichia</i> spp. of sheep	No	Yes	Exotic (Ministry of Agriculture and Forestry 2007)	Exotic	No
Fungi					
<i>Trichopyton</i> spp.	No	No	Endemic*	Presumed endemic	No
<i>Zygomycosis</i> group	No	No	Endemic (Vermunt and Parkinson 2000b)	Presumed endemic	No
Arthropods					
Screwworm (<i>Cochlia hominivorax</i> , <i>Chrysomya bezziana</i>)	Yes	No	Exotic (OIE 2007b)	Exotic (OIE 2007b)	No
Ticks	No	Yes	Exotic except for one species	Endemic	Yes
Other exotic external parasites (lice and mites)	No	No	Exotic	Some species endemic	Yes
Internal parasites					
<i>Echinococcus granulosus</i>	Yes	Yes	Exotic (Pharo 2002)	Endemic (OIE 2007b)	Yes
Other exotic internal parasites	No	No	Exotic	Some species endemic	Yes
Weeds and seeds	No	No	Some species exotic	Some species endemic	Yes

* Commonly identified in New Zealand and reported in the quarterly reports of diagnostic laboratories that are published in the MAF publication *Surveillance*. For less commonly diagnosed endemic organisms a reference is given to substantiate the classification of the organisms as endemic.

** Two organisms (Palyam viruses and *Acholeplasma oculi*) have been listed as exotic on the basis that they have not been recorded as occurring in New Zealand. All other organisms listed as exotic have been classified by MAF as unwanted, exotic or notifiable organisms (Ministry of Agriculture and Forestry 2007).

*** *Mycoplasma agalactiae* has been reported from Australia but it is claimed that the Australian strains do not cause contagious agalactia (OIE 2007b).

**** Review of the literature revealed no evidence of occurrence in Australia

4.2. RISK ANALYSIS FOR IMPORTATION OF LIVE SHEEP AND GOATS FROM AUSTRALIA

For each organism identified as requiring further consideration in Table 1, the epidemiology is discussed, including a consideration of the following questions:

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

For any organism, if the answer to question one is “yes” (and the answer to question 2 is “yes” in the cases of organisms requiring a vector) and the answers to either questions three or four are “yes”, it is classified as a potential hazard requiring risk assessment.

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

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

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

Risk Assessment

- | | |
|-----------------------------|---|
| a) Release assessment - | the likelihood of the organism being imported in the commodity. |
| b) Exposure assessment - | the likelihood of animals or humans in New Zealand being exposed to the potential hazard. |
| c) Consequence assessment - | the consequences of entry, establishment or spread of the organism. |
| d) Risk estimation - | a conclusion on the risk posed by the organism based on the release, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a hazard. |

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

4.3. RISK MANAGEMENT

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

4.4. RISK COMMUNICATION

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

Following this process of consultation and review, the Imports Standards team of MAF Biosecurity New Zealand 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.

4.5. SPECIAL CONSIDERATIONS

The incubation period and the time for which an animal may remain infectious are critical parameters for determining quarantine periods. In the case of diseases for which long-term carriers are not known to occur the infectious period generally relates to the period during

which they are septicaemic (viraemic or bacteraemic). In some diseases the infectious period may be extended for long periods and sometimes for the lifetime of the animal. An animal could have been infected with a disease on the day it goes into quarantine. After the incubation period for the disease, it could then be viraemic or bacteraemic for a period that differs for each disease.

Generally animals should be quarantined for the maximum known incubation period plus the maximum period for which viraemia can last. Ideally the maximum periods would be the mean period plus three standard deviations. This would cover 99.7% of cases. However, usually the true distribution of incubation period and viraemia is not known because data are not available from a sufficiently large number of cases or because of technical difficulties in obtaining accurate data. Data quoted for the period of viraemia or bacteraemia is also unreliable because of the small number of animals that can be used in experiments to define these parameters and because the presence of viraemia is usually not measured continuously but at discrete intervals. The measurement of viraemia is also dependant on the accuracy and sensitivity of the detection method used. As the sensitivity of the methodology to detect organisms increases our perception of the length of viraemia has tended to increase.

For these reasons a conservative margin for error should be added to the best available estimates when determining quarantine periods. The margin of error added cannot be scientifically determined but relies on judgement taking into account such things as amount and perceived accuracy of the available data, type of disease and methods that were used to measure viraemia. Generally in this risk analysis suggested quarantine periods are adjusted to whole weeks or months.

For diseases in which long-term carriers are known to occur, quarantine is not a useful method to prevent the introduction of those diseases.

References

References marked * have been sighted as summaries in electronic media.

Anonymous (1975a). Laboratory reports. Invermay Animal Health Laboratory. *Surveillance*, 5, 22.

Anonymous (1975b). Laboratory reports. Palmerston North Animal Health Laboratory. *Surveillance*, 4, 14.

Belton D (1990). Mycoplasmas of sheep and goats in New Zealand. *Surveillance*, 17(2), 18-9.

Belton D (1996). Abattoir surveillance of Mycoplasmas in the lungs and udders of New Zealand goats. *Surveillance*, 23(1), 21.

Biosecurity New Zealand (2006). Risk Analysis Procedures. Ministry of Agriculture and Forestry, Wellington.

Broadbent DW (1975). Infections associated with ovine perinatal mortality in Victoria. *Australian Veterinary Journal*, 51(2), 71-4.

Burgess GW (1982). Ovine contagious epididymitis: a review. *Veterinary Microbiology*, 7(6), 551-75.

Cybinski DH, St George TD, Paul INI (1978). Antibodies to Akabane virus in Australia. *Australian Veterinary Journal*, 54(1), 1-3.

- Durham PJK, Stevenson BJ, Farquharson BC (1979).** Rotavirus and coronavirus associated with diarrhoea in domestic animals. *New Zealand Veterinary Journal*, 27(2), 30-2.
- Geering WA, Forman AJ, Nunn MJ (1995).** Exotic Diseases of Animals. Pp. Australian Government Publishing Service, Canberra.
- Gill J (1990).** An Eperythrozoon ovis outbreak in merino lambs. *Surveillance*, 17(4), 15-6.
- Jackson R, King C (2002).** Mycoplasma mycoides subsp. mycoides (Large Colony) infection in goats. A review with special reference to the occurrence in New Zealand. *Surveillance*, 29(2), 8-12.
- Lim CF, Carnegie PR (1984).** A survey of hairy shaker disease (border disease, hypomyelinogenesis congenita) in sheep. *Australian Veterinary Journal*, 61(6), 174-7.
- MacDiarmid SC (1993).** Risk analysis and the importation of animals and animal products. *Revue Scientifique et Technique OIE*, 12(4), 1093-107.
- Ministry of Agriculture and Forestry (2007).** The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 20/12/2007.
- Motha J (2003).** A serological survey for Mycoplasma conjunctivae infection in sheep and goats. *Surveillance*, 30(3), 9-10.
- Motha J, Hansen M (1997).** A serological survey for bovine respiratory syncytial virus in New Zealand. *Surveillance*, 24(4), 28.
- OIE (2004).** Handbook on Import Risk Analysis for Animals and Animal Products Pp. OIE, Paris.
- OIE (2007a).** In: OIE (ed). Terrestrial Animal Health Code. OIE, Paris http://www.oie.int/eng/normes/MCode/en_sommaire.htm, downloaded 20/12/2007.
- OIE (2007b).** Handistatus II. <http://www.oie.int/hs2/report.asp>, downloaded 20/12/2007.
- Pharo H (2002).** New Zealand declares 'provisional freedom' from hydatids. *Surveillance*, 29(3), 3-7.
- Shanker S, Rosenfield JA, Davey GR, Sorrell TC (1982).** Campylobacter jejuni: incidence in processed broilers and biotype distribution in human and broiler isolates. *Applied Environmental Microbiology*, 43(5), 1219-20.*
- Shortridge EH, Cordes DO (1971).** Neoplasms of sheep: A survey of 256 cases recorded at Ruakura Animal Health Laboratory. *New Zealand Veterinary Journal* 19(4), 55-65.
- Shrubbs O (1998).** Gram negative glucose non-fermenting bacteria in New Zealand. *Surveillance*, 25(3) 19.
- Swanepoel R, Burt FJ (2004).** Crimean-Congo haemorrhagic disease. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Pp. 1077-85. Oxford University Press, Cape Town.
- Swanepoel R, Coetzer JAW (2004).** Rift Valley fever. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Pp. 1037-70. Oxford University Press, Cape Town.
- Vermunt JJ, Parkinson TJ (2000a).** Infectious diseases of cattle in New Zealand. Part 1 - Calves and growing stock. *Surveillance*, 27(2), 3-8.
- Vermunt JJ, Parkinson TJ (2000b).** Infectious diseases of cattle in New Zealand. Part 2 - Adult animals. *Surveillance*, 27(3), 3-9.

5. Akabane Disease

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological Agent

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

5.1.2. OIE List

Not listed.

5.1.3. New Zealand Status

Exotic, unwanted organism (Ministry of Agriculture and Forestry 2007).

5.1.4. Epidemiology

Cattle and other ruminants including sheep (Charles 1994; Haughey et al 1988) and goats (Han and Du 2003) are susceptible to the virus.

Viruses in the Simbu-group are endemic in Australia (Charles 1994; Haughey et al 1988; St George and Kirkland 2004). The incubation period (infection to start of viraemia) is from 1 to 6 days (St George 1998). In non-pregnant animals infection does not lead to the development of any signs of disease and virus has been isolated from naturally infected sentinel cattle without clinical signs (Gard et al 1989). Virus crosses from maternal to foetal circulation in infected pregnant females and causes the development of malformed lambs and kids, particularly cases of arthrogryposis and hydraencephaly (Charles 1994; Parsonson et al 1977; Parsonson et al 1988; St George and Kirkland 2004). In cattle maximal damage occurs when infection takes place at about the 12th to 16th week of gestation (St George and Kirkland 2004). Once a foetus has become immuno-competent it can mount an immune reaction and damage is less apparent or does not occur. Infected calves are usually non-viable (Charles 1994). It can be assumed that sheep and goats will be maximally affected from some time before mid gestation until the foetus becomes immuno-competent at about the 65-70th day of gestation (St George and Kirkland 2004). Lambs or kids born or aborted will not be contagious and will not infect vectors.

Major epidemics of foetal malformations have been reported in cattle in Australia (St George and Kirkland 2004). Outbreaks of arthrogryposis/ hydranencephaly in Australia, involved 3-5,000 calves and in Japan 30,000 calves (St George and Kirkland 2004). Large scale outbreaks have also occurred in Israel (Brenner 2004). In Australia there is an ongoing economic loss in the tropical endemic area with at least a quarter of a million cattle at risk each year (St George and Kirkland 2004). Animals that have been exposed to the infection become immune and this leads to the establishment of a mainly immune population of animals in endemic areas. For this reason foetal abnormalities usually occur sporadically in endemically infected areas but sero-conversion in subclinically infected animals is common (Cybinski and St George 1978; Cybinski et al 1978; Fukutomi et al

2003; St George and Kirkland 2004). Outbreaks of foetal abnormalities occur in areas where naïve cattle are exposed to the virus. This has typically occurred when weather conditions favourable to long distance dispersal of *Culicoides brevitarsis* into areas where this vector is not normally present, have occurred (Murray 1987).

Akabane and related viruses have been isolated from *Culicoides* spp. (midges) and mosquitoes (St George and Kirkland 2004). In one study akabane virus was isolated from non-blood fed *Ochlerotatus* spp. in northern Vietnam (Bryant et al 2005). At least five species of the *Ochlerotatus* (*Aedes*) genus are known to occur in New Zealand (Holder et al 1999). However mosquitoes have not been proven to be biological vectors of the virus. In Australia the disease occurs in tropical areas. Serologically positive animals were confined to the northern half of New South Wales and areas further north and all serologically positive animals were from the area infested with *Culicoides brevitarsis*. *Culicoides* spp. are assumed to be the vectors of the virus in Australia (St George and Kirkland 2004). Murray states that *Culicoides brevitarsis* is the only know vector of Akabane virus and provides circumstantial evidence that outbreaks of the disease were correlated with movements of *Culicoides brevitarsis* into non-endemic areas (Murray 1987).

It is concluded that in Australia infection is confined to areas where *Culicoides brevitarsis* occurs and this insect can be assumed to be the principle vector.

5.1.5. Hazard Identification Conclusion

Since Akabane and other Simbu viruses are not present in New Zealand and are unwanted organisms (Ministry of Agriculture and Forestry 2007) they are classified as potential hazards in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry Assessment

Since the disease is endemic in northern Australia and virus has been isolated from animals without clinical signs, the likelihood of imported animals from northern Australia being viraemic is considered to be non-negligible.

5.2.2. Exposure Assessment

Viraemic animals would not be contagious to contacted animals. Since *Culicoides* spp. are not present in New Zealand, the likelihood that the virus could establish in New Zealand is considered to be negligible.

5.2.3. Risk Estimation

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

References

References marked * have been sighted as summaries in electronic media.

Brenner J (2004). Congenital bovine abnormalities outbreaks of large scale in Israel. Israel Veterinary Medical Association, 59(1-2), http://www.isrvma.org/article/59_1_3.htm, downloaded 20/12/2007.

Bryant JE, Crabtree MB, Nam VS, Yen NT, Duc HM, Miller BR (2005). Isolation of arboviruses from mosquitoes in northern Vietnam. American Journal of Tropical Medicine and Hygiene, 73(2), 479-3.*

Charles JA (1994). Akabane virus. The Veterinary Clinics of North America. Food animal practice, 10(3), 525-46.*

Cybinski DH, St George TD (1978). A survey of antibody to Aino virus in cattle and other species in Australia. Australian Veterinary Journal, 54(8), 371-3.

Cybinski DH, St George TD, Paull NI (1978). Antibodies to Akabane virus in Australia. Australian Veterinary Journal, 54(1), 1-3.

Fukutomi T, Ngai M, Okuda K, Akashi H, Hada M, Kayahara Y, Hatano Y (2003). Antigenic characteristics of the Akabane viruses isolated from sentinel cattle in Okayama Prefecture. Journal of the Japan Veterinary Medical Association, 57, 2101-5.*

Gard GP, Melville LF, Shorthose JE (1989). Investigations of bluetongue and other arboviruses in the blood and semen of naturally infected bulls. Veterinary Microbiology, 20(4), 315-22.

Han D, Du H (2003). Congenital abnormalities in Korean native goat with Akabane virus. Journal of Veterinary Clinics, 20(3), 427-30.*

Haughey KG, Hartley WJ, Della-Porta AJ, Murray MD (1988). Akabane disease in sheep. Australian Veterinary Journal, 65(5), 136-40.

Holder P, Brown G, Bullians M (1999). The mosquitoes of New Zealand and their animal disease significance. Surveillance, 26(4), 12-5.

Ministry of Agriculture and Forestry (2007). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 20/12/2007.

Murray MD (1987). Akabane epizootic in New South Wales: Evidence for the long-distance dispersal of the biting midge *Culicoides brevitarsis*. Australian Veterinary Journal, 64(10), 305-8.

Parsonson IM, Della-Porta AJ, Snowdon WA (1977). Congenital abnormalities in newborn lambs after infection of pregnant sheep with Akabane virus. Infection and Immunity, 15(1), 254-62.

Parsonson IM, McPhee DA, Della-Porta AJ, McClure S, McCullagh P (1988). Transmission of Akabane virus from the ewe to the early fetus (32 to 53 days). Journal of Comparative Pathology, 99(2), 215-27.

St George TD (1998). Akabane. Foreign Animal Diseases . "The Gray Book", http://www.vet.uga.edu/vpp/gray_book02/fad/aka.php, downloaded 8/1/2008.

St George TD, Kirkland PD (2004). Diseases caused by Akabane and related Simbu-group viruses. In: Coetzer JAW, Tustin RC (eds). Infectious Diseases of livestock. Pp. 1029-36. Oxford University Press, Oxford.

6. Bluetongue

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological Agent

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

6.1.2. OIE List

Listed

6.1.3. New Zealand Status

Exotic, notifiable (Ministry of Agriculture and Forestry 2007).

6.1.4. Epidemiology

Bluetongue virus can infect many ruminant species. It occurs in most tropical and sub-tropical countries. It is absent in southern hemisphere countries south of 34° south, including New Zealand, and northern hemisphere countries north of 53° north (OIE 2007). The virus occurs in northern Australia but large areas of southern, western and central Australia are free from the disease. Serotypes 1 and 21 occur widely in the infected parts of Australia but serotypes 3, 9, 15, 16, 20 and 23 have only been reported from the top end of northern Australia. The Australian national animal health monitoring programme monitors the presence of bluetongue in Australia in sentinel herds of cattle. The endemic area varies from season to season depending on climatic and other changes. However large areas of Australia remain uninfected. Maps showing the current situation are available on the internet and are updated regularly (Animal Health Australia 2006).

In Africa and other countries the virus causes disease mainly in sheep, occasionally in goats, and rarely in cattle and deer. In most other species infections are subclinical. The virus is transmitted by *Culicoides* spp. (midges) and outbreaks of the disease usually occur in late summer to autumn when midges are most active. In temperate areas outbreaks of disease cease with the advent of winter, when *Culicoides* spp. become inactive. In sheep mortality varies from 2% to 30% and many inapparent infections occur. The severity of the disease varies between breeds (Verwoerd and Erasmus 2004).

6.1.5. Hazard Identification Conclusion

Since bluetongue virus is an exotic notifiable organism (Ministry of Agriculture and Forestry 2007), it is classified as a potential hazard in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry Assessment

Sheep usually remain viraemic for 6 to 8 days and rarely up to 14 days (Verwoerd and Erasmus 2004). However, it was reported that in Lesbos sheep and goats the viraemic

period lasted up to 54 days but not up to 64 days (Koumbati et al 1999). Many adult sheep in endemic areas will be immune to the serotypes of virus circulating in the area, but young animals and animals newly imported into the endemic area are likely to be susceptible. Older sheep will also be susceptible to new serotypes of virus introduced to an area. In summer and for a period of approximately 60 days after *Culicoides* spp. become inactive at the onset of winter, susceptible animals may be viraemic. In countries where many strains of virus are endemic a few strains usually dominate in any one season but as the population becomes immune to these strains the dominant strains are replaced by other strains that then become dominant. However, in Australia apart from serotypes 1 and 21 other strains have remained confined to the far north. The likelihood that bluetongue virus could be introduced with infected sheep is considered to be non-negligible if they originated from endemic areas of Australia, but negligible if imported from areas that are well removed from the infected or surveillance zones defined by the Australian national animal health monitoring programme.

6.2.2. Exposure Assessment

Infected sheep could not infect other ruminants in contact with them as the disease can only be transmitted by *Culicoides* spp. An annual *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al 1991). Seroconversion has never occurred in sentinel cattle to bluetongue, epizootic haemorrhagic disease, Akabane, or Palyam viruses which are all transmitted by *Culicoides* spp. In a typical year *Culicoides* spp. were not found in 15,000 insects collected from light traps (Motha et al 1997). The *Culicoides* monitoring programme has continued up to the present time with annual reports of the serology programme appearing in the MAF *Surveillance* magazine. Therefore it would not be possible for animals in contact with imported animals to become infected with the virus.

Infected sheep may remain viraemic for about 60 days after infection (Koumbati et al 1999). It is not known whether rams excrete the virus in their semen. However, bulls can excrete the virus in their semen while they remain viraemic (Bowen et al 1983; Howard et al 1985; Parsonson et al 1981). Therefore it seems likely that rams could also excrete the virus in their semen and the likelihood of exposure of ewes with which they have mated or which have been inseminated with a viraemic ram's semen is non-negligible.

6.2.3. Consequence Assessment

Female sheep and goats that have mated with an infected ram or inseminated with his semen could become infected and could remain viraemic for about 60 days. However, these animals are unlikely to develop clinical signs and would not be infectious for ruminants they were in contact with. The virus could only be transmitted to *Culicoides* vectors and these vectors are not present in New Zealand (see Section 4.2.2). In addition the *Code* states that countries that are south of 34° S and are not adjacent to a country not having a bluetongue virus free status may be considered free from bluetongue. New Zealand is entirely south of 34° S. In addition New Zealand has carried out surveys that show that it is free from *Culicoides*. Therefore New Zealand is free from both *Culicoides* spp. and bluetongue virus. Furthermore the *Code* states that "A bluetongue virus free country in which surveillance and monitoring has found no evidence that *Culicoides* likely to be competent bluetongue virus vectors are present, will not lose its free status through the importation of vaccinated, seropositive or infective animals, or semen or embryos/ova from infected countries" (OIE 2007).

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

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

Bluetongue virus could not be transmitted or establish in New Zealand and, provided that ongoing arbovirus sentinel and *Culicoides* surveillance programmes continue to demonstrate freedom from competent vectors, the consequences of importing the virus are considered to be negligible.

6.2.4. Risk Estimation

Because the consequence assessment is negligible, the risk estimate for bluetongue virus is negligible and it is not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

References

References marked * have been sighted as summaries in electronic media.

Animal Health Australia (2007). National Arbovirus Monitoring Program (NAMP). <http://www.namp.com.au/>, downloaded 20/12/2007.

Bowen RA, Howard TH, Entwistle KW, Pickett BW (1983). Seminal shedding of bluetongue virus in experimentally infected mature bulls. *American Journal of Veterinary Research*, 44(12), 2268-70.

Howard TH, Bowen RA, Pickett BW (1985). Isolation of bluetongue virus from bull semen. *Progress in Clinical and Veterinary Research*, 178, 127-34.

Koumbati M, Mangana O, Nomikou K, Mellor PS, Papadopoulos O (1999). Duration of bluetongue viraemia and serological responses in experimentally infected European breeds of sheep and goats. *Veterinary Microbiology*, 64(4), 277-85.

Ministry of Agriculture and Forestry (2007). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 20/12/2007.

Motha J, Hansen M, Irwin G (1997). Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. *Surveillance*, 24(4), 18-9.

OIE (2007). Bluetongue. Chapter 2.2.13. Terrestrial Animal Health Code http://www.oie.int/eng/normes/MCode/en_chapitre_2.2.13.htm, downloaded 20/12/2007.

Parsonson IM, Della-Porta AJ, McPhee DA, Cybinski DH, Squire KRE, Standfast HA, Uren MF (1981). Isolation of bluetongue virus serotype 20 from the semen of an experimentally-infected bull. *Australian Veterinary Journal*, 57, 252-3.

Ryan TJ, Frampton ER, Motha MXJ (1991). Arbovirus and arbovirus vector surveillance in New Zealand. *Surveillance*, 18(5), 24-6.

Verwoerd DW, Erasmus BJ (2004). Bluetongue. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of livestock*. Pp. 1201-20. Oxford University Press, Oxford.

7. Palyam Virus Infections

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological Agent

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

7.1.2. OIE List

Not listed.

7.1.3. New Zealand Status

Exotic organism not listed as unwanted.

7.1.4. Epidemiology

The Palyam serogroup of the orbiviruses are represented by a large number of viruses that occur in Australia, Africa, and Asia (Swanepoel 2004). There is some confusion about the identification of some of the viruses and further new viruses are likely to be found in the future. Most of what is known about the viruses applies to cattle, but neutralizing antibody has been found in sheep and goats (Swanepoel 2004). Because specific evidence about sheep is lacking this review focuses on cattle and it is assumed that the information is applicable to sheep and goats. The main vectors for the viruses are *Culicoides* spp. but Palyam viruses have also been isolated from ticks in Africa and mosquitoes in India (Swanepoel 2004). In one review 15 viruses were listed (Swanepoel 2004), others have been reported (Doyle and Walton 1992). Large numbers of isolations of arboviruses including many Palyam viruses have been made from the blood of naturally infected, subclinical cattle and *Culicoides* midges in South Africa and Australia (Cybinski and St George 1982; Gard et al 1989; Gard et al 1988a; Gard et al 1988b; Littlejohns et al 1988; Nevill et al 1992; Theodoridis et al 1979). Although the viruses usually cause mild or subclinical infections they have been associated with abortions in Zimbabwe. Kasba virus was associated with congenital abnormalities such as hydranencephaly and cerebellar hypoplasia in calves in Japan (Goto et al 1988; Miura et al 1990). Similar congenital abnormalities were reported from Australia (Kirkland et al 1992). After infection with Kasba virus, Muria, Goto, Kubo and Kono reported that cattle were consistently viraemic for 2 weeks and intermittently viraemic for 8 weeks (Swanepoel 2004). An arbovirus and *Culicoides* surveillance programme has been in operation in New Zealand since 1991 (Ryan et al 1991). In a typical year seroconversion did not occur to bluetongue, epizootic haemorrhagic disease, Akabane and Palyam viruses in samples from 10 sentinel cattle from each of 17 herds and *Culicoides* spp. were not found in 15,000 insects collected from light traps (Motha et al 1997). The *Culicoides* monitoring programme has continued up to the present time with results of the serology programme reported regularly in the MAF Surveillance magazine. No seroconversion has been detected in sentinel cattle and no *Culicoides* have been trapped.

7.1.5. Hazard Identification Conclusion

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

7.2. RISK ASSESSMENT

7.2.1. Entry Assessment

Palyam viruses occur commonly in northern Australia and viruses have been isolated from cattle in these regions. Since the vectors for Bluetongue and Palyam viruses are similar, Palyam viruses will be confined to the areas defined in the Australian National Animal Health Monitoring programme as bluetongue infected or surveillance areas. Maps showing the current situation are available on the internet and are updated regularly (Animal Health Australia 2007). The likelihood that sheep imported from areas in which bluetongue occurs could be infected with Palyam viruses is considered to be non-negligible. However, large areas of Australia remain free from bluetongue and the likelihood that sheep imported from these areas would be infected with Palyam viruses is considered to be negligible.

7.2.2. Exposure Assessment

Sheep or goats infected with Palyam viruses will not be contagious and will not infect contacted sheep or goats. They could infect *Culicoides* spp. that could then transmit the viruses. However, since *Culicoides* are not present in New Zealand the likelihood of infection of competent vectors and transmission to New Zealand animals is considered to be negligible.

7.2.3. Risk Estimation

Because the exposure assessment is negligible, the risk estimate for Palyam viruses is negligible and they are not classified as hazards in the commodity. Therefore, risk management measures are not justified.

References

References marked * have been sighted as summaries in electronic media.

Animal Health Australia (2007). National Arbovirus Monitoring Program (NAMP). <http://www.namp.com.au/>, downloaded 20/12/2007.

Cybinski DH, St George TD (1982). Preliminary characterization of D'Aguiar virus and three Palyam group viruses new to Australia. *Australian Journal of Biological Science*, 35(3), 343-51.*

Doyle KA, Walton TE (1992). An overview and perspective on orbivirus disease prevalence and occurrence of vectors in Australia and Oceania. In: Osborne BI (Editor), *Bluetongue, African horse sickness and related orbiviruses: Proceedings of the Second International Symposium*. CRC Press, Boca Raton, USA, pp. 44-57.

Gard GP, Melville LF, Shorthose JE (1989). Investigations of bluetongue and other arboviruses in the blood and semen of naturally infected bulls. *Veterinary Microbiology*, 20(4), 315-22.

Gard GP, Shorthose JE, Weir RP, Walsh SJ, Melville LF (1988a). Arboviruses recovered from sentinel livestock in northern Australia. *Veterinary Microbiology*, 18(2), 109-18.

Gard GP, Weir RP, Walsh SJ (1988b). Arboviruses recovered from sentinel cattle using several virus isolation methods. *Veterinary Microbiology*, 18(2), 119-25.

Goto Y, Miura Y, Kono Y (1988). Serologic evidence for the etiologic role of Chuzan virus in an epizootic of congenital abnormalities with hydranencephaly-cerebellar hypoplasia syndrome of calves in Japan. *American Journal of Veterinary Research*, 49(12), 2026-9.

Kirkland PD, Cybinski DH, Walker DH, Uren MF, Blok J (1992). Congenital defects in animals in Australia - the role of arboviruses. In: Manderson LH (Editor), *Arbovirus research in Australia. Proceedings of the Fifth Symposium*. CSIRO Division of Tropical Animal Production. Indooroopilly, Queensland, Australia, Brisbane, Australia.

Littlejohns IR, Burton RW, Sharp JM (1988). Bluetongue and related viruses in New South Wales: isolations from, and serological tests on samples from sentinel cattle. *Australian Journal of Biological Science*, 41(4), 579-87.

Miura Y, Kubo M, Goto Y, Kono Y (1990). Hydranencephaly-cerebellar hypoplasia in a newborn calf after infection of its dam with Chuzan virus. *Nippon Juigaku Zasshi*, 52 (4), 689-94.

Motha J, Hansen M, Irwin G (1997). Continued freedom from arbovirus infections and arbovirus vectors in New Zealand. *Surveillance*, 24(4), 18-9.

Nevill EM, Erasmus BJ, Venter GJ, Walton TE (1992). A six year survey of viruses associated with *Culicoides* biting midges throughout South Africa (Diptera: Ceratopogonidae). In: Osburn BI (Editor), *Bluetongue, African horse sickness and related orbiviruses. Proceedings of the Second International Symposium*. CRC Press, Boca Raton, USA.

Ryan TJ, Frampton ER, Motha MXJ (1991). Arbovirus and arbovirus vector surveillance in New Zealand. *Surveillance*, 18(5), 24-6.

Swanepoel R (2004). Palyam serogroup orbivirus infections. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of livestock*. Pp. 1252-55. Oxford University Press, Oxford.

Theodoridis A, Nevill EM, Els HJ, Boshoff ST (1979). Viruses isolated from *Culicoides* midges in South Africa during unsuccessful attempts to isolate bovine ephemeral fever virus. *Onderstepoort Journal of Veterinary Research*, 46(4) 191-8.

8. Ross River and Barmah Forest Virus Infections

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological Agent

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

8.1.2. OIE List

Not listed.

8.1.3. New Zealand Status

Exotic.

8.1.4. Epidemiology

Ross River and Barmah Forest viruses are mosquito-borne alphaviruses that occur in Australia. Ross River virus also occurs in Papua New Guinea and the Solomon Islands (Harley et al 2001; Russell 2002; Russell and Doggett 2006). They are zoonotic viruses and are not known to cause clinical disease in any domestic animals except for rare cases in horses (Azuolas et al 2003; Studdert et al 2003). Approximately 5,000 cases of Ross River virus infection are notified annually in Australia (Harley et al 2001; Russell 2002; Russell and Doggett 2006). Virus has been isolated from at least 30 species of mosquitoes and transmission has been demonstrated from at least 13 species (Harley et al 2001). Mosquitoes involved in transmission of the disease include the major vector *Culex annulirostris* in freshwater habitats, and *Aedes vigilax* and *Aedes camptorynchus* in northern and southern coastal regions. Other species involved in transmission include *Aedes normanensis*, *Coquillettidia linealis*, and *Aedes notoscriptus*. Recently another mosquito, *Verrallina funerea*, has been added to the list of known competent vectors (Jeffery et al 2006). Based mainly on serological evidence, the reservoir hosts of the virus are believed to be macropods such as kangaroos and wallabies (Russell 2002; Russell and Doggett 2006; Vale et al 1991). However, viral isolations from naturally infected vertebrates have only been recorded in eight cases including two cases from macropods and two from horses (Harley et al 2001).

In Australia infections with Barmah Forest virus occur less commonly than infections with Ross River virus and little is known about the hosts of the virus (Russell and Doggett 2006). Effects of infection with both viruses are similar and include subclinical infection, a transient rash and mild illness, polyarthritis, and chronic illness. Recovery may occur in a few weeks but sometimes signs may persist for months or years (Harley et al 2001; Russell 2002).

The virus is normally confined to Australia, Papua New Guinea, and the Solomon Islands. In the latter two countries the virus may be introduced periodically from Australia (Russell 2002). A massive outbreak that occurred in the Pacific region in 1979-80 involved outbreaks in Fiji, American Samoa, the Cook Islands, and New Caledonia and probably also Tonga, Kiribati, and Western Samoa. The outbreak seems to have been started by a single traveler from Australia infecting mosquitoes in Fiji (Harley et al 2001; Russell

2002). Since the virus is known to be transmitted by *Aedes aegypti* and *Aedes albopictus* the potential exists for outbreaks of disease to occur in countries where these species of mosquitoes are present.

Ross River and Barmah Forest viruses have not occurred in New Zealand. Two exotic species of mosquitoes have become established in New Zealand; *Ochlerotatus notoscriptus*, a probable vector of Ross River virus (Russell and Doggett 2006) and *Ochlerotatus camptorhynchus* a known vector of the virus (Derraik and Calisher 2004). However, *Ochlerotatus camptorhynchus* is the subject of an eradication campaign, the outcome of which remains uncertain. Possible reservoir hosts are wallabies, which occur in some areas, and possums (Derraik and Calisher 2004). Possums have been shown to have antibody against the virus but isolation of virus from naturally occurring cases has not been demonstrated (Harley et al 2001). Experimental infection of possums resulted in high viraemia, sufficient to infect mosquitoes, for less than 48 hours after infection and they did not become carriers of the virus (Boyd et al 2001). The role played by possums in the persistence of the disease therefore remains speculative and the role they could play in New Zealand is unknown.

Sheep have been found to have antibody to Ross River virus (Cloonan et al 1982; Doherty et al 1966). There are no records of isolation of virus from naturally infected sheep or evidence that sheep can act as reservoirs of the virus. Sheep that were experimentally infected with the virus were viraemic for 2-5 days with virus titres of up to log 5 TCID 50% /ml. They did not remain carriers of the virus. In another study, lymph draining from the site of infection was infected with virus for up to 36 hours. This data indicates sheep may be transiently infected with low titres of virus but do not remain carriers of virus. In Australia, a country that has in recent times had populations of more than 100 million sheep, it seems highly unlikely that an epidemiological link between sheep and human infection would have gone unrecognised if it existed. Outbreaks of the disease have occurred in urban areas and a link between sheep farmers or people in close contact with sheep has not been suggested. Although most authorities suggest that macropods are the major host species, this has not been clearly established (Harley et al 2001). The disease was possibly carried to Fiji and the Pacific basin by a single infected human and sustained in a human mosquito/cycle during the outbreak of disease that occurred there. Other authors have suggested that humans may act as sources of infection during an epidemic but that the disease may require an animal host for establishment of the virus (Harley et al 2001).

It has been estimated that approximately 100 humans viraemic with Ross River virus travel between Australia and New Zealand every year (Kelly-Hope et al 2002). The number of overseas visitors that are residents of Australia that traveled to New Zealand in 2005 was 874,736 (Statistics-New-Zealand 2007). In addition many New Zealand residents that have visited Australia return to New Zealand each year. In comparison only about 10 to 20 permits to import sheep from Australia are issued each year, these figures are available from the annual reports published in MAF's Surveillance magazine.

8.1.5. Hazard Identification Conclusion

There is no evidence that sheep act as maintenance hosts of the Ross River or Barmah Forest viruses. Therefore these viruses are not classified as potential hazards in the commodity.

References

References marked * have been sighted as summaries in electronic media.

Azuolas JK, Wishart E, Bibby S, Ainsworth C (2003). Isolation of Ross River virus from mosquitoes and from horses with signs of musculo-skeletal disease. *Australian Veterinary Journal*, 81(6), 344-7.

Boyd AM, Hall RA, Gemmell RT, Kay BH (2001). Experimental infection of Australian brushtail possums, *Trichosurus vulpecula* (Phalangeridae: Marsupialia), with Ross river and Barmah forest viruses by use of a natural mosquito vector system. *American Journal of Tropical Medicine and Hygiene*, 65(6), 777-82.*

Cloonan MJ, O'Neill BJ, Vale TG, Carter IW, Williams JE (1982). Ross River virus activity along the south coast of New South Wales. *Australian Journal of Experimental biology and Medicine*, 60(6), 701-6.*

Derraik JG, Calisher CH (2004). Is New Zealand prepared to deal with arboviral diseases? *Australian and New Zealand Journal Public Health*, 28(1), 27-31.

Doherty RL, Gorman BM, Whitehead RH, Carley TG (1966). Studies of arthropod-borne virus infections in Queensland. V. Survey of antibodies to group A viruses in man and other animals. *Australian Journal of Experimental biology and Medicine*, 44(4), 365-77.*

Harley D, Sleight A, Ritchie S (2001). Ross river virus transmission, infection and disease: a cross-disciplinary review. *Clinical Microbiology reviews*, 14(4), 909-32.*

Jeffery JA, Kay BH, Ryan PA (2006). Role of *Verrallina funerea* (Diptera: Culicidae) in transmission of Barmah Forest virus and Ross River virus in coastal areas of eastern Australia. *Journal of Medical Entomology*, 43(6), 1239-47.*

Kelly-Hope LA, Kay BH, Purdie DM, Williams GM (2002). The risk of Ross River and Barmah Forest virus disease in Queensland: implications for New Zealand. *Australian and New Zealand Journal of Public Health*, 26(1), 69-77.*

Russell RC (2002). Ross River virus: Ecology and distribution. *Annual Review of Entomology*, 47, 1-31.*

Russell RC, Doggett SL (2006). Ross river and Barmah forest.
<http://medent.usyd.edu.au/fact/ross%20river%20&%20barmah%20forest.htm>, downloaded 20/12/2007.

Statistics-New-Zealand (2007). Tourism and migration 2005
<http://www.stats.govt.nz/NR/rdonlyres/FF45C870-E441-44E6-9BBE-323A03AA68D4/0/Table201.xls>,
downloaded 20/12/2007.

Studdert MJ, Azuolas JK, Vasey JR, Hall RA, Ficorilli N, Huang JA (2003). Polymerase chain reaction tests for the identification of Ross River, Kunjin and Murray Valley encephalitis virus infections in horses. *Australian Veterinary Journal*, 81(1-2), 76-80.

Vale TG, Spratt DM, Cloonan MJ (1991). Serological evidence of arbovirus infection in native and domestic mammals on the south coast of New South Wales. *Australian Journal of Zoology*, 39, 1-7.*

9. Anthrax

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological Agent

Bacillus anthracis.

9.1.2. OIE List

Listed.

9.1.3. New Zealand Status

Exotic, notifiable disease last diagnosed in 1954 (Ministry of Agriculture and Forestry 2007).

9.1.4. Epidemiology

Anthrax is a bacterial disease of most warm-blooded vertebrates including man. The disease occurs in Australia with the latest outbreak reported in February 2007 (Barrett 2007). However, anthrax is a rare disease in Australia and is only reported sporadically. It was estimated that the probability that an Australian animal would be infected with anthrax at the time of slaughter was 9.94×10^{-7} (MacDiarmid 1993). New Zealand has been free from the disease for about 50 years (Gill 1992).

The infectious agent is a spore forming bacillus that can survive in the spore state in suitable soils for many decades. In 1999 an outbreak occurred in Australia on farms where the disease had not occurred for about 100 years. On these properties earthworks in relation to an irrigation scheme possibly resulted in disturbance of old burial sites of cattle (Turner et al 1999a; Turner et al 1999b). A related spore-forming bacillus has been cultivated from palaeozoic slate plugs believed to be 500 million years old (De Vos 1994). *Bacillus anthracis* multiplies in infected animals and if a carcass is opened, it sporulates resulting in contamination of soil and the environment. In unopened carcasses the organism does not sporulate and is destroyed by putrefaction (De Vos and Turnbull 2004). The disease is not directly transmissible from animal to animal and infection is believed to be associated with ingestion of contaminated soil or other infected material. Biting flies may carry the infection but they were not considered to be important in the transmission of the disease in an outbreak in Australia (Turner et al 1999a). Blowflies may be important in the spread of the disease when they have been feeding on infected carcasses (De Vos and Turnbull 2004). Infection through skin wounds and abrasions may also occur and is a common route of infection for humans (De Vos and Turnbull 2004). In some circumstances infection can occur by inhalation (wool sorter's disease and bioterrorism in humans). Carriers of the disease may occur in partially immunized cattle that recover from natural infection and in some other circumstances (De Vos 1994). However, carriers are not usually described by most authors and their occurrence is ignored in the *Code*. Therefore the occurrence of carriers must be considered controversial and, if they do occur, rare.

The incubation period probably varies from one to 14 days and in the peracute form in susceptible species the course of the disease is only a few hours (De Vos and Turnbull 2004). For purposes of international trade the Code gives the incubation period as 20 days (OIE 2007). In the acute form of the disease, death usually occurs within 48 hours (Blood and Radostits 1989). Sub-acute and chronic forms of the disease occur in less susceptible animals such as pigs and carnivores (De Vos and Turnbull 2004).

Efficient live spore vaccines are available for control of the disease. The vaccine strain developed by Sterne (Sterne 1937) is usually used. It is a rough strain that has lost plasmid pX02 which codes for the bacterial capsule. The vaccine is non-pathogenic in sheep and goats and provides good immunity for about a year (De Vos and Turnbull 2004).

9.1.5. Hazard Identification Conclusion

Anthrax is an exotic, notifiable (Ministry of Agriculture and Forestry 2007c) and zoonotic disease and is therefore classified as a potential hazard in the commodity.

9.2. RISK ASSESSMENT

9.2.1. Entry Assessment

If imported directly into New Zealand, sheep and goats could be in the incubation period of the disease which could be up to 14 days. Therefore the likelihood of entry is considered to be non-negligible.

9.2.2. Exposure Assessment

Imported animals could die from the disease and if their carcasses were opened the organism could contaminate the environment, particularly soil, with spores which could survive for many years. Animals coming into contact with the infected environment created by an infected carcass, even many years after the introduction of the infected animals, could become infected with the disease. Therefore the likelihood of exposure is considered to be non-negligible.

9.2.3. Consequence Assessment

Once an infected site has been established the organism could be spread by animals that become infected with the disease and are transported to other sites where they die. Therefore *Bacillus anthracis* could become established and lead to animal mortality and the need for vaccination to control the disease.

Since anthrax is a zoonotic organism, once the disease became established, sporadic cases of human disease could occur. These cases would require treatment and some fatalities could be expected.

Since a wide range of animals, especially ruminants can be infected with the organism, cases of anthrax with further contamination of the environment could occur in feral animals such as deer and pigs.

Bacillus anthracis is a zoonotic organism that could infect animals and humans causing fatalities and further contamination of the environment. The consequences of introducing the organism are therefore considered to be non-negligible.

9.2.4. Risk Estimation

Because the entry, exposure, and consequence assessments are all non-negligible, the risk estimate is non-negligible and *Bacillus anthracis* is classified as a hazard in the commodity. Therefore risk measurement measures can be justified.

9.3. RISK MANAGEMENT

9.3.1. Options

The incubation period of the disease is short and carriers of the disease are rare, therefore a suitable period of quarantine could be an effective method for preventing the introduction of the organism. Anthrax occurs only rarely in Australia. Efficient methods of vaccination against *Bacillus anthracis* are available. Carriers of this organism are rare and are ignored by the *Code*.

There is a negligible risk of anthrax associated with the importation of germplasm (Ministry of Agriculture and Forestry 2005) and the importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

The *Code* states that “there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs” (OIE 2007). The *Code* chapter on Anthrax contains the following recommendations (Article 2.2.1.2.) relating to the importation of animals:

Veterinary Authorities of importing countries should require (for ruminants, equines and pigs), 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 options could therefore be considered in order to effectively manage the risk associated with *Bacillus anthracis* in the commodity:

- Sheep and goats could be imported without restrictions.
- Animals could be vaccinated, not less than 20 days and not more than 6 months prior to shipment.
- Sheep and goats could be quarantined for at least the 20 days immediately prior to shipment.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

- Barrett B (2007).** Anthrax, human, bovine - Australia(Victoria). Promed Mail, Archive number 2007.0219.0626, <http://www.promedmail.org/pls/promed>, downloaded 8/1/2008.
- Blood DC, Radostits OM (1989).** Anthrax. Veterinary Medicine. Pp. 592-6. Balliere Tindall, London.
- De Vos V (1994).** Anthrax. In: Coetzer JAW, Thomson GE, Tustin RC (eds). Infectious diseases of livestock. Pp. 1262-89. Oxford University Press, Cape Town, Oxford, New York.
- De Vos V, Turnbull PCB (2004).** Anthrax. In: Coetzer JAW, Tustin RC (eds). Infectious Diseases of Livestock. Pp. 1788-818. Oxford University Press, Cape Town.
- Gill J (1992).** Anthrax - still history after all these years. Surveillance, 20(121-2).
- MacDiarmid SC (1993).** Risk analysis and the importation of animals and animal products. Revue Scientifique et Technique, OIE, 12(4), 1093-107.
- Ministry of Agriculture and Forestry (2005).** Import risk analysis: sheep and goat genetic material. Biosecurity New Zealand, Wellington, New Zealand.
<http://www.biosecurity.govt.nz/files/pests/risk/risk-analysis-sheep-goat-genetic-material.pdf>
- Ministry of Agriculture and Forestry (2007).** The Unwanted Organisms Register.
<http://mafusp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.
- OIE (2007).** Anthrax. Chapter 2.2.1. In: OIE (ed). Terrestrial Animal Health Code. Pp. OIE, Paris.
- Sterne M (1937).** The effect of different carbon dioxide concentrations on the growth of virulent anthrax strains. Onderstepoort Journal of Veterinary Science and Animal Industry, 9, 49-67.
- Turner AJ, Galvin JW, Rubira RJ, Condron RJ, Bradley T (1999a).** Experiences with vaccination and epidemiological investigations on an anthrax outbreak in Australia. Journal of Applied Microbiology, 87(2), 294-7.
- Turner AJ, Galvin JW, Rubira RJ, Miller GT (1999b).** Anthrax explodes in an Australian summer. Journal of Applied Microbiology, 87(2) 196-9.

10. Melioidosis

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

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

10.1.2. OIE list

Not listed.

10.1.3. New Zealand status

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

10.1.4. Epidemiology

Melioidosis is a disease of man and animals that occurs predominantly in the tropical and subtropical regions of Asia and northern Australia and in some foci in Africa (Groves and Harrington 1994; Inglis 2004; Inglis et al 2004). A human case has occurred in New Zealand in a traveller returning from Fiji (Corkill and Cornere 1987). The aetiological agent occurs in the environment and is widely distributed in water and soil (Sprague and Neubauer 2004). It has been transmitted to animals via oral mucosa, nasal mucosa, ingestion, parental inoculation, and skin scarification (Groves and Harrington 1994). Infection in natural cases is 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 (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 and Currie 2005). Transmission from animal to animal has not been described.

10.1.5. Hazard identification conclusion

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

References

References marked * have been sighted as summaries in electronic media.

Cheng AC, Currie BJ (2005). Melioidosis: epidemiology, pathophysiology and management. *Clinical Microbiology Reviews*, 18(2), 383-416.

Corkill MM, Cornere B (1987). Melioidosis: a new disease in New Zealand. *New Zealand Medical Journal*, 100, 106-7.

Groves MG, Harrington KS (1994). Glanders and melioidosis. In: Beran GW (ed). *Handbook of zoonoses. Section A: Bacterial, Rickettsial Chlamydial and Mycotic*. CRC Press, Boca Raton, Ann Arbor, London, Tokyo, 149-65.

Inglis TJJ (2004). Melioidosis in man and other animals: epidemiology, ecology and pathogenesis. *Veterinary Bulletin*, 74(10), 39N-48N.

Inglis TJJ, Foster NF, Gal D, Powell K, Mayo M, Norton R, Currie BJ (2004). Preliminary report on the northern Australian melioidosis environmental surveillance project. *Epidemiology and Infection*, 132(5), 813-20.

Sprague LD, Neubauer H ((2004). Melioidosis in animals: A review on epizootiology, diagnosis and clinical presentation. *Journal of Veterinary Medicine B, Infectious Diseases and Public Health*, 51(7), 305-20.

Thomas AD (1981). Prevalence of melioidosis in northern Queensland. *Australian Veterinary Journal*, 57(3), 146-8.

11. Infections with Mycoplasmas and related Mollicutes

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological Agent

Class: Mollicutes; Order: *Mycoplasmatales*; Family: *Mycoplasmataceae*; Genera: *Mycoplasma*, *Ureaplasma*, and *Acholeplasma*. A description of the relevant *Mycoplasma* organisms and diseases is given in Section 8.1.4.

11.1.2. OIE List

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

11.1.3. New Zealand Status

Mycoplasma capricolum subsp. *caripneumoniae*, *Mycoplasma agalactiae* and *Mycoplasma mycoides mycoides* SC (small colony) are exotic and notifiable (Ministry of Agriculture and Forestry 2007).

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

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

11.1.4. Epidemiology

Mycoplasma capricolum subsp. *caripneumoniae* and *Mycoplasma mycoides* subsp. *mycoides* SC do not occur in Australia.

There are many species of *Mycoplasmas* and closely related organisms belonging to the class Mollicutes and the family Mycoplasmataceae which contains the genera *Mycoplasma*, *Ureaplasma*, and *Acholeplasma*. Organisms that are not considered in this risk analysis include the following organisms that occur in New Zealand: *Mycoplasma mycoides mycoides* LC (Jackson and King 2002), *Mycoplasma arginini* (Belton 1990; Belton 1996), *Mycoplasma canadense* (Mackereth 2007), *Mycoplasma bovis* (Mackereth 2007), *Mycoplasma conjunctivae* (Motha et al 2003) and *Mycoplasma mycoides mycoides* SC that does not occur in Australia (OIE 2007).

Acholeplasma spp. are of no known veterinary significance (Anonymous 2004), and no evidence could be found that they are significant human pathogens, therefore these organisms are also excluded from the risk analysis.

The *Ureaplasma* spp. include a few species that may be significant pathogens but their role as pathogens is not yet well defined and understood. They are included in the risk analysis

Mycoplasma spp. consist of a diverse group of organisms that cause two clearly defined diseases of sheep and goats (contagious caprine pleuropneumonia and contagious

agalactia) and a number of less well defined syndromes. Many of the organisms are not easily fitted into well defined species, they may appear similar when grown on culture medium in the laboratory, and in some cases have antigens that cross react with other species in the genus. This has led to the creation of several sub-species and periodic reorganizations of the taxonomy of organisms in the group. Some organisms have been associated with disease syndromes that are similar to defined diseases and difficult to distinguish from them. It is not clear whether some organisms are primary pathogens, commensals or opportunistic pathogens.

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

- Mycoplasma mycoides* subsp. *mycoides* SC (exotic to Australia)
- Mycoplasma mycoides* subsp. *mycoides* LC (present in New Zealand)
- Mycoplasma mycoides* subsp. *capri*
- Mycoplasma capricolum* subsp. *capripneumoniae*
- Mycoplasma capricolum* subsp. *capricolum*
- Mycoplasma* sp. Group 7 (this group is associated with pathology in cattle).

Other pathogens or potential pathogens of sheep and goats include:

- Mycoplasma agalactiae*
- Mycoplasma putrefaciens*

The organisms of concern in this risk analysis are summarized in Table 2.

Table 2. Diseases/syndromes of sheep and goats caused by *Mycoplasma* spp.

Organism	sheep	goats	cattle	signs
<i>Mycoides capricolum</i> subsp. <i>capripneumoniae</i>	no	yes	no	pleuropneumonia, respiratory symptoms acute fever high mortality (Rurangirwa and Kinyili 2004)
<i>M. capricolum</i> subsp. <i>capricolum</i>	yes	yes	no	mastitis, arthritis, keratoconjunctivitis, pneumonia (Nicholas 2004)
<i>M. mycoides</i> subsp. <i>capri</i>	?	yes	no	pneumonia, arthritis, mastitis (Rurangirwa and Kinyili 2004)
<i>M. agalactiae</i>	yes	yes	no	mastitis, arthritis, keratoconjunctivitis (Nicholas 2004)
<i>M. putrefaciens</i>	rare	yes	no	mastitis, arthritis, keratoconjunctivitis (Nicholas 2004)

Mycoplasma mycoides subsp. *capri* and *Mycoplasma capricolum* subsp. *capricolum*. are associated with disease syndromes that are similar to the OIE listed disease contagious caprine pleuropneumonia. However, the diseases caused by these organisms are not as severe or as infectious as the disease caused by *Mycoplasma capricolum* subsp. *capripneumoniae*.

Contagious agalactia is mainly caused by *Mycoplasma agalactiae*. It occurs in Europe, western Asia, the United States of America, and North Africa (Nicholas 2004). It is a disease of both sheep and goats. Typically the disease causes mastitis, arthritis and keratoconjunctivitis and sometimes abortion (Bergonier et al 1997; Ruffin 2001). All of these signs are likely to be seen in the same flock but may not necessarily be seen in the same animal. Occasional cases of septicaemia also occur (Ruffin 2001). In typical cases there is high morbidity and a mortality rate of up to 25% (Ruffin 2001), but in some flocks subclinical carriers of the organism are known to occur. It is highly contagious and spreads rapidly through a naïve flock by the intranasal and intramammary routes and possibly through wound infection (Ruffin 2001). Following recovery from the acute disease the organism is excreted in the milk for up to a year (Bergonier et al 1997) or even up to 8 years (Madanat et al 2001). It can be diagnosed by isolation of the organism from milk (Nicholas 2004), demonstration of the organism in milk by PCR (Madanat et al 2001; Nicholas 2004; Tola et al 1997) or on a flock basis by serological tests including complement fixation and ELISA (Madanat et al 2001; Nicholas 2004; Tola et al 1997). Immunoblotting has also been used (Nicholas 2004). Similar syndromes are caused by *Mycoplasma mycoides* subsp. *mycoides* LC, *Mycoplasma capricolum* subsp. *capricolum* and *Mycoplasma putrefaciens* and it has been proposed that these species could also be considered to be causal agents of contagious agalactia (Nicholas 2004). Some flocks carry the *Mycoplasma agalactia* without showing signs of mastitis.

Mycoplasma agalactiae has been isolated in Australia but contagious agalactia has not been seen. It is therefore claimed that the Australian strains do not cause the disease (OIE 2007).

Other syndromes and *Mycoplasma* spp. that are found in sheep and goats include the following:

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

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

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

Mycoplasma bovis commonly infects cattle and causes pneumonia in calves and mastitis in dairy cattle. The organism is excreted in milk, nasal, and vaginal secretions and can infect calves and adult cattle by the respiratory route or by the teat canal during milking. It is thought that it may be transmitted on clothing (Tenk 2005). Infected cows can excrete the organism in their milk before showing clinical signs of infection and for many months after clinical recovery (Pinnow et al 2004). *Mycoplasma bovis* has been isolated from mastitic sheep (Ayling et al 2004; Egwu et al 2001) and experimental infection of the udder of sheep has been described (Bocklisch et al 1991). However, it is not a recognized pathogen of sheep, but sheep may act as a reservoir of the organisms for cattle (Pfutzner and Sachse 1996; Tenk 2005). Transmission occurs by respiratory or oral routes. *Mycoplasma bovis* is widely considered to be the most pathogenic of the *Mycoplasma* spp. associated with disease in cattle. It occurs commonly in Australian dairy cattle (Ghadersohi 2003; Ghadersohi et al 2005; Ghadersohi et al 1999). However the strains that occur in Australia have not been associated with gross disease in cattle, only with increased somatic cell counts in milk. In Australia the organism has been identified by PCR but reports of isolation of organisms were not found. Therefore the virulence of the Australian strains has not been determined in experimentally infected animals. *Mycoplasma bovis* has not been found in New Zealand and antibodies to *Mycoplasma bovis* were not detected in 353 sera from dairy cattle (Reichel et al 1999). In a recent survey done on bulk milk samples from dairy herds *Mycoplasma bovis* was not identified by PCR or culture in approximately 240 samples (McDonald 2007).

Organisms that have been isolated from sheep in Australia include *Mycoplasma putrefaciens*, *Mycoplasma agalactiae*, and *Mycoplasma capricolum* (presumably *Mycoplasma capricolum* subsp. *capricolum*) (Cottew and Yeats 1982). The position regarding *Mycoplasma mycoides* subsp. *capri* and *Ureaplasma* spp. is not known but reports of their isolation were not found.

Mycoplasma spp. are sensitive to several antibiotics. Several recent investigations indicate that all strains tested have been sensitive to the fluoroquinolone antibiotics such as enrofloxacin (Godinho et al 2005; Rosenbusch et al 2005; Stipkovits et al 2005; Thomas et al 2003). Other effective antibiotics include tulathromycin (Godinho et al 2005) and valnemulin which has been effective in removing *Mycoplasma bovis* from the lungs (Stipkovits et al 2005). Resistance to some of the older antibiotics such as tetracyclines, lincomycin and spectinomycin has developed and is now becoming evident (Ayling et al 2000; Nicholas and Ayling 2003; Thomas et al 2003).

11.1.5. Hazard Identification Conclusion

Diseases caused by *Mycoplasma* spp. are economically important and it is possible that several *Mycoplasma* spp. can be carried by sheep. For the purposes of this analysis the following species are considered to be potential hazards in the commodity:

Mycoplasma mycoides subsp. *capri*
Mycoplasma capricolum subsp. *capricolum*
Mycoplasma agalactiae (contagious agalactia)
Mycoplasma putrefaciens

Mycoplasma bovis
Ureaplasma spp.

11.2. RISK ASSESSMENT

11.2.1. Entry Assessment

Mollicutes of the following species are known to be present in Australia:

Mycoplasma Mycoides subsp. *capri*
Mycoplasma capricolum subsp. *capricolum*
Mycoplasma agalactiae (contagious agalactia)
Mycoplasma putrefaciens
Mycoplasma bovis
Ureaplasma spp.

With the exception of *Mycoplasma bovis*, which is associated with high somatic cell counts in milk, reports of disease caused by these organisms in Australia were not found and they are best considered as opportunistic pathogens. Antibodies to *Mycoplasma bovis* are common in Australian cattle (Ghadersohi 2003; Ghadersohi et al 2005; Ghadersohi et al 1999). *Mycoplasma bovis* has been described in sheep. Therefore the likelihood of importing *Mycoplasma bovis* in sheep is considered to be non-negligible. Since all the organisms could be found in subclinically infected sheep the likelihood of introduction of these Mollicutes in imported sheep is considered to be non-negligible.

11.2.2. Exposure Assessment

Since imported sheep and goats will be integrated into New Zealand flocks the transmission of introduced mollicutes to sheep is likely to occur. Introduction of *Mycoplasma bovis* in sheep could result in exposure of cattle to the organism. Therefore the likelihood of exposure is considered to be non-negligible.

11.2.3. Consequence Assessment

Australian strains of *Mycoplasma agalactiae* are not known to cause contagious agalactiae in Australia. Other Mollicutes considered in this section with the exception of *Mycoplasma bovis* and *Mycoplasma agalactiae* may be opportunistic pathogens or secondary invaders and the consequences of introducing Australian strains of these organisms is uncertain, but likely to be low. However, *Mycoplasma agalactiae* causes an OIE listed disease and the introduction of *Mycoplasma bovis* and the subsequent infection of cattle could result in the introduction of a production limiting disease of cattle. *Mycoplasma bovis* has been described as a major cause of respiratory disease, mastitis, and arthritis, responsible for a quarter to a third of the cases of calf pneumonia in Europe (Nicholas and Ayling 2003). The virulence of the Australian strains of *Mycoplasma agalactiae* and *Mycoplasma bovis* has not been determined but is claimed to be low on the basis that the typical diseases associated with the organisms have not been described.

Since the organisms are not zoonotic there would be no consequences for human health. Feral goats and thar may be susceptible to infection with the organisms although other wild or feral animals have not been described as affected.

Since the importation of infected sheep or goats could lead to the establishment of new *Mycoplasma* spp. in New Zealand and the introduction of a serious pathogen of cattle, the consequences of introduction are considered to be non-negligible.

11.2.4. Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible, and exotic *Mycoplasma* spp. are classified as hazards in the commodity. Therefore, risk management measures can be justified.

11.3. RISK MANAGEMENT

11.3.1. Options

Mycoplasma bovis and *Mycoplasma agalactiae* are important pathogens, whilst other Mollicutes are considered to be opportunistic pathogens or secondary invaders. No suitable diagnostic tests are available for detection of all the Mollicutes. Serological tests are available for *Mycoplasma bovis* and *Mycoplasma agalactiae* but the specificity and sensitivity of the tests is not well defined. There are no prescribed or alternative tests for *Mycoplasma agalactiae* given in the Manual of Diagnostic tests and vaccines (OIE 2004). *Mycoplasma* spp. are sensitive to several antibiotics which are effective in treatment of clinical cases. Resistance is known to have developed to some antibiotics but several highly effective antibiotics are still available (Section 8.1.4.1).

The *Code* chapter on contagious agalactia contains the following recommendations (Article 2.4.3.1.) relating to the importation of animals:

Veterinary Authorities of importing countries should require (for sheep and goats), the presentation of an international veterinary certificate attesting that the animals

1. showed no clinical sign of contagious agalactia on the day of shipment;
2. were kept since birth or for the 6 months prior to shipment in an establishment where no case of contagious agalactia was officially reported during that period;
3. were kept in a quarantine station for the 21 days prior to shipment.

There are no code chapters relating to other relevant Mollicutes.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with *Mycoplasma bovis* and *Mycoplasma agalactiae* in the commodity:

- Sheep and goats for importation could be required to originate from farms on which there has been no evidence of respiratory disease, mastitis, or arthritis caused by *Mycoplasma* spp. during the previous 3 years.
- Sheep and goats could be placed in quarantine for at least 3 weeks and tested by serological tests for *Mycoplasma bovis* and *Mycoplasma agalactiae* with a requirement for negative results. The test could be carried out within the 14 days prior to shipment.

- Animals could be treated with antibiotics recognised as effective against *Mycoplasma* spp. in the exporting country and approved by MAF BNZ, while in quarantine.

References

References marked * have been sighted as summaries in electronic media.

Anonymous (2002). Review of submissions on the disease risk assessment: The use in New Zealand of imported semen derived from an Argali (*Ovis ammon polii*) sheep. Appendix 1, Ministry of Agriculture and Forestry, Wellington.

Anonymous (2004). Mycoplasmas. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Pp. 2043-44. Oxford University Press, Cape Town.

Ayling RD, Baker SE, Peek ML, Simon AJ, Nicolas RAJ (2000). Comparison of the in vitro activity of danofloxacin, florfenicol, oxytetracycline, spectinomycin, and tilmicosin against recent field isolates of *Mycoplasma bovis*. *Veterinary Record*, 146(26), 745-7.

Ayling RD, Bashiruddin SE, Nicholas RA (2004). *Mycoplasma* species and related organisms isolated from ruminants in Britain between 1990 and 2000. *Veterinary Record*, 155, 413-6.

Ball HJ, McCaughey WJ (1987). An examination of the effects of *Ureaplasma* infection on the fertility of synchronised ewes. *Animal Reproduction Science*, 13(1), 61-6.

Ball HJ, McCaughey WJ, Kennedy S (1986). Experimental intrauterine inoculation of ewes in early pregnancy with ureaplasma. *Irish Veterinary Journal*, 40(11/12), 187-8.

Ball HJ, McCaughey WJ, Kennedy S, McLoughlin M (1985). Experimental intrauterine inoculation of pregnant ewes with ureaplasmas. *Veterinary Research Communications*, 9(1), 35-43.

Belton D (1990). Mycoplasmas of sheep and goats in New Zealand. *Surveillance*, 17(2), 18-9.

Belton D (1996). Abattoir surveillance of mycoplasmas in the lungs and udders of New Zealand goats. *Surveillance*, 23(1), 21.

Bergonier D, Berthelot X, Poumarat F (1997). Contagious agalactia of small ruminants: current knowledge concerning epidemiology diagnosis and control. *Revue Scientifique et Technique, OIE*, 16(3), 848-73.

Bocklisch H, Kreusel S, Brys A, Pftzner H (1991). Experimental infection of the udder of ewes due to *Mycoplasma bovis*. *Zentralblatt für Veterinärmedizin B*, 38(5), 385-90.*

Cottew GS, Yeats FR (1982). Mycoplasmas and mites in the ears of clinically normal goats. *Australian Veterinary Journal*, 59, 77-81.

Egwu GO, Ameh JA, Aliyu MM, Mohammed FD (2001). Caprine mycoplasmal mastitis in Nigeria. *Small Ruminant Research*, 39(1), 87-91.*

Ghadersohi A (2003). Development of a PCR for specific detection of *Mycoplasma bovis* from bovine milk and mucosal samples: a critique. *Veterinary Microbiology*, 97(1-2), 167-8; author reply 9-71.

Ghadersohi A, Fayazi Z, Hirst RG (2005). Development of a monoclonal blocking ELISA for the detection of antibody to *Mycoplasma bovis* in dairy cattle and comparison to detection by PCR. *Veterinary Immunology and Immunopathology*, 104(3-4), 183-93.*

Ghadersohi A, Hirst RG, Forbes-Faulkener J, Coelen RJ (1999). Preliminary studies on the prevalence of *Mycoplasma bovis* mastitis in dairy cattle in Australia. *Veterinary Microbiology*, 65(3), 185-94.

Godinho KS, Rae A, Windsor GD, Tilt N, Rowan TG, Sunderland SJ (2005). Efficacy of tulathromycin in the treatment of bovine respiratory disease associated with induced *Mycoplasma bovis* infections in young dairy calves. *Veterinary Therapeutics*, 6(2), 96-112.*

Howard CJ (1984). Animal ureaplasmas: their ecological niche and role in disease. *Israel Journal of Medical Science*, 20(10), 954-7.

Howard CJ, Pocock DH (1983). Comparison of ureaplasmas from sheep and goats with *Ureaplasma diversum* and *U. urealyticum*. *Journal of General Microbiology*, 129(10), 3197-202.

Jackson R, King C (2002). *Mycoplasma mycoides* subsp. *mycoides* (Large Colony) infection in goats. A review with special reference to the occurrence in New Zealand. *Surveillance*, 29(3), 8-12.

Koshimizu K, Kotani H, Yamamoto K, Magaribuchi T, Harasawa R, Ito M, Ogata M (1984). Serological analysis of ureaplasmas isolated from various animals. *Israel Journal of Medical Science*, 20(10), 950-3.*

Livingstone CW, Gauer BB (1982). Effect of venereal transmission of ovine ureaplasma on reproductive efficiency of ewes. *American Journal of Veterinary Research*, 43(7), 1190-3.

Mackereth G (2007). Personal communication.

Madanat A, Zendulkova D, Pospisil Z (2001). Contagious agalactia of sheep and goats, a review. *Acta veterinaria. Brno*, 70,, 403-12.

McCaughey WJ, Ball HJ (1985). The physical appearance of the vulva in ureaplasma infected ewes. *Veterinary Research Communications*, 9(2), 123-5.

McDonald W (2007). Personal communication.

Ministry of Agriculture and Forestry (2007). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 9/1/2008.

Motha M, Frey J, Hansen MF, Jamaludin R, Tham KM (2003). Detection of *Mycoplasma conjunctivae* in sheep affected with conjunctivitis and infectious keratoconjunctivitis. *New Zealand Veterinary Journal*, 51(4), 186-90.

Nicholas RA (2004). Contagious agalactia. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Pp. 606-14. OIE, Paris.

Nicholas RA, Ayling RD (2003). *Mycoplasma bovis*: disease, diagnosis, and control. *Research in Veterinary Science*, 74(2), 105-12.

Nicolet J (1994). *Mycoplasma* infections in cattle, sheep, and goats: methods for diagnosis and prophylaxis. In: OIE (Editor), *Comprehensive reports on technical items presented to the International Committee or to Regional Commissions*. OIE, Paris, pp. 43-54.

OIE (2004). *Manual of Diagnostic Tests and Vaccines*. OIE, Paris, pp. xii.

OIE (2007). Handistatus II. <http://www.oie.int/hs2/report.asp>, downloaded 9/1/2008.

Pfutzner H, Sachse K (1996). *Mycoplasma bovis* as an agent of mastitis, pneumonia, arthritis and genital disorders in cattle. *Revue Scientifique et Technique OIE*, 15(4), 1477-94.

Pinnow C, Butler J, Rosenbusch R, Timms L (2004). Development of a nested PCR method for detection of *Mycoplasma bovis* in preserved milk samples. *Iowa University Animal Industry Report*, [http://www.ans.iastate.edu/report/air/\(2004\).pdf/AS1917.pdf](http://www.ans.iastate.edu/report/air/(2004).pdf/AS1917.pdf), downloaded 9/1/2008.

Reichel M, Nicolas RAJ, Ross GP, Penrose ME (1999). Survey results for exotic *Mycoplasma* infections in cattle, goats and sheep. *Surveillance*, 26(3), 12-3.

Rosenbusch RF, Kinyon JM, Apley M, Funk ND, Smith S, Hoffman LJ (2005). In vitro antimicrobial inhibition profiles of *Mycoplasma bovis* isolates recovered from various regions of the United States from 2002 to 2003 *Journal of Veterinary Diagnostic Investigation*, 17(5), 436-41.*

Ruffin DC (2001). *Mycoplasma* infections in small ruminants. *Veterinary clinics of North America: food animal practice*, 17(2), 315-32.*

Rurangirwa FR, Kinyili JH (2004). Contagious caprine pleuropneumonia. In: OIE (ed). *Manual of Diagnostic Tests and Vaccine for Terrestrial Animals*. Pp. 623-34. OIE, Paris.

Stipkovits L, Ripley PH, Tenk M, Glavits R, Molnar T, Fodor L (2005). The efficacy of valnemulin (Econor) in the control of disease caused by experimental infection of calves with *Mycoplasma bovis*. *Research in Veterinary Science*, 78(3), 207-15.*

Tenk M (2005). Examination of *Mycoplasma bovis* infection in cattle. Doctoral thesis, <http://phd.univet.hu/lapok/ertekezes/Tenk-D-E.pdf>, downloaded 9/1/2008.

Thomas A, Nicolas C, Dizier I, Mainil J, Linden A (2003). Antibiotic susceptibilities of recent isolates of *Mycoplasma bovis* in Belgium. *Veterinary Record*, 153(14), 428-31.

Tola S, Angioi A, Rocchigiani AM, Idini G, Manunta D, Galleri G, Leori G (1997). Detection of *Mycoplasma agalactiae* in sheep milk samples by polymerase chain reaction. *Veterinary Microbiology*, 54(1), 17-22.

12. Salmonellosis

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological Agent

There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2004). Most of these belong to the species *enterica* and the subspecies *enterica* and using correct conventions the names such as *dublin* and *abortus ovis*, which do not have species status, should not be italicised. However, in this review for the sake of simplicity and convenience they are italicised as though they were species.

This analysis is concerned mainly with three important serovars: *Salmonella dublin*, *Salmonella typhimurium* and *Salmonella abortus ovis* but also refers to other serovars. Phage typing of *Salmonella* is also commonly used to classify strains. In the case of *Salmonella typhimurium*, only the definitive phage type (DT) 104, which occurs very rarely in New Zealand, is considered in this analysis. *Salmonella typhimurium* DT104 is of particular significance because it exhibits multiple resistance to common antibiotics and is considered a threat to human health (Hogue et al 1997; Jones et al 2002). It is now widely distributed in the world.

12.1.2. OIE List

Salmonella abortus ovis is not a listed disease in the OIE Terrestrial Animal Code (OIE 2007b) but is listed in the index of the OIE manual of diagnostic tests and vaccines. Salmonellosis is covered in the OIE Manual of Diagnostic Tests and Vaccines under “Diseases not covered by List A and List B” (Davies 2004).

12.1.3. New Zealand Status

Salmonella abortus ovis is exotic and notifiable (Ministry of Agriculture and Forestry 2007). *Salmonella dublin* is exotic and notifiable (Ministry of Agriculture and Forestry 2007) *Salmonella typhimurium* is endemic in New Zealand but phage type 104 has only occurred rarely in humans and not in animals. It is classified in the category of “other unwanted organisms” (Ministry of Agriculture and Forestry 2007).

12.1.4. Epidemiology

Salmonella spp. isolated in New Zealand are identified to serovar and phage type by the Environmental Science and Research (ESR) laboratory and recorded on a database (ESR 2005). Isolations from both medical and animal health laboratories are recorded.

Salmonella dublin and *Salmonella abortus ovis* have not been isolated in New Zealand.

Salmonella typhimurium is endemic in New Zealand in both animals and man but the definitive phage type DT 104 has only been isolated very rarely from humans and from three dogs in a household in which the owners suffered from diarrhoea after returning from an overseas visit (Julian 2002). The sporadic occurrence of *Salmonella typhimurium* type DT 104 in a few cases in humans and once in dogs does not indicate that it has become established. There is no indication that the New Zealand animal population has become

infected. *Salmonella typhimurium* DT104 has been isolated in Australia in animals and humans (Brockman 2001; Mackie et al 1996).

Salmonella abortus ovis has not been reported from Australia (OIE 2007). *Salmonella dublin* is endemic in Australia (Trueman et al 1996).

Infection is mainly by the oral route and factors such as infecting dose, the particular strain and species of *Salmonella* involved and various stress factors play a role in determining the outcome of the infection (Fenwick and Collett 2004; Naser et al 2004). The incubation period is from 1-7 days in experimental infections and 6-30 days after natural exposure (Naser et al 2004). The intestine is initially infected and an inflammation of the gut is the primary lesion. Initial infection may be followed by penetration of the gut and mesenteric lymph node barrier followed by bacteraemia. Within about a week, most infected lambs develop a multifocal necrotic hepatitis and nephrosis (Naser et al 2004). Animals that survive may recover fully after 3-4 weeks (Naser et al 2004). In the case of infected pregnant animals, abortion is common (Naser et al 2004). Serious illness and mortality following abortions caused by *Salmonella dublin* and *Salmonella typhimurium* is common (Radostits et al 2007). Animals that recover frequently become carriers for up to a year and sometimes for life. Three types of carrier have been described (Radostits et al 2007):

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

Excreted organisms contaminate the environment and become a source of infection (Radostits et al 2007). Young animals are more often affected by the disease than adults and very young animals may die after a short period of bacteraemia. Serious disease and mortality also occurs in some adults particularly following abortion (Radostits et al 2007). Ewes that abort excrete large numbers of organisms in their uterine discharges.

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

In the MAF BNZ risk analysis for the importation of small ruminant germplasm², it was concluded that the risk of introducing *Salmonella* spp. in germplasm was non-negligible. However, since *Salmonella* can be readily cultured it is possible to test germplasm before it is imported.

² See: www.biosecurity.govt.nz/files/pests-diseases/animals/risk/risk-analysis-sheep-goat-genetic-material.pdf

12.1.5. Hazard Identification Conclusion

Salmonella dublin is an exotic, notifiable, zoonotic organism and *Salmonella typhimurium* type DT104 is an unwanted and zoonotic organism that has not been isolated from production animals. Therefore these organisms are classified as potential hazards in the commodity. Other exotic *Salmonella* spp. should also be considered to be potential hazards.

12.2. RISK ASSESSMENT

12.2.1. Entry Assessment

Since *Salmonella dublin* and an unknown number of other *Salmonella* spp. are endemic in Australia and exotic to New Zealand, the likelihood that subclinical carriers of exotic *Salmonella* spp. could be imported is considered to be non-negligible.

12.2.2. Exposure Assessment

Imported sheep and goats will be integrated into New Zealand flocks and under suitable circumstances carriers could excrete the organism in their faeces and infect animals in contact with them. Therefore, the exposure assessment is considered to be non-negligible.

12.2.3. Consequence Assessment

The introduction and establishment of any of the species covered in this section could result in gradual spread of the organisms in New Zealand and the establishment of production limiting diseases of livestock and human disease. The emergence of a new serovar, *Salmonella brandenburg*, demonstrated how a new *Salmonella* serovar was able to spread through the South Island sheep population (Kerslake and Perkins 2006).

Because of its resistance to antibiotics, establishment of *Salmonella typhimurium* DT 104 in animal populations would constitute a source of infection for humans and therefore be of particular concern to human health (Davies 2001; Hogue et al 1997). *Salmonella dublin* is also zoonotic and could cause disease in humans.

The consequences for the environment would be limited to sporadic cases of salmonellosis in wild or feral animals and birds. An outbreak of a new phage type of *Salmonella typhimurium* (DT160) occurred in sparrows and in humans in 2001. The outbreak was associated with the death of several hundred sparrows (Alley et al 2002). While that outbreak was self limiting and did not cause lasting damage to the sparrow population, *Salmonella* infections can establish in wild bird populations and possibly cause mortalities over many years (Pennycott 2001). *Salmonella typhimurium* DT 160 and DT195 have been isolated and cause clinical signs in silvereye, kaka, kakariki and hihi (Alley 2007). However, the effects that introducing new *Salmonella* spp. might have on native birds is not known.

Introduction of infected sheep and goats could lead to the establishment of new *Salmonella* spp. that have the potential to cause human disease and production limiting disease of animals and infections in wild birds. Therefore the consequences are considered to be non-negligible.

12.2.4. Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and exotic *Salmonella* spp. are classified as hazards in the commodity. Therefore, risk management measures can be justified.

12.3. RISK MANAGEMENT

12.3.1. Options

Animals may be long term, subclinical carriers of *Salmonella* spp. and therefore quarantine of animals is not considered to be useful. Salmonellae can be cultured from faeces and tissues. Carrier animals may shed *Salmonella* spp. in their faeces over long periods of time, but shedding may be intermittent. There is no information about how many times animals should be tested to reach a high level of certainty that they are not infected.

Because many strains of *Salmonella* spp. have developed antibiotic resistance treatment of animals with antibiotics is not considered to be a reliable method of eliminating the organism. Vaccines are not available for a wide spectrum of *Salmonella* spp. Serological tests are not available for many individual *Salmonella* spp.

Although detailed and accurate information about the freedom of properties from salmonellosis may not be available, a statement from a veterinarian or a farm owner that a property has been free from infection for a long period could be considered useful information for determining the likelihood that an animal may carry the organism.

Germplasm could be tested for freedom from *Salmonella* spp. before importation. Therefore importation of tested germplasm could be considered to represent an alternative method of importing genetic material.

The *Code* does not give any guidance about the risk management options relating to *Salmonella* spp. when importing animals.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with exotic *Salmonella* spp. in the commodity:

- Sheep and goats to be imported could be required to be healthy, and in particular be free from clinical signs of enteric infections.
- Animals could be required to originate from farms where outbreaks of salmonellosis due to *Salmonella* *Dublin* or *Salmonella* *typhimurium* DT104 are not known to have occurred in the last 3 years.
- Animals could be held for at least 3 weeks in a quarantine station in which cases of salmonellosis have not occurred for the previous 3 months.
- Whilst in quarantine, faeces samples could be cultured on at least 2 occasions with an interval of 10 days using suitable pre-enrichment and enrichment media (Davies 2004). All *Salmonella* spp. isolated could be serotyped (and, where appropriate phage typed) and the results reported to

MAFBNZ. If pathogenic *Salmonella* spp., exotic to New Zealand are isolated, importation of the animals could be prohibited. Where *Salmonella* spp. that are endemic to New Zealand are isolated, the importer of the animals could make a decision as to whether to proceed with the importation.

- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm that has been tested and shown to be free from exotic *Salmonella* spp.
- If an option involving testing of faeces is used, importers could be encouraged to test animals at their own expense before they enter quarantine. In this way the expense of quarantining infected animals could be avoided.

References

References marked * have been sighted as summaries in electronic media.

Alley MR (2007). Email to H. J. Pharo.

Alley MR, Connolly JH, Fenwick SG, Mackereth GF, Leyland MJ, Rogers LE, Haycock M, Nicol C, Reed CEM (2002). An epidemic of salmonellosis caused by *Salmonella* Typhimurium DT 160 in wild birds and humans in New Zealand. *New Zealand Veterinary Journal*, 50(5), 170-6.

Brockman S (2001). International outbreak of *Salmonella* Typhimurium DT 104 due to contaminated sesame seed products - update from Germany (Baden Wurtemberg). *Eurosurveillance*, 5(13), <http://www.eurosurveillance.org/ew/2001/010816.asp>, downloaded 8/1/2008.

Davies R (2001). *Salmonella* Typhimurium DT 104 in Great Britain. *Udgivet af Dansk Zoonoscenter*, <http://zoonyt.dzc.dk/0101/artikler/art5.htm>, downloaded 8/1/2008.

Davies R (2004). Salmonellosis. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Pp. 1018-32. OIE, Paris.

ESR (2005). Database of the enteric reference laboratory. http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php, downloaded 8/1/2008.

Fenwick SG, Collett MG (2004). Bovine salmonellosis. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Pp. 1582-93. Oxford University Press, Cape Town.

Hogue A, Agula F, Johnson R, Petersen K, Saini P, Schlosser W (1997). Situation Assessment: *Salmonella* Typhimurium DT 104, United States Department of Agriculture, Food Safety and Inspection Service, Washington DC 20250. <http://www.fsis.usda.gov/OPHS/stdt104.htm>, downloaded 8/1/2008.

Jones YE, Chappell S, McLaren IM, Davies RH, Wray C (2002). Antimicrobial resistance in *Salmonella* isolated from animals and their environment in England and Wales from 1988 to 1999. *Veterinary Record*, 150, 649-54.

Julian A (2002). Quarterly review of diagnostic cases: Gribbles Veterinary Pathology: Dogs. *Surveillance*, 29(3), 28.

Kerslake JI, Perkins NR (2006). *Salmonella* Brandenburg: case-control survey in sheep in New Zealand. *New Zealand Veterinary Journal*, 54(3), 125-31.

Mackie JL, Lightfoot D, Adamson M, Wishart M (1996). Antibiotic resistant phage types of Salmonella Typhimurium in dairy cattle. Australian Veterinary Journal, 73(3) 194-5.

Ministry of Agriculture and Forestry (2006). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.

Neser JA, Fenton MM, Fenwick SG (2004). Ovine and caprine salmonellosis. In: Coetzer JAW, Tustin RC (eds). Infectious Diseases of Livestock. Pp. 1594-600. Oxford University Press, Cape Town.

Nielsen LR, Ersboll AK (2004). Age-stratified validation of an indirect Salmonella Dublin serum enzyme-linked immunosorbent assay for individual diagnosis in cattle. Journal of Veterinary Diagnostic Investigation, 16(3), 212-8.*

Nielsen LR, Toft N, Ersboll AK (2004). Evaluation of an indirect serum ELISA and a bacteriological faecal culture test for diagnosis of Salmonella serotype Dublin in cattle using latent class models. Journal of Applied Microbiology, 96(2), 311-9.

OIE (2007a). Handistatus II. <http://www.oie.int/hs2/report.asp>, downloaded 8/1/2008.

OIE (2007b). Terrestrial Animal Health Code. http://www.oie.int/eng/normes/MCode/en_sommaire.htm, downloaded 8/1/2008.

Pennycott T (2001). Death in finches and sparrows. <http://www.bvpa.org.uk/papers/penn01wb.htm>, downloaded 8/1/2008.

Radostits O, Gay C, C., Hinchcliff KW, Constable PD (2007). Diseases associated with Salmonella species. Veterinary Medicine. A Textbook of the Diseases of Cattle, Horses, Sheep, Pigs, and Goats. Pp. 896-921. Saunders Elsevier, Edinburgh, London, New York, Oxford, Philadelphia, St Louis, Sydney, Toronto.

Trueman KF, Thomas RJ, Mackenzie AR, Eaves LE, Duffy PF (1996). Salmonella Dublin infection in Queensland dairy cattle. Australian Veterinary Journal, 74(5), 367-9.

13. Leptospirosis

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

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

13.1.2. OIE List

The *Code* lists leptospirosis as a disease of multiple species, but since 2003 has not had a chapter on Leptospirosis. The chapter is listed as “under study”. This situation may have arisen because of the ubiquity of the organism the absence of meaningful control programmes and because effective treatments are available.

13.1.3. New Zealand Status

Leptospira hardjo, *Leptospira pomona*, *Leptospira balcanica*, *Leptospira copenhageni*, *Leptospira ballum* and *Leptospira tarrasovi* have been isolated from animals in New Zealand (Midwinter 1999). A single isolation of *Leptospira australis* has been reported from a human (Thompson 1980). *Leptospira canicola* was isolated from a human (Chereshsky et al 1993). However an extensive survey of dogs did not reveal any evidence of infection (Hilbink et al 1992). In humans, serological diagnosis indicates that five of the species endemic in farm animals infect humans but *Leptospira balcanica* which is associated with possums has not been diagnosed in man (Anonymous 2004). Other *Leptospira* spp. are classified by MAF as “other exotic organisms” (Ministry of Agriculture and Forestry 2005).

13.1.4. Epidemiology

Leptospirosis is not a single disease but a complex of diseases caused by at least 200 different organisms. Many *Leptospira* serovars are adapted to a particular host species in which an almost symbiotic relationship has been formed. Species other than the maintenance host may be more resistant to infection but if infected are more susceptible to disease. For example *Leptospira hardjo* infects many sheep in New Zealand but clinical disease has not been reported while the few cases of clinical disease that occur are caused by *Leptospira Pomona* (Orr 1998). However, *Leptospira hardjo* causes sporadic cases of disease in other species including 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. In New Zealand the prevalence of clinical leptospirosis in humans is relatively high for a temperate climate country and *Leptospira hardjo* accounts for nearly half the cases (Thornley et al 2002). Leptospirosis occurs world-wide including Australia. The endemic serotypes that occur in each country differ but world-wide *Leptospira hardjo* is the most common serovar found in cattle and sheep. Leptospirosis of goats is rare but antibody to *Leptospira ballum*, *Leptospira bratislava* (believed to be a cross reaction with *Leptospira hardjo*), and *Leptospira pomona* were found (Thompson 2001).

Leptospire are spread in water and mud contaminated with infected urine. Infection can occur by mouth or through the skin particularly through abrasions and wounds. Clinically infected 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 to 16 days and is followed by a period of bacteraemia. A variety of signs may be shown by diseased animals including abortion, haemolytic anaemia, icterus, and nephritis. The disease can be diagnosed by the isolation of the organism, but because this is a difficult process it is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low level titre indicating resolution or a chronic infection. The microscopic agglutination test is still the most commonly used herd test and a number of variations of ELISAs are also available but ELISAs generally lack serovar specificity (Bolin 2004). Leptospirosis is seldom the cause of economically serious disease in animals. However it is important because it is a zoonotic infection that occasionally causes serious disease in humans (Thornley et al 2002).

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

Vaccination against the main serovars occurring in New Zealand is widely practised. Vaccination is mainly aimed at developing an immune population and thereby reducing the risk to humans that are in contact with the infected animals.

13.1.5. Hazard Identification Conclusion

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

13.2. RISK ASSESSMENT

13.2.1. Entry Assessment

Acutely infected animals or chronic carriers of infection may excrete the organism in urine and in their semen. Therefore the likelihood of entry is considered to be non-negligible.

13.2.2. Exposure Assessment

Carriers shed the organism in their urine and are likely to infect animals that are in contact with them. Venereal transmission of the organism is also possible. Since imported sheep and goats will be introduced into New Zealand flocks the likelihood of exposure of New Zealand animals to the organisms is considered to be non-negligible.

13.2.3. Consequence Assessment

Introduction of new serovars of *Leptospira* are unlikely to have a significant impact on the New Zealand animals. Sporadic cases of disease may occur.

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

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

Since the position in new Zealand has remained stable for many years, the likelihood of establishment of new *Leptospira* serovars is low but non-negligible. Establishment of new serovars could cause sporadic cases of disease in humans. Therefore the consequences of establishment are considered to be non-negligible.

10.2.4 Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and exotic *Leptospira* spp. are classified as hazards in the commodity. Therefore, risk management measures can be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

Leptospirosis is not a common disease of sheep and goats and is not generally economically important in these species. Diagnosis of the disease by culturing the organisms is difficult and seldom undertaken in diagnostic laboratories. Serological diagnosis generally lacks specificity and differentiation of species of infecting organisms is often not possible. There are no widely accepted standards for cut-off points for interpretation of serological test results. Treatment with antibiotics is highly effective and has been used successfully for many years in international trade. Many *Leptospira* spp are zoonotic.

There is no current *Code* chapter for leptospirosis.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with exotic *Leptospira* spp. in the commodity:

- Sheep and goats could be imported without restrictions
- Animals could be quarantined for 4 weeks and tested serologically on entry into quarantine and again after 2 weeks. Those that are serologically negative or clearly identifiable as having antibody that indicates infection or previous infection only with a serovar that occurs in New Zealand, could be imported.

- Sheep and goats to be imported could be treated with suitable antibiotics before shipment.

References

References marked * have been sighted as summaries in electronic media.

Alt DP, Sterner RL, Bolin CA (2001). Evaluation of antibiotics for treatment of cattle infected with *Leptospira borgpetersenii* serovar hardjo. *Journal of the American Veterinary Medical Association*, 219(5), 636-9.

Anonymous (2004). Notifiable and other diseases in New Zealand. Annual Report (2003)., pp. 26-7., Institute of Environment and Science Research, New Zealand.
[http://www.surv.esr.cri.nz/PDF_surveillance/AnnSurvRpt/\(2003\).AnnualSurvRpt.pdf](http://www.surv.esr.cri.nz/PDF_surveillance/AnnSurvRpt/(2003).AnnualSurvRpt.pdf), downloaded 8/1/2008.

Bolin CA (2004). Leptospirosis. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Pp. 316-27. OIE, Paris.

Chereshsky A, Cameron G, Marshall R (1993). A case of human *Leptospira canicola* in New Zealand. *New Zealand Veterinary Journal*, 41, 101.

Gerritsen MJ, Koopmans MJ, Dekker TC, De Jong MC, Moerman A, Olyhoek T (1994). Effective treatment with dihydrostreptomycin of naturally infected cows shedding *Leptospira interrogans* serovar hardjo subtype hardjobovis. *American Journal of Veterinary Research*, 55(3), 339-43.

Gerritsen MJ, Koopmans MJ, Olyhoek T (1993). Effect of streptomycin treatment on the shedding of and the serologic responses to *Leptospira interrogans* serovar hardjo subtype hardjobovis in experimentally infected cows. *Veterinary Microbiology*, 38(1-2), 129-35.

Hilbink F, Penrose MF, McSporran K (1992). Antibodies in dogs against *Leptospira interrogans* serovars copenhageni, ballum and canicola. *New Zealand Veterinary Journal*, 40, 123-5.

Horsch F (1989). Leptospirosis. In: Blaha T (ed). *Applied Veterinary Epidemiology*. Pp. 95-102. Elsevier Science Publishers, Amsterdam.

Midwinter A (1999). Spirochaetes in New Zealand. *Surveillance*, 26(3), 10-2.

Ministry of Agriculture and Forestry (2005). The Unwanted Organisms Register.
<http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.

Murray CK, Hospenthal DR (2004). Determination of susceptibilities of 26 *Leptospira* sp. serovars to 24 antimicrobial agents by a broth microdilution technique. *Antimicrobial Agents and Chemotherapy*, 48(10), 4002-5.*

Oie S, Hironaga K, Koshiro A, Konishi H, Yoshii Z (1983). In vitro susceptibilities of five *Leptospira* strains to 16 antimicrobial agents. *Antimicrobial Agents and Chemotherapy*, 24(6), 905-8.*

Orr M (1998). Infectious diseases of sheep in New Zealand. *Surveillance*, 25(3), 10-2.

Thompson A (1980). The first New Zealand isolation of *Leptospira interrogans* serovar australis. *New Zealand Medical Journal*, 91(651), 28.

Thompson KG (2001). Infectious diseases of goats in New Zealand. *Surveillance*, 28(2), 3-7.

Thornley CN, Baker MG, Weinstein P, Maas EW (2002). Changing epidemiology of human leptospirosis in New Zealand. *Epidemiology and Infection*, 128(1), 29-36.*

14. Q Fever

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological Agent

Coxiella burnetii.

14.1.2. OIE List

Listed.

14.1.3. New Zealand Status

Exotic, notifiable disease (Ministry of Agriculture and Forestry 2007).

14.1.4. Epidemiology

Coxiella burnetii is endemic in Australia (OIE 2007).

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

Transmission may occur from contact with infected uterine discharges and placentas or the inhalation of dust contaminated by animals and their birth products (Behymer and Riemann 1989; Hawker et al 1998; Marin and Raoult 1999; Marrie 1990; Selvaggi et al 1996; Tissot-Dupont et al 1999). Infected ticks may also play a role in spreading the disease. At least 40 species of ticks from 11 genera can be infected (Kelly 2004) and their infected dried faeces forms dust that can contaminate animal coats. *Haemaphysalis longicornis* (previously known as *Haemaphysalis bispinosa*) has been infected with *Coxiella burnetii* but failed to transmit the organism to guinea pigs (Smith 1942). *Haemaphysalis humerosa* did transmit the disease to guinea pigs (Smith 1941). Sheep shed the organism in vaginal secretions for up to 2 months after parturition and may shed organisms at subsequent pregnancies (Kelly 2004). Infection in goats is also reported to probably be limited to two seasons (Hatchette et al 2003).

Infected animals generally show no clinical signs, thus making the determination of the incubation period and the interval to the development of antibodies problematic. Data are available for humans and the incubation period is given as 1-3 weeks. The development of detectable antibody titres takes 2-3 weeks after the onset of symptoms (Marin and Raoult 1999). Extrapolating from this information it is assumed that infected sheep or goats will develop antibody within 6 weeks of infection.

The infection is diagnosed by serological tests or by identification or isolation of the organism (Rousset et al 2004). The ELISA is the test of choice for serological diagnosis (Rousset et al 2004).

There is evidence that *Coxiella burnetii* may be transmitted in semen (Kruszewska et al 1996; Kruszewska and Tylewska-Wierzbanowska 1997; Kruszewska and Tylewska-Wierzbanowska 1993). However, semen or embryos could be tested for the presence of the organism before importation and importation of germplasm would therefore represent an alternative method of importing genetic material.

14.1.5. Hazard Identification Conclusion

Coxiella burnetii is an exotic, notifiable (Ministry of Agriculture and Forestry 2007) and zoonotic organism that occurs in Australia. Therefore, it is classified as a potential hazard in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry Assessment

Since the organism is endemic in Australia and infected sheep and goats can be long-term carriers of the organism, the likelihood of introduction of the organism with sheep and goats from Australia is considered to be non-negligible.

14.2.2. Exposure Assessment

Imported sheep and goats would be integrated into New Zealand flocks. Since infected sheep have been shown to excrete large numbers of organisms in their birth products at parturition (Welsh HH, Lenette EH, Albatini FR, Winn JF, cited by Marrie 1990), the organism could be shed at lambing/kidding and transmitted to New Zealand animals or humans. The exposure assessment is therefore considered to be non-negligible.

14.2.3. Consequence Assessment

Establishment of the infection in New Zealand would be likely to have a negligible effect on the livestock industries as infected animals are usually subclinical. However, there is a small likelihood that the introduction into a naïve population might initially cause some abortions (Arricau-Bouvery and Rodolakis 2005). Some species of *Haemaphysalis* can be infected with *Coxiella burnetii* and *Haemaphysalis humerosa* was shown to transmit the organism to guinea pigs (Heath 2002; Smith 1941; Smith 1942). Therefore the ability of the New Zealand cattle tick to transmit the disease remains uncertain.

Establishment of the disease would result in sporadic cases of serious disease in people. Virtually all animals including birds, and fish could be infected although these infections are likely to be sub-clinical. Noticeable effects on the environment would be unlikely.

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

14.2.4. Risk Estimation

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

14.3. RISK MANAGEMENT

14.3.1. Options

Infected sheep and goats could be subclinical, long term carriers of infection and quarantine would not prevent the entry of the organism. However, quarantine in tick free premises together with serological testing would ensure that animals are not in the incubation period of the disease and are serologically negative at the time of importation.

Since suitable samples such as placenta or organs of aborted foetuses are not available in animals being tested for importation, culture or demonstration of organisms or specific DNA sequences are not suitable methods for diagnosing infection in carrier animals. Although the sensitivity of ELISAs and the complement fixation test in chronically infected animals is poorly defined, serological testing has been the traditional method of testing imported animals for many years.

Reliable methods of treatment and vaccination are not available.

Since ticks may be involved in transmitting the organism, imported animals should be tick-free.

Germplasm could be tested for the presence of *Coxiella burnetii* before being imported. Therefore importation of tested germplasm could be an alternative method of importing genetic material.

There is not a chapter on Q fever in the *Code*.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with *Coxiella burnetii* in the commodity:

- Animals to be imported could be required to be serologically tested within the two weeks prior to shipment, with negative results.
- All measures to prevent the importation of ticks discussed in Section 13.3. could be implemented including a requirement that animals for export be quarantined in tick-free premises for at least 30 days. Animals could be tested by an ELISA, with negative results at least 23 days after entry into pre-export isolation and within 7 days prior to shipment.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm that has been tested and shown to be free from *Coxiella burnetii*.

References

References marked * have been sighted as summaries in electronic media.

- Arricau-Bouvery N, Rodolakis A (2005).** Is Q fever an emerging or re-emerging zoonosis. *Veterinary Research*, 36, 327-49.
- Behymer D, Riemann HP (1989).** Coxiella burnetii infection (Q Fever). *Journal of the American Veterinary Medical Association* 194, 764-7.
- Hatchette T, Campbell N, Hudson R, Raoult D, Marrie TJ (2003).** Natural history of Q fever in goats. *Vector Borne Zoonotic Diseases*, 3(1), 11-5.
- Hawker JI, Ayres JG, Blair L (1998).** A large outbreak of Q fever in the West Midlands, a windborne spread to a metropolitan area? *Communicable Diseases and Public Health*, 1(3), 180-7.*
- Heath ACG (2002).** Vector competence of *Haemaphysalis longicornis* with particular reference to blood parasites. *Surveillance*, 29(4), 12-4.
- Kelly J (2004).** Q fever. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Pp. 565-72. Oxford University Press, Oxford.
- Kruszewska D, Lembowicz K, Tylewska-Wierzbanska S (1996).** Possible sexual transmission of Q fever among humans. *Clinical Infectious Diseases*, 22(6), 1087-8.*
- Kruszewska D, Tylewska-Wierzbanska S (1997).** Isolation of *Coxiella burnetii* from bull semen. *Research in Veterinary Science*, 62(3), 299-300.*
- Kruszewska D, Tylewska-Wierzbanska SK (1993).** *Coxiella burnetii* penetration into the reproductive system of male mice, promoting sexual transmission of infection. *Infection and Immunity*, 61(10), 4188-95.*
- Marin M, Raoult D (1999).** Q fever. *Clinical Microbiology Reviews*, 12, 518-53.
- Marrie TJ (1990).** Q fever - a review. *Canadian Veterinary Journal*, 31, 551-63.
- Ministry of Agriculture and Forestry (2007).** The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.
- OIE (2007).** Handistatus, OIE. <http://oie.int/hs2/report.asp>, downloaded 8/1/2008.
- Raoult D, Fenollar F, Stein A (2002).** Q fever during pregnancy: diagnosis, treatment, and follow-up. *Archives of Internal Medicine*, 162(6), 701-4.*
- Rousset E, Russo P, Pepin M, Aubert MF (2004).** Q fever. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Pp. 387-98. OIE, Paris.
- Selvaggi TM, Rezza G, Scagnelli M, Rigoli R, Rassu M, De Lalla F, Pellizzer GP, Tramarin A, Bettini C, Zampieri L, Belloni M, Pozza ED, Marangon S, Marchioretto N, Togni G, Giacobbo M, Todescato A, Binkin N (1996).** Investigation of a Q-fever outbreak in Northern Italy. *European Journal of Epidemiology*, 12(4), 403-8.*
- Smith DJW (1941).** Studies on the epidemiology of Q fever. III The transmissio of Q fevr by the tick *Haemaphysalis humerosa*. *Australian Journal of Experimental Biology and Medical Science*, 18, 103-6.
- Smith DJW (1942).** Studies on the epidemiology of Q fever. II Experimental infection of the ticks *Haemaphysalis bispinosa* and *Ornithodoros* sp. with *Rickettsia burnetii*. *Australian Journal of Experimental Biology and Medical Science*, 20, 295-6.
- Tissot-Dupont H, Torres S, Nezri Mea (1999).** Hyperepidemic focus of Q fever related to sheep and wind. *American Journal of Epidemiology* 150(1), 67-74., 150(1), 67-74.*

Woldehiwet Z (2004). Q fever (coxiellosis): epidemiology and pathogenesis. *Research in Veterinary Science*, 77(2), 93-100.

15. Theileriosis

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological Agent

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

15.1.2. OIE List

Not listed

15.1.3. New Zealand Status

Theileria spp. (pathogenic species) are classified as exotic notifiable organisms (Ministry of Agriculture and Forestry 2006). Non pathogenic *Theileria orientalis* occurs in cattle (James et al 1984).

15.1.4. Epidemiology

Four species of *Anaplasma* have been described in Australia, *Theileria buffeli* (in cattle), and three species in marsupials of which only one is unequivocally a *Theileria* species (Stewart et al 1996).

15.1.5. Hazard Identification Conclusion

Since *Theileria* spp. of sheep and goats are not known to occur in Australia, they are not classified as potential hazards in the commodity.

References

References marked * have been sighted as summaries in electronic media.

James MP, Saunders BW, Guy LA, Brookbanks EO, Charleston WAG, Uilenberg G (1984). *Theileria orientalis*, a blood parasite of cattle. First report in New Zealand. *New Zealand Veterinary Journal.*, 32, 154-6.

Ministry of Agriculture and Forestry (2006). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.

Schnittger L, Yin H, Gubbels MJ, Beyer D, Niemann S, Jongejan F, Ahmed JS (2003). Phylogeny of sheep and goat *Theileria* and *Babesia* parasites. *Parasitology Research*, 91(5), 398-406.*

Stewart P, Uilenberg G, de Vos AP (1996). Review of Australian species of *Theileria*, with special reference to *Theileria buffeli* of cattle. *Tropical Animal Health and Production*, 28(1), 81-90.*

Yin H, Luo J, Schnittger L, Lu B, Beyer D, Ma M, Guan G, Bai Q, Lu C, Ahmed J (2004). Phylogenetic analysis of *Theileria* species transmitted by *Haemaphysalis qinghaiensis*. *Parasitology Research*, 92(1), 36-42.

16. Ticks

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological Agents

World wide there are around 170 species of Argasidae or soft ticks and 650 species of Ixodidae or hard ticks (Allan 2001). Many of these species are known to infest sheep and goats.

16.1.2. OIE List

Not listed. However, several tick species are vectors of diseases included in the OIE list.

16.1.3. New Zealand Status

Only one species of livestock tick, *Haemaphysalis longicornis*, occurs in New Zealand. Some species of ticks are listed as unwanted organisms in New Zealand e.g. *Amblyomma* spp. and *Ixodes* spp. (Ministry of Agriculture and Forestry 2006).

16.1.4. Epidemiology

Ticks cause serious economic losses to countries that are infested with them. Fifty four species of ticks have been reported as occurring in Australia (Tick-alert-support-group 2007) but another source suggested that there are 75 known species (Department-of-Medical-Entomology 2003). The important diseases carried by ticks in Australia include the cattle diseases babesiosis and anaplasmosis. Ticks are generally less of a problem in sheep and goats than in cattle but at least 6 genera of ticks are known to infest sheep; *Amblyomma*, *Boophilus*, *Dermacentor*, *Haemaphysalis*, *Ixodes*, and *Rhipicephalus*. Species of five of these genera are present in Australia. The most important tick in Australia is the cattle tick *Boophilus microplus* a vector of babesiosis and anaplasmosis. *Boophilus microplus* is confined to a defined zone in tropical parts of northern and eastern Australia. Another important species is *Ixodes holocyclus* that causes tick paralysis in humans and animals. Introduction of ticks from Australia would, even if they were not carrying diseases, render New Zealand livestock susceptible to tick-borne diseases should they be introduced at a later stage. Ticks are vectors for a large number of diseases and also cause tick toxicoses. Norval and Horak (2004) list 33 tick-associated diseases and toxicoses of livestock that occur in southern Africa. The list is not complete even for Africa and does not include diseases of cats, dogs, wildlife species, and humans. Nine diseases that occur in North America have been listed (Allan 2001). The livestock diseases carried by ticks include economically important diseases such as heartwater, babesiosis, anaplasmosis, theileriosis, and African swine fever. Although many of the Australian ticks are primarily parasites of native animals the likelihood that some species could be found parasitizing sheep or goats is non-negligible.

Worldwide losses due to tick-borne diseases and tick control have been estimated to cost several billion dollars annually (Jongejan and Uilenberg 1994). Apart from losses due to diseases carried by ticks, infestations with ticks also cause significant production losses and losses for tick control (Norval and Horak 2004, Jonsson et al 2001). Infestations with

Ixodes holocyclus, the paralysis tick, cause significant loss of livestock particularly in calves, in the endemic areas in eastern Australia.

New Zealand has only one livestock tick and no significant tick-borne diseases. Many important ticks such as *Amblyomma* spp. might not be able to establish in the New Zealand environment. However, others such as *Ixodes* spp. will probably be able to establish, and all ticks should be excluded.

Hard ticks (Ixodidae) have a life cycle that is divided into 4 stages: egg; larva with 6 legs; nymphs with 8 legs and no genital pore; adults with 8 legs and a genital pore. Different species of ticks may have one-host, two-host, or three-host life cycles. 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 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 some *Rhipicephalus* spp. 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 and Horak 2004).

Soft ticks (Argasidae) are economically less important than hard ticks but there are still several undesirable species that infest cattle including *Otobius megnini* the spinous ear tick that occurs in Australia. 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 unlikely to be imported on live animals.

Consideration of the life cycles of ticks is important when designing programmes to prevent the entry of ticks into New Zealand (see Section 13.3).

Many species of ticks in several countries including Australia have developed resistance to acaricides used to control them (Jongejan and Uilenberg 1994; Jonsson et al 2000; Li et al 2003; Li et al 2004; Mekonnen et al 2002). Development of resistance is an ongoing, developing problem.

16.1.5. Hazard Identification Conclusion

All except one species of tick are exotic to New Zealand. A large number of tick species occurs in Australia and the Australian cattle tick *Boophilus microplus* is a particularly important vector of diseases of cattle. Exotic ticks are therefore classified as potential hazards in the commodity.

16.2. RISK ASSESSMENT

16.2.1. Entry Assessment

Sheep and goats that have been incorrectly treated for ticks, inadequately inspected or are carrying ticks that are resistant to acaricides used to treat them could introduce ticks into New Zealand. In some cases small tick larvae may be almost impossible to detect during physical inspections of sheep and goats. Therefore the likelihood of introducing tick species is considered to be non-negligible.

16.2.2. Exposure Assessment

Introduced sheep and goats will be integrated into New Zealand flocks. Ticks being carried could leave their hosts, complete their life cycles, and infest New Zealand sheep, goats, or cattle. This could result in establishment of new species of ticks. The likelihood of exposure is therefore considered to be non-negligible.

16.2.3. Consequence Assessment

Exposure of New Zealand sheep, goats, and cattle to ticks, and establishment of new species of ticks in New Zealand could result in transmission of diseases carried by the ticks to domestic ruminants. It could also result in heavy infestations of ticks with associated production losses as well as the expenses incurred to control ticks. In addition, introduced ticks, even if not infected with disease agents, would represent an ongoing potential threat since subsequently introduced disease agents would have a ready source of vectors waiting to propagate and transmit them.

Since several human diseases are transmitted by ticks, establishment of new species of ticks could result in endemic foci of human tick borne diseases being established.

Feral mammals could become infested by ticks imported on sheep and goats and could be infected with several diseases transmitted by ticks.

Since farmed animals, feral animals and humans could become infected with diseases carried by ticks, the consequences of introducing ticks are considered to be non-negligible.

16.2.4. Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and ticks are classified as hazards in the commodity. Therefore, risk management measures can be justified.

16.3. RISK MANAGEMENT

16.3.1. Options

Resistance of ticks to acaricides is a real and increasing problem. Therefore the response to any acaricide treatment should be monitored and only acaricides that are effective should be used.

Quarantine facilities should be tick free and constructed in a manner in which they can be maintained free from ticks. Bedding and feed used in quarantine could be contaminated

with ticks. If bedding is not removed regularly animals in quarantine could be reinfested with larva of ticks that have hatched from eggs deposited in the bedding by ticks introduced with the quarantined animals.

The following points relevant to the life cycles of ticks should be considered when designing a quarantine protocol to prevent the importation of ticks:

- While in pre-export isolation sheep and goats kept in an open paddock could become infested with ticks. Therefore sheep and goats should be kept in a building with a smooth impervious floor (preferably concrete) and smooth painted walls, or on a fenced, impervious (preferably concrete) pad without walls and surrounded by a cleared area free from vegetation.
- Before introduction into the building or holding pad, sheep or goats could be dipped or sprayed to reduce or eliminate their tick burden.
- The animals could then be moved into the quarantine premises which have been thoroughly cleaned by high pressure hosing or preferably by steam cleaning and sprayed with an acaricide of proven efficacy.
- Bedding should not be grass, straw, or other plant material that could be infested with ticks. Suitable materials are wood shavings, sterilised peat, or other inert materials.
- The food supply could also be tick free. Processed pellets which have been heated in the pelleting process could be used. The pellets could be lucerne pellets or pellets containing some grain etc.
- If the animals are infested with three-host ticks it can be assumed that the larvae or nymphs will leave the animal within about 3 days. When conditions are favourable (temperature and humidity), moulting may occur in as little as 10 days and recently moulted nymphs or adult ticks could re-infest the same host within one quarantine period. It is important to prevent re-infestation taking place. All bedding could be removed from the building every ten days and disposed of so that ticks cannot re-infest the animals in quarantine. After removal of the bedding the walls and floors could be steam cleaned or cleaned by high pressure hosing and sprayed with an insecticide and clean bedding used in the holding premises. If this procedure is repeated every 10 days eggs and other stages of ticks will be removed and will not be present to re-infest the quarantined animals.
- Two-host ticks may be on the animal somewhat longer than one-host ticks but could also be caught up and removed during the regular clean-ups.
- One-host ticks, such as the very important *Boophilus microplus* remain on the host animal through larval, nymph and adult stages and mate on the host. They are likely to be on the host for about 3 weeks before dropping off and laying some 2-4,000 eggs. Eggs can hatch within 19 days and a life cycle could be completed in 40 days (Allan 2001). If sheep and goats are housed in a building that is not regularly cleaned or if quarantined in a paddock then it would be possible for a *Boophilus* ticks to be fully engorged at the time of entering quarantine and to leave the animals and lay a package of several thousand eggs. These eggs could be hatched and ready to find a host within one 30-day quarantine period. If animals are quarantined in paddocks or in buildings that are not properly cleaned, ticks could be imported into New Zealand. However, regular and conscientious cleaning and disinfection will catch all ticks leaving the host while in quarantine and no single female tick is likely to stay on the host for more than 30 days.

Ticks cannot be introduced in germplasm.

There is not a chapter in the *Code* relating to ticks.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with ticks in the commodity:

- Animals to be imported could be treated with an acaricide within 2 days of shipment.
- Animals to be imported could be treated with a pour-on acaricide 7-10 days before entering pre-export isolation.
- Animals to be imported could be treated during the 48 hours immediately prior to entering pre-export isolation 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).
- Animals to be imported could be held isolated for 30 days in quarantine premises with impervious washable floor and walls or on a 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 are suitable. The animals could be fed rations (preferably pelleted) that are free from potential contamination with ticks, tick eggs, larvae or nymphs. Pelleted rations are recommended.
- Bedding used in pre-export isolation could be removed every ten days during the quarantine period and at this time, the walls and floor could be cleaned by high pressure hosing or steam cleaning and sprayed with an acaricide.
- Animals held in pre-export isolation could be meticulously inspected for ticks and other ectoparasites, 10 days after entering pre-export isolation. If still infested, acaricide treatment could be repeated and animals inspected again 10 days later. Treatments and testing could be repeated until the animals are found to be free from evidence of ticks. The acaricide could be altered if the previously used treatment has not been effective.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

References marked * have been sighted as summaries in electronic media.

Allan SA.(2001). Ticks (Class Arachnida: Order Acarina). In: Samuel WM, Pybus MP, Kocan AA (eds). Parasitic Diseases of Wild Mammals. Pp. 72-106. Manson Publishing Ltd, London.

Department-of-Medical-Entomology (2003). Ticks. <http://medent.usyd.edu.au/fact/ticks.htm> , downloaded 8/1/2008.

Jongejan E, Uilenberg G (1994). Ticks and tick control methods. Revue Scientifique et Technique. OIE, 13(4), 1201-20.

Jonsson NN, Davis R, De Witt M (2001). An estimate of the economic effects of cattle tick (*Boophilus microplus*) infestation on Queensland dairy farms. Australian Veterinary Journal, 79(12), 826-31.

Jonsson NN, Mayer DG, Green PE (2000). Possible risk factors on Queensland dairy farms for acaricide resistance in cattle tick (*Boophilus microplus*). Veterinary Parasitology, 88(1-2), 79-92.*

Li AY, Davey RB, Miller RJ, George JE (2003). Resistance to coumaphos and diazinon in *Boophilus microplus* (Acari: Ixodidae) and evidence for the involvement of an oxidative detoxification mechanism. Journal of Medical Entomology, 40(4), 482-90.*

Li AY, Davey RB, Miller RJ, George JE (2004). Detection and characterization of amitraz resistance in the southern cattle tick, *Boophilus microplus* (Acari: Ixodidae). Journal of Medical Entomology, 41(2) 193-200.*

Mekonnen S, Bryson NR, Fourie LJ, Peter RJ, Spickett AM, Taylor RJ, Strydom T, Horak IG (2002). Acaricide resistance profiles of single- and multi-host ticks from communal and commercial farming areas in the Eastern Cape and North-West Provinces of South Africa. Onderstepoort Journal of Veterinary Research, 69(2), 99-105.

Ministry of Agriculture and Forestry (2006). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.

Norval RAI, Horak IG.(2004). Vectors: Ticks. In: Coetzer JAW, Tustin RC (eds). Infectious Diseases of Livestock. Pp. 3-42. Oxford University Press, Cape Town.

Tick-alert-support-group (2007). Ticks of Australia. <http://www.tickalert.org.au/ticksaust.htm#AustralianTicks>., downloaded 8/1/2008.

17. Lice

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological Agents

Lice of sheep: *Bovicola ovis*, *Linognathus ovillus*, *Linognathus pedalis*,

Lice of goats: *Bovicola caprae*, *Bovicola limbata*, *Linognathus stenopsis*, *Linognathus africanus*

17.1.2. OIE List

Not listed.

17.1.3. New Zealand Status

All the organisms listed in Section 14.1.1 except *Linognathus africanus* are present in New Zealand (Kettle 1974; Merral 1986; Tenquist and Charleston 2001).

17.1.4. Epidemiology

In both New Zealand and Australia the most common lice of sheep are *Bovicola ovis* with a flock prevalence of 20-40% in Australia (James and Riley 2004; Morcombe et al 1994; Veterinary-Education-and-Information-News 2002). The parasite occurs commonly in New Zealand and resistance to insecticides, especially the pyrethroids is widespread in both countries (Heath 2007). The literature on lice in Australia is dominated by articles on *Bovicola ovis*. *Linognathus africanus* is rarely mentioned and it is assumed to be uncommon and of little economic importance.

17.1.5. Hazard Identification Conclusion

Only *Linognathus africanus* is exotic to New Zealand. This parasite is therefore classified as a potential hazard in the commodity. Other lice are not potential hazards and steps to control them are considered to be the responsibility of the importer.

17.2. RISK ASSESSMENT

17.2.1. Entry Assessment

Since infestation of sheep and goats with *Linognathus africanus* in Australia is uncommon the likelihood that they will be introduced with imported sheep and goats is considered to be low but non-negligible.

17.2.2. Exposure Assessment

Imported sheep and goats will be integrated into New Zealand flocks and there would be ample opportunity to transfer their parasites to New Zealand sheep and goats. The exposure assessment is therefore considered to be non-negligible.

17.2.3. Consequence Assessment

The introduction of the parasite would result in minor and sporadic economic loss due to irritation of infested animals, downgrading of fibrewool and skins, and costs for treatment.

Since lice are generally species specific and *Linognathus africanus* infestations of animals other than sheep and goats have not been described, there are unlikely to be any consequences for human health or the environment

Since the parasite is likely to cause only minor problems for individual farmers, the consequences of introducing the parasite are considered to be low but non-negligible.

17.2.4. Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and *Linognathus africanus* is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

17.3. RISK MANAGEMENT

17.3.1. Options

The economically important lice species occur in both New Zealand and Australia. *Linognathus africanus* probably occurs only rarely in Australia. It has not been described in New Zealand but it is uncertain whether or not it is present.

Effective treatment for lice involves repeated treatments at suitable intervals to eliminate adult lice and lice that have hatched after the first treatment.

Lice could not be introduced if germplasm was imported instead of live animals.

The *Code* does not contain a chapter on lice.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with lice in the commodity:

- Unrestricted importation of sheep and goats could be allowed.
- Sheep and goats to be imported could be inspected to verify that they are free from lice.
- Sheep and goats could be quarantined for 3 weeks. Animals could be treated prior to entering pre-export isolation and twice while in isolation with an interval of 14 days. Insecticides could be chosen that are already being used for the elimination of other parasites. Treatments for all types of parasites could be integrated and regularly adapted so as to use the most effective insecticides, taking into account the availability of new insecticides and the development of insecticide resistance to commonly used chemicals. Sheep and goats could be inspected before shipment to establish that the treatment has been effective. If treatment has not been effective shipment of animals could be prohibited and quarantine and treatment could be repeated.

- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

Heath ACG (2007). Sheep biting-louse resistance management.

<http://www.nzpps.org/resistance/sheeplouse.php>, downloaded 8/1/2008.

James PJ, Riley MJ (2004). The prevalence of lice on sheep and control practice in South Australia. Australian Veterinary Journal, 82(9), 563-8.

Kettle PR (1974). Epidemiology and damage caused by external parasites of sheep in New Zealand. Proceeding of the Society of Sheep and Beef Cattle Veterinarians of the New Zealand Veterinary Association, 4, 3-14.

Merral M (1986). Skin diseases of goats. Proceeding of the Society of Sheep and Beef Cattle Veterinarians of the New Zealand Veterinary Association, 16, 83-9.

Morcombe PW, Thompson ND, Buckman PG (1994). The prevalence of lice (*Bovicola ovis*) infested sheep flocks in Western Australia (1987-93). Australian Veterinary Journal, 71(3), 71-4.

Tenquist JD, Charleston WAG (2001). A revision of the annotated checklist of ectoparasites of terrestrial mammals in New Zealand. Journal of the Royal Society of New Zealand, 31(3), 481-54

[http://www.rsnz.org/publish/jrsnz/\(2001\)/33.php](http://www.rsnz.org/publish/jrsnz/(2001)/33.php), downloaded 8/1/2008.

Veterinary-Education-and-Information-News (2002). Sheep health and production. Chapter 10. Special diseases of the integument. <http://vein.library.usyd.edu.au/sheephealth/Chapter10.html>, downloaded 8/1/2008.

18. Mites

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological Agents

Chorioptes bovis, *Demodex aries*, *Demodex ovis*, *Psorobia ovis* (*Psorergates ovis*), *Psoroptes ovis*, and *Psoroptes cuniculi*.

18.1.2. OIE List

Not listed.

18.1.3. New Zealand Status

All species listed in above are present in New Zealand except *Psoroptes ovis* which has been eradicated (Heath 1978; Heath et al 1983; Heath et al 1989; Heath 2002; Rhodes 1975; Rhodes 1976; Tenquist and Charleston 2001).

18.1.4. Epidemiology

Demodex aries and *Demodex ovis* may be the same species. *Psoroptes ovis* and *Psoroptes cuniculi* are closely related and their taxonomic status is not yet finalised. However, sheep scab caused by *Psoroptes ovis* is distinct from the clinical picture caused by *Psoroptes cuniculi* which is characterized by infestations in the ears of goats and rabbits.

Sheep scab has been eradicated from both New Zealand and Australia. No evidence was found of the occurrence of any mites that infect sheep and goats in Australia that do not occur in New Zealand.

18.1.5. Hazard Identification Conclusion

There are no mite parasites of sheep and goats that occur in Australia that do not occur in New Zealand. Therefore mites are not considered to be a potential hazard in the commodity and it is a quality issue for importers if they wish to ensure that animals they import are free from mange mites. Despite this, treatments that may be applied for ticks, lice, and internal parasites will almost certainly eliminate any mange mites that are present on imported sheep and goats.

References

Heath AC (1978). The scrotal mange mite, *Chorioptes bovis* (Hering, 1845) on sheep: seasonality, pathogenicity and intra-flock transfer. *New Zealand Veterinary Journal*, 26(12), 299-300.

Heath AC, Bishop DM, Tenquist JD (1983). The prevalence and pathogenicity of *Chorioptes bovis* (Hering, 1845) and *Psoroptes cuniculi* (Delafond, 1859) (Acari: Psoroptidae) infestations in feral goats in New Zealand. *Veterinary Parasitology*, 13(2), 159-69.*

Heath AC, Bishop DM, Tenquist JD (1989). Observations on the potential for natural transfer of *Psoroptes cuniculi* and *Chorioptes bovis* (Acari: Psoroptidae) between goats and sheep. *New Zealand Veterinary Journal*, 37(2), 56-8.

Heath ACG (2002). Recently introduced exotic animals and their parasites: what risk to New Zealand's biosecurity? *Surveillance*, 29(4), 15-7.

Rhodes AP (1975). Seminal degeneration associated with chorioptic mange of the scrotum of rams. *Australian Veterinary Journal*, 51(9), 428-32.

Rhodes AP (1976). The effect of extensive chorioptic mange of the scrotum on reproductive function of the ram. *Australian Veterinary Journal*, 52(6), 250-7.

Tenquist JD, Charleston WAG (2001). A revision of the annotated checklist of ectoparasites of terrestrial mammals in New Zealand. *Journal of the Royal Society of New Zealand*, 31(3), 481-542
[http://www.rsnz.org/publish/jrsnz/\(2001\)/33.php](http://www.rsnz.org/publish/jrsnz/(2001)/33.php), downloaded 8/1/2008.

19. Internal Parasites

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological Agents

All internal parasites including nematodes, cestodes, and trematodes except for *Echinococcus granulosus*, which is considered separately in Section 17.

19.1.2. OIE List

Not listed.

19.1.3. New Zealand Status

Many parasites occur commonly in New Zealand. *Nematodirus battus* is classified as an other exotic organism (Ministry of Agriculture and Forestry 2007). *Oesophagostomum columbianum* is not listed in the MAF register of unwanted organisms but has not been described in New Zealand (McKenna 1997) and is a significant parasite of sheep.

19.1.4. Epidemiology

Internal parasites belong to three basic groups:

- i. Cestodes or tapeworms.
- ii. Trematodes or flukes (paramphistomes and liver flukes).
- iii. Nematodes which include mainly intestinal parasites but also include lungworms and a few other curiosity parasites such as eyeworms (*Thelazia* spp).

Internal parasites occur commonly in New Zealand and the importation of species of parasites that already occur here is not regarded as a biosecurity risk. However, anthelmintic resistance of parasites is a major problem that occurs world-wide. Introduction of parasites that are resistant to an anthelmintic type for which resistant parasites do not presently occur in New Zealand should be considered a biosecurity risk. Anthelmintic resistance to the commonly used anthelmintics used for nematode control is widespread in New Zealand (Waghorn et al 2006). Anthelmintic resistance in liver fluke has not yet been described in New Zealand but occurs in Australia and Europe (Boray 1999; Moll et al 2000; Sargison 2005).

Paramphistomes are present in New Zealand, and no reports were found of anthelmintic resistance in paramphistomes. Therefore resistant paramphistomes are not considered to be a potential hazard in introduced sheep and goats.

The intestinal (*Moniezia* spp.) and liver (*Stillesia hepatica*) tapeworms of sheep, goats, and cattle occur in New Zealand, but are of minor economic importance. Reports of resistance to anthelmintics in these parasites were not found. Therefore these parasites are not considered to be potential hazards in the commodity. The human/cattle tapeworm *Taenia saginata* and its cyst form *Cysticercus cellulosae* that occurs in the muscles of cattle is not a parasite of sheep and goats. *Cysticercus tenuicollis* the cyst form of *Taenia hydatigena*,

Cysticercus ovis the cyst form of *Taenia ovis* and *Coenurus cerebralis* the cyst form of *Taenia multiceps* occur in both Australia and New Zealand. Hydatid cysts, the cyst form of *Echinococcus granulosus* is covered in Section 17. Cestodes other than *Echinococcus granulosus* are not considered to be potential biosecurity hazards.

The liver fluke (*Fasciola hepatica*) is present in New Zealand. Resistance to the anthelmintics used to control liver flukes has not been reported in New Zealand. However, anthelmintic resistance to triclabendazole, the main anthelmintic used for control, has been reported in Australia and Europe (Boray 1999; Moll et al 2000; Sargison 2005). Therefore drench resistant liver fluke are considered to be potential hazards.

The numbers of nematode parasites are too large to be considered individually and since most of them occur universally only a few could be considered to be biosecurity threats. Important species of nematodes that are not established in New Zealand include *Oesophagostomum columbianum* and *Nematodirus battus* (McKenna 1997).

19.1.5. Hazard Identification Conclusion

The importation of new species of parasites and anthelmintic resistant parasites should be avoided. New parasites and anthelmintic resistant parasites are therefore classified as potential hazards in the commodity. Parasites of particular importance are *Nematodirus battus*, *Oesophagostomum columbianum* and drench resistant *Fasciola hepatica*.

19.2. RISK ASSESSMENT

19.2.1. Entry Assessment

New species of parasites or anthelmintic resistant parasites could be introduced with imported sheep and goats and would not be obvious at a clinical examination. Therefore the likelihood of entry is considered to be non-negligible.

19.2.2. Exposure Assessment

Imported sheep and goats will be introduced into New Zealand flocks and shed eggs and larvae of internal parasites on pastures. New Zealand sheep and goats could therefore be exposed to the parasites. The likelihood of exposure is therefore considered to be non-negligible.

19.2.3. Consequence Assessment

Some species of exotic parasites may have the potential to cause more severe disease syndromes than the species presently in New Zealand and the introduction of anthelmintic resistant parasites could hasten the emergence of anthelmintic resistance in New Zealand.

Some sheep and goats parasites can infest other ruminants such as cattle and could infest wild and feral ruminants. However, since many parasites are broadly species specific and wild and feral ruminants are not intensively farmed the effect on them is likely to be minimal. The impact on the environment is therefore likely to be negligible.

Since introduction of new or anthelmintic resistant parasites could have a detrimental effect on sheep and goat farming, the consequence assessment is considered to be non-negligible.

19.2.4. Risk Estimation

Because entry, exposure, and consequence assessments are non-negligible, the risk estimate is non-negligible and internal parasites are classified as hazards in the commodity. Therefore, risk management measures can be justified.

19.3. RISK MANAGEMENT

19.3.1. Options

Keeping sheep and goats on clean impervious floors and regular cleaning and removal of soiled bedding will prevent build up of parasite eggs and larvae and prevent re-infestation of animals while in quarantine. Since larvae of important species of parasites may develop to the infective stage within 4-7 days, it is suggested that bedding should be removed and floors cleaned every 5 days.

Anhelmintic treatment is the only available method of removing parasites from host animals. However the efficacy of drenching should be monitored and repeat treatments with different anthelmintics is necessary where drench resistance is encountered.

To detect all types of parasites methods used to examine faeces samples should include flotation, sedimentation and larval culture methods. Diagnosis of parasite infections is done by identification of eggs or hatched larvae in faeces. Reliance on diagnosis by faecal examination and treatment with anthelmintics has been the method specified for many years in New Zealand's import health standards and those of our trading partners. No other practical methods are available for this purpose. Identification of single species of parasites as part of a quarantine procedure is not possible. Therefore the criterion generally used for imported animals is that they should be entirely free from all parasite eggs in the standard egg flotation method.

External parasites are not transmitted in germplasm.

The *Code* does not have a chapter on internal parasites.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with internal parasites in the commodity:

- Sheep and goats for importation could be treated with endoparasiticides effective against endoparasites (including liver fluke) within the seven days prior to shipment.
- Sheep and goats for importation could be held in quarantine for a period of 30 days in premises with an impervious washable floor. While in quarantine soiled bedding could be removed at least every 5 days and floors could be washed by high pressure hosing or steam cleaning.
- Individuals in pre-export isolation could be treated with an endoparasiticide within 48 hours after entering quarantine. The efficacy of the endoparasiticides could be checked 7-14 days after the endoparasite treatment by examining faeces samples from the treated sheep and goats by the faecal floatation concentration method (Egwang and Slocombe 1982)

and sedimentation methods and give zero parasite egg counts. Treatments and testing could be repeated on animals that have positive egg counts until they give zero parasite egg counts, the anthelmintic type could be changed as necessary.

- Where faecal examinations of isolated animals have demonstrated surviving parasites, larval cultures could be made, the parasites identified, and MAFBNZ notified of the results. On receipt of the results MAFBNZ could make a ruling as to whether the importation could proceed.
- Isolated animals could be treated with suitable endoparasiticides within 3 days of shipment.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

References marked * have been sighted as summaries in electronic media.

Boray JC (1999). AGFACTS. NSW Agriculture > Liver fluke disease of sheep and cattle. http://www.dpi.nsw.gov.au/data/assets/pdf_file/0004/114691/liver-fluke-disease-in-sheep-and-cattle.pdf, downloaded 8/1/2008.

Egwang TG, Slocombe JOD (1982). Evaluation of the Cornwell-Wisconsin centrifugal flotation technique for recovering trichostrongylid eggs from bovine feces. Canadian Journal of Comparative Medicine, 46, 133-7.

McKenna PB (1997). Checklist of helminth parasites of terrestrial mammals in New Zealand. New Zealand Journal of Zoology, 24, 277-90.

Ministry of Agriculture and Forestry (2007). The Unwanted Organisms Register. <http://mafuwsp6.maf.govt.nz/uor/searchframe.htm>, downloaded 8/1/2008.

Moll L, Gaasenbeek CP, Vellema P, Borgsteede FH (2000). Resistance of *Fasciola hepatica* against triclabendazole in cattle and sheep in the Netherlands. Veterinary Parasitology, 91(1-2), 153-8.*

Sargison N (2005). NADIS, Cattle Disease Focus, Liver Fluke. http://www.qmScotland.co.uk/analysis/downloads/Subacute_Liver_Fluke-nadis.pdf, downloaded 8/1/2008.

Waghorn TS, Leathwick DM, Rhodes AP, Lawrence KE, Jackson R, Pomroy WE, West DM, Moffat JR (2006). Prevalence of anthelmintic resistance on sheep farms in New Zealand. New Zealand Veterinary Journal, 54(6), 271-7.

20. Hydatidosis

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological Agents

Echinococcus granulosus

20.1.2. OIE List

Echinococcosis / hydatidosis listed.

20.1.3. New Zealand Status

Echinococcus granulosus has been eradicated from New Zealand (Pharo 2002).

20.1.4. Epidemiology

The adult parasite is a small tapeworm that occurs in the intestines of dogs that have eaten offal from sheep infested with hydatid cysts. Hydatid cysts, the cyst form of *Echinococcus granulosus*, occur particularly in the lungs, liver, and occasionally in other organs of sheep that have ingested tapeworm eggs from dog faeces.

New Zealand has been declared provisionally free from *Echinococcus granulosus* / hydatidosis (Pharo 2002). The parasite could be re-introduced if an imported animal infested with hydatid cysts were to be fed to dogs.

Echinococcus granulosus can cause a severe (potentially fatal) disease in humans when the cyst stage develops.

The parasite occurs commonly in some regions of Australia where control is rendered impossible as various life cycles have developed involving feral dogs, dingoes, and foxes as primary hosts and sheep, kangaroos, wallabies, and feral pigs as secondary hosts. The prevalence of hydatid cysts and the fertility of the cysts is high in macropod species. The occurrence of the parasite in sheep is highest in areas near to National Parks and where macropods are common. The prevalence of adult tapeworms was up to 100% in wild dogs around the Kosciuszko National Park (Jenkins and Morris 2003). The problems associated with the possible re-introduction of the parasite when importing animals from Australia and possible options to prevent re-introduction have been addressed in an internal report to MAF and are discussed below (Section 17.3).

Diagnosis of hydatids in live sheep is difficult since they show no signs of infestation. Serological diagnosis 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. There is no literature indicating that ultrasound scanning is reliable or has been validated.

Vaccination with a recombinant vaccine has proved to be highly effective when lambs are vaccinated at a young age according to the recommended vaccination regimen. It is not

effective in sheep that are already infested and therefore for control of the infestation vaccination of young lambs is recommended (Gauci et al 2005; Heath and Holcman 1997; Heath et al 2003). However, since the vaccine is not yet being commercially produced and is not registered for use in Australia vaccination is not an option at the present time but may be in the future.

Legal requirements that apply to owners of imported animals and dogs that relate directly to the control of *Echinococcus granulosus* / hydatidosis are:

- i. The Biosecurity (Imported Animals, Embryos, and Semen Information) Regulation 1999. Under this regulation owners of imported animals are obliged to report:
 - (a) the date that ownership of an animal is transferred and the name and address of the new owner;
 - (b) if an animal dies;
 - (c) the date that an animal is slaughtered or consigned to slaughter;
 - (d) if the animal cannot be located;
 - (e) if eartags issued in respect of the importation of an animal are lost or become illegible.

MAF and freezing works also keep registers of imported animals and MAF annually verifies that the imported animal is still alive and living at the declared place.

- ii In addition the Biosecurity (Declaration of a Controlled Area) Notice No 1204 of 02 August 2001 declares the whole of New Zealand to be a controlled area in which:
 - (i) The slaughter of ruminants and pigs at home killing facilities within the controlled area shall be conducted within a dog-proof enclosure in such a manner as to ensure that raw offal is not accessible to dogs;
 - (ii) owners shall control their dogs at all times in such a manner as to prevent them from having access to raw offal of ruminants and pigs;
 - (iii) the offal of ruminants and pigs shall be cooked by boiling for a minimum of 30 minutes before feeding to dogs within the controlled area.

20.1.5. Hazard Identification Conclusion

Provided the relevant regulations are strictly followed, it is unlikely that hydatids could become established in New Zealand. However, it is not reasonable to expect that an animal that dies in a remote part of a farm will be discovered by the owner before it is discovered and eaten by dogs and therefore *Echinococcus granulosus* / hydatidosis is classified as a potential hazard in the commodity.

20.2. RISK ASSESSMENT

20.2.1. Entry Assessment

Echinococcus granulosus occurs commonly in some areas of Australia. Since sheep and goats with hydatid cysts would not show any clinical signs and suitable diagnostic methods

to recognize infested sheep are not available, the likelihood of entry is considered to be non-negligible.

20.2.2. Exposure Assessment

Although the whole of New Zealand is a controlled area in which the feeding of uncooked offal is forbidden (see Section 17.1.4), access of dogs to dead sheep may be impossible to prevent in some cases. Imported sheep and goats will be introduced into New Zealand flocks and could be eaten by dogs when they die. The likelihood of exposure is therefore considered to be non-negligible.

20.2.3. Consequence Assessment

The re-establishment of *Echinococcus granulosus* could result in sporadic cases of human disease and the necessity for an expensive eradication campaign.

Echinococcus granulosus can infest cattle, goats, 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. Re-eradication of the parasite could be expensive depending on the extent to which the parasite has become dispersed before steps are taken to control the incursion.

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

20.2.4. Risk Estimation

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

20.3. RISK MANAGEMENT

20.3.1. Options

New Zealand has eradicated *Echinococcus granulosus* and should seek to preserve this status.

Australian sources of animals free from hydatids are not officially defined since the condition is not reportable in Australia. However, sourcing sheep from properties with a well-documented and reliable history of *Echinococcus granulosus* freedom during the previous 5 years, and from areas which are known to be free from the infestation could reduce the likelihood of importing infected animals. However, it is doubtful that a property would be able to provide sufficient evidence to conclusively demonstrate freedom from *Echinococcus granulosus*. Areas adjacent to national parks and known high risk areas should be avoided (Pharo 2002).

No reliable tests are available for the diagnosis of hydatid cysts in ruminants. Serological tests have low sensitivity and are not generally available. Newly developed vaccines show promise but are not yet available for general use.

The parasite could not establish in New Zealand unless infected carcasses are eaten by dogs. Regulations that apply to controlling the management, reporting movements of imported animals and prohibition of feeding of sheep offal to farm dogs should be enforced (see Section 17.1.4).

To ensure that *Echinococcus granulosus* is not re-introduced it may be appropriate not to allow the importation of live ruminants and to allow only the importation of germplasm for upgrading the New Zealand gene pool. This could be considered for preventing the introduction of all protozoal, internal and external parasites and many viral and bacterial diseases.

One or a combination of the following options could therefore be considered in order to effectively manage the risk associated with *Echinococcus granulosus* in the commodity:

- Sheep and goats could be sourced from properties with reliable records demonstrating that no case of *Echinococcus granulosus* infestation in dogs or hydatid cysts in ruminants or macropods is known to have occurred in the previous 5 years.
- Importers could be made specifically aware of the legal requirements relating to the feeding of carcass material from imported animals to dogs and the requirements for identifying imported animals and reporting deaths or slaughter of imported animals (Section 17.1.4). A prepared information pack detailing this information could be given to all importers of live sheep and goats.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

References marked * have been sighted as summaries in electronic media.

Gauci C, Heath D, Chow C, Lightowlers MW (2005). Hydatid disease: vaccinology and development of the EG95 recombinant vaccine. *Expert Review of Vaccines*, 4(1), 103-12.*

Heath DD, Holcman B (1997). Vaccination against *Echinococcus* in perspective. *Acta Tropica*, 67(1-2), 37-41.

Heath DD, Jensen O, Lightowlers MW (2003). Progress in control of hydatidosis using vaccination--a review of formulation and delivery of the vaccine and recommendations for practical use in control programmes. *Acta Tropica*, 85(2), 133-43.*

Jenkins DJ, Morris B (2003). *Echinococcus granulosus* in wildlife in and around the Kosciuszko National Park, in south-eastern Australia. *Australian Veterinary Journal*, 81(1-2), 81-5.

Kittelberger R, Reichel MP, Jenner J, Heath DD, Lightowers MW, Moro P, Ibrahim MM, Craig PS, O'Keefe JS (2002). Evaluation of three enzyme-linked immunosorbent assays (ELISAs) for the detection of serum antibodies in sheep infected with *Echinococcus granulosus*. *Veterinary Parasitology*, 110(1-2), 57-76.

Pharo H (2002). New Zealand declares 'provisional freedom' from hydatids. *Surveillance*, 29(3), 3-7.

21. Weed Seeds, Plants, and Plant Materials

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological Agent

All plant seeds and plant material.

21.1.2. OIE List

Not listed.

21.1.3. New Zealand Status

Organisms of concern are all exotic plants and plant seeds.

21.1.4. General Considerations

Weeds and weed seeds could be found attached to the wool and hair of sheep and goats. Large seed heads and pieces of plant material would be easily visible and could be removed before shipment but small seeds would not be visible.

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 leemings burrow that was dated as 10,000 years old germinated within 48 hours when placed in favourable conditions (Anonymous 2007). 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 animal's digestive systems and are 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 sheep 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 (MAF 1999) recommended that animals should 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 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.

Some plants can replicate asexually and are able to be grown from cuttings, and could grow from pieces of plants introduced on animals.

21.1.5. Hazard Identification Conclusion

It is concluded that weed seeds or plant material could be introduced on animal's hair or in their faeces. Therefore weed seeds and plant material are classified as potential hazards in the commodity.

21.2. RISK ASSESSMENT

21.2.1. Entry Assessment

Seeds and plant material could be introduced attached to hair or in faeces. The entry assessment is therefore considered to be non-negligible.

21.2.2. 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.

21.2.3. 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.

21.2.4. 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 can be justified.

21.3. RISK MANAGEMENT

21.3.1. Options

The risks of introducing seeds and plant material attached to sheep and goats wool and hair will 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.

Weed seeds, plants and plant material cannot be introduced in germplasm.

There is nothing in the *Code* relating to hitch hiker weeds and plants associated with animals.

One or a combination of the following options could therefore be considered in order to effectively manage the risk due to weed seeds, plants, and plant material in the commodity:

- 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; and.
- Animals could be fed a high quality, seed-free diet to speed passage time in the digestive tract, for at least ten days prior to their arrival in New Zealand.
- Measures discussed in Section 13.3 for the management of risk associated with ticks could also be considered for the control of weeds, weed seeds, and plant material.
- The importation of live sheep and goats could be prohibited and importation of new genetic material restricted to germplasm.

References

Encyclopædia Britannica (2008). Dormancy and life-span of seeds. Encyclopædia Britannica Online. <http://britannica.com/eb/article-75927>, downloaded 8/1/2008.

Barton K, Williams P.A (2001). Passage time for weed seeds in the digestive tract of herbivorous livestock. Landcare Research Contract Report: LC 0001/065.

Katovich J, Becker R, Doll J (undated). Weed seed survival in livestock systems. A publication of University of Minnesota extension service. <http://www.manure.umn.edu/assets/WeedSeedSurvival.pdf>, downloaded 8/1/2008.

MAF (1999). Import Risk Analysis : Importation of Weed Species by Live Animals and Unprocessed Fibre of Sheep and Goats MAF, Wellington, New Zealand <http://www.biosecurity.govt.nz/files/pests-diseases/animals/risk/weeds-seeds-ra.pdf>, downloaded 8/1/2008.).