

**Import Risk Analysis:
White Rhinoceros
(*Ceratotherium simum*)
from Australia.**



ISBN 978-0-478-35760-8 (print)
ISBN 978-0-478-35761-5 (online)

23 December 2009

This page is intentionally blank

MAF Biosecurity New Zealand
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6140
New Zealand

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

Policy and Risk
MAF Biosecurity New Zealand



Import Risk Analysis: White Rhinoceros
(*Ceratotherium simum*) from Australia

23 December 2009

Approved for public 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 Page

Executive Summary	1
1. Introduction	2
2. Scope	2
3. Commodity Definition	2
4. Risk Analysis Methodology	2
5. Akabane and other Simbu Group Viruses	10
6. Orbiviruses: Bluetongue and Epizootic haemorrhagic disease of deer	13
7. Ross River Fever Virus	16
8. Bovine Herpes Viruses	19
9. Anthrax (<i>Bacillus anthracis</i>)	22
10. Salmonellosis (<i>Salmonella</i> spp.)	26
11. Leptospirosis (<i>Leptospira</i> spp)	32
12. Babesiosis (<i>Babesia</i> spp.)	37
13. Internal Parasites	39
14. Ticks	44
15. Weed Seeds	49

Contributors to this risk analysis

Primary author

R Somervell	Senior Advisor Risk Analysis (Animals)	MAF Biosecurity New Zealand
-------------	---	-----------------------------

Internal peer review

R Worthington	Contractor Risk Analysis (Animals)	MAF Biosecurity New Zealand
---------------	---------------------------------------	-----------------------------

L Broad	Senior Advisor Risk Analysis (Animals)	MAF Biosecurity New Zealand
---------	---	-----------------------------

M Hernandez	Senior Advisor Animal Imports and Exports	MAF Biosecurity New Zealand
-------------	--	-----------------------------

H Pharo	Team Manager Risk Analysis (Animals)	MAF Biosecurity New Zealand
---------	---	-----------------------------

External peer review

T Portas	Veterinary Services Manager	Australia Zoo QLD
----------	--------------------------------	-------------------

Executive Summary

This document is a qualitative analysis of the biosecurity risks associated with the importation of White Rhinoceros (*Ceratotherium simum*) into New Zealand zoological containment facilities from equivalent facilities in Australia.

From an initial hazard identification list of all disease-causing organisms potentially associated with rhinoceros, 12 agents were identified as being recorded in Australia, but not present in NZ. These organisms were subjected to further analysis in which the epidemiology of the disease including distribution, clinical signs, transmission, diagnosis and any available treatments were considered. As a result of this process organisms were classified as potential hazards or not in the commodity.

Those organisms identified as potential hazards have been subjected to ‘risk assessment’ to estimate the likelihood of entry (the disease agent being present at the time of animal importation) exposure (likelihood of spread and establishment if imported) and any adverse consequences likely to follow these events. Finally, risk management options that could be used to effectively manage the risk have been presented for organisms considered to be hazards in the commodity. These options are suggested options to be considered when drafting an Import Health Standard (IHS) for the commodity.

Risk management measures were not required for certain arthropod-borne agents because the specific vectors (mainly ticks) are not present in New Zealand. However, if new vector species were to establish here, ‘risk assessment’ would become necessary for organisms which would otherwise be eliminated from the preliminary hazard list.

The risk analysis concluded that the following agents present non-negligible risks, and that sanitary measures can be justified to effectively manage them:

- Anthrax (*Bacillus anthracis*)
- Salmonella spp.
- Leptospira spp.
- Ticks
- Internal Parasites
- Weeds/weedseeds

Any of the proposed risk management options selected should be the least trade restrictive while providing an appropriate level of protection to effectively manage the risk.

1. Introduction

This risk analysis has been developed in order to support the high priority Southern White Rhinoceros Conservation Programme managed by ARAZPA. A founder population was established in New Zealand zoological facilities from South African imports in 1999 and 2002. Rhinos from these imports were also added to existing rhino populations in Australian Zoos. Captive breeding within Australasia has been successful, and in order to sustainably manage the population it has become necessary to transfer animals between Australia and New Zealand. This will enable genetic diversity to be maintained, birth/sex ratios and social structures to be managed; and therefore ensure that the breeding programme can successfully continue.

2. Scope

Rhinoceros are odd-toed ungulates (mammals with hooves). There are five species of rhinos, from the Family: Rhinocerotidae, Order: Perissodactyla. The Families Equidae (horses) and Tapiridae (Tapirs) are also odd-toed ungulates from the Perissodactyla Order.

This risk analysis qualitatively examines the risks due to exotic disease-causing organisms associated with the importation of white rhinoceroses (*Ceratotherium simum*) from Australia.

Other risk factors that may be of commercial importance to importers (for example genetic diseases) have not been considered in the analysis. The risk analysis does not consider speculative events that could occur in the future, such as the possible establishment of disease vectors (for example *Culicoides* spp.) due to climate change. MAF has the ability to modify any Import Health Standards based on a change in the risk profile when appropriate.

All rhinos imported into New Zealand must be directed into permanent zoological containment facilities. Rhinos must have been born in and been continuously resident in a government registered or licensed zoo or wildlife park.

To be eligible for import the Biosecurity Act 1993 requires MAF to be satisfied that the imported animals do not harbour potentially harmful organisms. A pre-arrival requirement of current Import Health Standards (IHS) is that animals must be certified on the day of travel to be showing no clinical signs of infectious or parasitic disease.

3. Commodity Definition

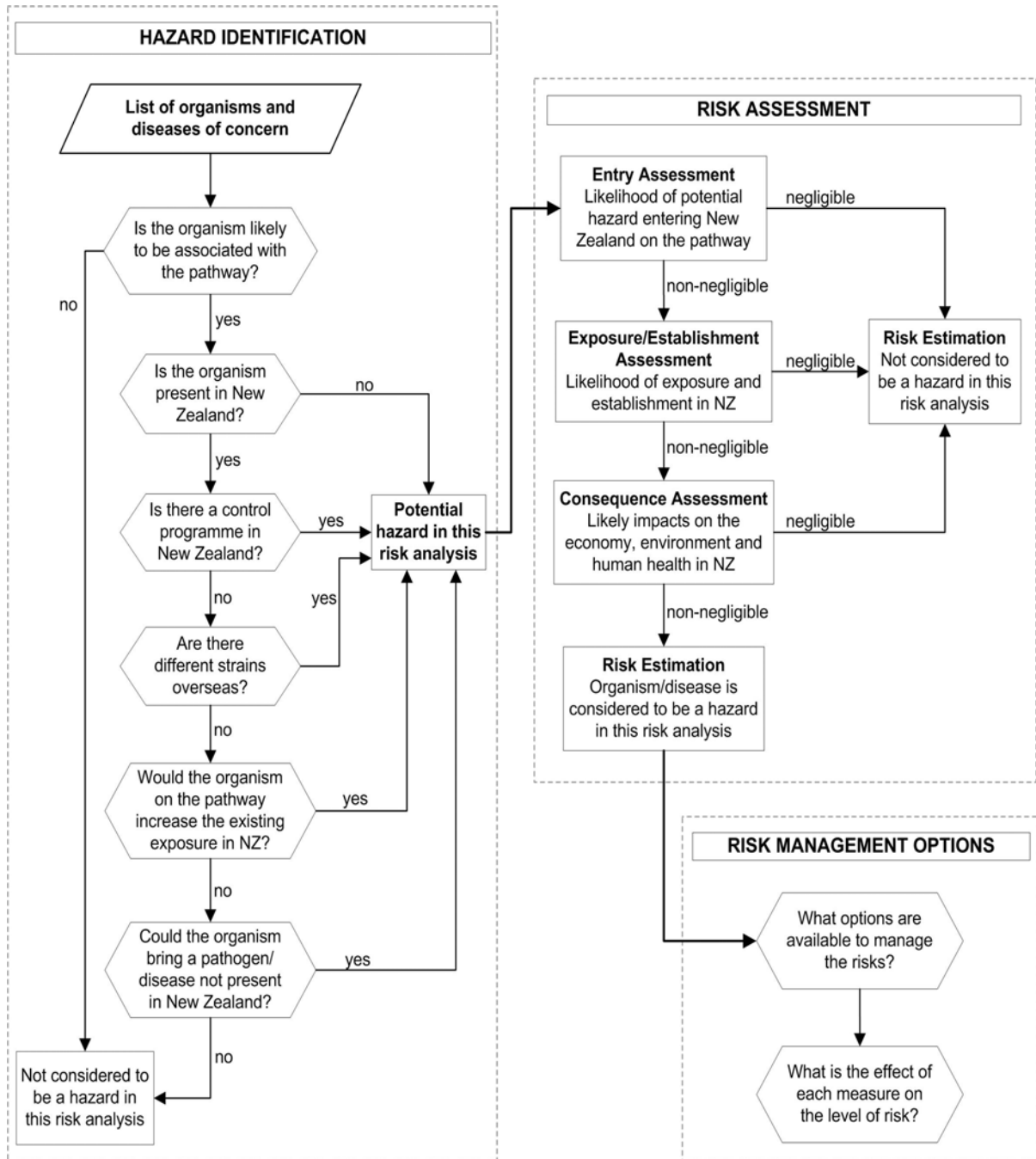
The commodity considered in this risk analysis is white rhinoceroses (*Ceratotherium simum*) from Australia.

4. Risk Analysis Methodology

The methodology used in this risk analysis follows the MAF Biosecurity New Zealand risk analysis procedures (Biosecurity New Zealand 2006). These procedures combine the guidelines in the *Terrestrial Animal Health Code* (hereafter referred to as the *Code*) of the

World Organisation for Animal Health and International Plant Protection Convention guidelines. The procedures provide a framework which adheres to the requirements set out under the World Trade Organisation Agreement on the application of Sanitary and Phytosanitary measures, 1995 and of the Biosecurity Act, 1993.

Figure 1. The risk analysis process.



4.1. PRELIMINARY HAZARD LIST

The process of hazard identification begins with the collation of a list of organisms that might be associated with rhinoceroses. The diseases of interest are those that could be

transmitted by rhinos and that could infect domestic, feral or wild animals, or humans in New Zealand. In this case an initial list was made of all organisms that may infect rhinoceros mentioned in the following sources:

- Rhinocerotidae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* 2003.
- AQIS Import Risk Analysis: Importation of Rhinoceros from South Africa 1999.
- MAF Import Health Standard for Rhinoceros from South Africa 1999.
- OIE 2008 listings of exotic diseases present in Australia.
- Internet database search for diseases reported in rhinoceroses, and for those diseases presence in Australia.

The organisms of potential concern associated with rhinos that were identified in these sources are listed in Table 1.

Many of the papers cited in this analysis refer to disease recorded in black rhino species (*Diceros bicornis*). Although the detailed reasons are incompletely understood, it should be noted that black rhinos appear to be more susceptible to a variety of diseases in captivity (Portas 2009). White rhinos have a lower overall prevalence of disease, and diseases of white rhinos more closely reflect those expected in related domestic species, such as the horse (Miller 2003).

Table 1. Organisms of Potential Concern

ORGANISM	NEW ZEALAND STATUS♣	AUSTRALIA STATUS	PRELIMINARY HAZARD
VIRUSES			
Akabane (Simbu group) viruses (ss)	Exotic	Endemic	Yes
Bluetongue virus (ss)	Exotic	Endemic	Yes
Bovine herpes virus -1 (IBR/IPV) (ss)	BHV-1.2b endemic. BHV-1.1, 1.2a, and 5 exotic	Endemic	Yes
Cowpox virus	Endemic	Endemic	No
Crimean Congo haemorrhagic fever virus (ss)	Exotic	Exotic	No
Rabies virus	Exotic	Exotic	No
Rift Valley fever virus (ss)	Exotic	Exotic	No
Ross River Fever virus (ss)	Exotic	Endemic	Yes
West Nile disease virus	Exotic	Exotic	No
African Horse Sickness (ss)	Exotic	Exotic	No
Epizootic Haemorrhagic disease of deer (ss)	Exotic	Endemic	Yes
Wesselsbron Disease (ss)	Exotic	Exotic	No
Parainfluenza type 3 (ss)	Endemic	Endemic	No
BACTERIA			
Anthrax <i>Bacillus anthracis</i>	Exotic	Endemic	Yes
<i>Campylobacter coli</i>	Endemic	Endemic	No
<i>Clostridium</i> spp.	Endemic	Endemic	No
<i>Sordelli, perfringens</i>			
<i>Escherichia coli</i>	Endemic	Endemic	No

<i>Leptospira</i> spp. (ss, 4 serovars)	6 serovars endemic	22 serovars endemic	Yes
<i>Mycobacterium bovis</i>	Endemic/ eradication programme	Exotic	No
<i>Mycobacterium tuberculosis</i>	Endemic	Endemic	No
<i>Mycobacterium paratuberculosis</i>	Endemic	Endemic	No
<i>Pseudomonas</i> spp. <i>pyocyanea</i>	Endemic	Endemic	No
<i>Salmonella</i> spp.	Some exotic	Some exotic	Yes
<i>Streptococcus</i> spp.	Endemic	Endemic	No
<i>Yersinia</i> spp. <i>pseudotuberculosis</i>	Endemic	Endemic	No
PROTOZOA			
<i>Babesia</i> spp.	Exotic	Endemic	Yes
<i>Cryptosporidium parvum</i>	Endemic	Endemic	No
<i>Neospora caninum</i>	Endemic	Endemic	No
<i>Theileria</i> spp. <i>parva, annulata, (bicornis</i> also in black rhino)	Exotic (non path endemic)	Exotic	No
<i>Trypanosoma</i> spp. (tsetse fly-borne) <i>evansi</i>	Exotic	Exotic	No
<i>Entamoeba</i> spp.	Endemic	Endemic	No
RICKETTSIAS AND CHLAMYDIAS			
<i>Ehrlichia ruminantium</i> (ss) Heartwater	Exotic	Exotic	No
PARASITES			
Ticks	9 spp. endemic (8 avian)	75 spp. endemic	Yes
Screwworm <i>Chrysomia bezziana</i>	Exotic	Exotic	No
Nematodes	Some exotic	Some exotic	Yes
Trematodes <i>Fasciola gigantica</i>	Exotic	Exotic	No
Botfly (stomach) <i>Gyrostigma rhinocerontis,</i> <i>Gyrostigma pavesii</i>	Exotic	Exotic	No
MISCELLANEOUS			
weeds and weed seeds	Some exotic	Some exotic	Yes
organisms causing superficial opportunistic skin infections: fungi, bacteria, yeasts.	Some exotic	Some exotic	No reports of exotic organisms causing skin infections in rhinos from Aus were found.

♣ The terms “exotic” and “endemic” are used in the context of MAF’s unwanted organisms policy (see: Biosecurity magazine, issue 6, 15/9/88, p 4) and the MAF unwanted organisms register (See: <http://www.biosecurity.govt.nz/pests/registers/uor>).
(ss) = serosurvey only: antibodies detected, but no organism isolated, or active infection reported.

The **preliminary hazard list** for the commodity is therefore as follows:

4.1.1. Viruses

Akabane disease (Simbu group) viruses
Bluetongue virus
Bovine herpes virus types 1.1 and 1.2a
Epizootic Haemorrhagic disease of deer
Ross River Fever Virus

4.1.2. Bacteria

Bacillus anthracis
Leptospira spp.
Salmonella spp.

4.1.3. Protozoa

Babesia spp.

4.1.4. Internal and external parasites

Ticks
Nematodes
Cestodes

4.1.5. Miscellaneous

Weeds and weed seeds

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

4.2. HAZARD IDENTIFICATION

Each organism identified as a preliminary hazard (those marked “yes” in column 4 of Table 1) is subjected to hazard identification.

Hazard identification begins with a discussion on the relevant aspects of the epidemiology for that organism, as far as it is relevant to germplasm, in particular:

1. Whether the imported commodity could act as a vehicle for the introduction of the organism.
2. The occurrence of the organism in countries of relevance in this risk analysis.
3. If an organism is present in New Zealand whether:
 - i. it is "under official control" in a pest management strategy as defined in the Biosecurity Act; or
 - ii. whether more virulent strains are known to exist in other countries?

Organisms that are present in New Zealand are by definition not potential hazards unless either;

- i) there is evidence that strains with higher pathogenicity than the endemic strains are likely to be present in the commodity to be imported, or
- ii) the organism is under official control in New Zealand by means of a Pest Management Strategy under the Biosecurity Act (1993).

If the hazard identification process identifies the organism as a potential hazard it is subjected to risk assessment.

4.3. RISK ASSESSMENT

Risk assessment consists of:

Entry assessment: The likelihood of a pathogenic organism being imported with the animal.

- a) *Exposure assessment*: The likelihood of susceptible species or environments in New Zealand being exposed to the potential hazard.
- b) *Consequence assessment*: The consequences of entry, establishment or spread of an imported organism.
- c) *Risk estimation*: An estimation of the risk posed by the imported commodity based on the entry, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is a potential threat and risk management measures are justified to effectively manage the risk.

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

4.4. RISK MANAGEMENT

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

As obliged under Article 3.1 of the WTO Agreement on the Application of 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).

4.5. 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.

4.6. COUNTRY FREEDOM

Several important diseases that may infect rhinos have not been included in this risk analysis because they are not present in Australia. Further assessment may be required for these diseases if Australia's country freedom status changes.

African Horse Sickness

Crimean Congo haemorrhagic fever virus

Bovine viral diarrhoea virus type 2

Old and New World screwworm

Rabies virus

Rift Valley fever virus

West Nile disease virus

Wesselsbron Disease

Ehrlichia ruminantium (Heartwater)

Mycobacterium bovis (Bovine Tuberculosis)

Theileria annulata and T. parva

Tsetse fly transmitted *Trypanosoma* spp.

Trypanosoma evansi (surra)

Dermacentor and Hyalomma tick species

For importation to be considered from other countries in the future, risk assessments for the relevant diseases from this list may need to be conducted.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

AQIS (1999) Import Risk Analysis: Importation of Rhinoceros from South Africa 1999.
http://www.daff.gov.au/_data/assets/word_doc/0018/17082/99-049a.doc

Biosecurity New Zealand (2006) Risk Analysis Procedures version 1.

MAF (1999) Import Health Standard for Rhinoceros from South Africa 1999
<http://www.biosecurity.govt.nz/imports/animals/standards/zoorhiic.saf.htm>

Miller RE (2003) Rhinocoridae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* Saunders Elsevier Science, USA, 558-569 ISBN 0-7216-9499-3.

Portas T (2009) Australia Zoo, Veterinary Services Manager, draft review to Somervell R *pers comm.*

OIE (2008) Country Animal Health Situation
http://www.oie.int/wahis/public.php?page=country_status&year=2008*

5. Akabane and other Simbu Group Viruses

5.1. HAZARD IDENTIFICATION

5.1.1. Aetiological agent

Family: *Bunyaviridae*; Genus: *Bunyavirus*, Serogroup Simbu.

Akabane disease virus and related viruses belong to a group known collectively as Simbu viruses. The group includes viruses such as *Aino*, *Tinaroo*, *Peaton* and *Cache Valley* viruses that cause similar syndromes.

5.1.2. OIE list

Not listed.

5.1.3. New Zealand status

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

5.1.4. Epidemiology

Akabane virus is common in many tropical and subtropical areas between about 35° North and 35° South latitude (Merck 2008). Viruses in the Simbu-group occur endemically in large areas of Africa, Asia, the Middle East and Australia (St George & Kirkland 2004).

Akabane and related viruses have been isolated from midges (*Culicoides* spp.) and mosquitoes. *Culicoides* spp. are assumed to be the vectors of these viruses (St George & Kirkland 2004). These viruses cause abortion, premature birth and severe congenital abnormalities including hydroencephaly and arthrogryposis in cattle, sheep and goats.

The main vector species for Akabane virus in Australia is *Culicoides brevitarsis*. This species is distributed across northern Australia and down the east coast as far as central New South Wales. Akabane virus infection is endemic in this area. Outbreaks of Akabane disease have occurred at irregular intervals in south-eastern New South Wales, at the fringe of the usual distribution range of *C. brevitarsis*. Such outbreaks occurred in 1951, 1955, 1960, 1964, 1968, 1974 and 1983. Other smaller outbreaks have also been observed in the New England tablelands and Hunter River valley areas of NSW (AHA 2005).

In endemic areas, herbivores are bitten by the vectors, become infected at an early age, and will develop a long lasting immunity by the time of breeding; thus congenital abnormalities are seldom seen, but seroconversion is common. However, under favorable environmental conditions such as an extended humid summer, the vector (and hence the virus) may spread beyond its usual range into new areas, and outbreaks of congenital infection may be expected in naïve populations. These outbreaks usually occur at the northern or southern limits of the vector distribution or in areas of higher altitude (Merck 2008).

The time from infection to viraemia for Akabane virus is short, and the viraemic period lasts only 3-4 days (St George & Kirkland 2004). In cattle maximal damage occurs when infection takes place at about the 12th to 16th week of gestation (St George & 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, but calves born alive are not contagious and will not infect vectors.

A high prevalence and wide distribution of antibody titres against Akabane virus has been reported from free-ranging black and white rhinoceroses in Africa. It was postulated that this suggests virus replication and that these rhino species might be susceptible to infection (Barnard & Botha 1997, Fischer-Tenhagen et al 2000). No disease has been linked to the infection of wildlife in Africa, but most new-born animals with congenital defects would be eaten by predators and scavengers (St George & Kirkland 2004). There have not been reports of abortion, premature birth or severe congenital abnormalities in captive-born rhino calves.

5.1.5. Hazard identification conclusion

As there is a high prevalence of seroconversion in rhinos, and the disease is endemic in Australia, Akabane and other Simbu group viruses are classified as potential hazards in the commodity.

5.2. RISK ASSESSMENT

5.2.1. Entry assessment

These viruses could only be introduced into New Zealand by animals that have been infected within a week of arrival. Therefore the likelihood of introducing Akabane virus is very low.

5.2.2. Exposure assessment

A viraemic animal introduced into New Zealand would not be infectious. These viruses could only be transmitted to other animals in New Zealand by competent insect vectors. Annual surveys reported in the MAF publication *Surveillance* have demonstrated that *Culicoides* spp. are not present in New Zealand.

A typical report shows that no *Culicoides* spp. were found in 15,000 insects trapped and that serological conversion to arboviruses did not occur in sentinel cattle (Motha et al 1997). Since *Culicoides* spp. are the main vectors of the disease it is highly unlikely that New Zealand ruminants would be exposed to the virus.

Since Akabane disease is arthropod-borne, and the vector *C.brevitarsis* is not present in New Zealand, the exposure assessment is considered to be negligible.

5.2.3. Risk estimation

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

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

AHA (2005) Animal Health Australia Akabane Australian Situation
<http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/aka.cfm>*

Barnard BJH, Botha MJ (1997) Antibodies against some viruses of domestic animals in southern African wild animals. *Onderstepoort Journal of Veterinary Research*, 64, 95-110.

Fischer-Tenhagen C, Hamblin C, Quandt S, Frolich K (2000). Serosurvey for selected infectious disease agents in free-ranging black and white rhinoceros in Africa. *Journal of Wildlife Diseases*, 36(2), 316-323.

Merck (2008) Akabane virus infection *The Merck Veterinary Manual* 9th Ed available at:
<http://merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/50804.htm>*

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

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*. Oxford University Press, Oxford, 1029-36.

6. Orbiviruses: Bluetongue and Epizootic haemorrhagic disease of deer

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family: *Reoviridae* Genus: *Orbivirus* Species: *Bluetongue virus* (BT), *Epizootic Haemorrhagic Disease virus* (EHD) There are 24 known serotypes of BT and 10 known serotypes of EHD.

6.1.2. OIE list

BT is listed, EHD is not.

6.1.3. New Zealand status

BT is listed on the Unwanted Organisms Register as an exotic, notifiable organism. EHD is listed on the Unwanted Organisms Register as an exotic organism.

6.1.4. Epidemiology

Bluetongue virus occurs in most tropical and sub-tropical countries. Eight of the 24 international serotypes of bluetongue virus, and several related orbiviruses, have been recorded in northern Australia, but many of the highly pathogenic strains of bluetongue virus encountered in Africa, North America and parts of Asia are exotic to Australia (Gard et al 1996).

Epizootic hemorrhagic disease occurs in North America, Australia, Asia and Africa, and seropositive animals have been found in South America (CFSPH 2006). Five of the 10 known serotypes of EHD virus have been recorded in northern Australia (Aradaib & Ali 2004).

BT and EHD are arboviral diseases of ruminants of variable clinical severity, characterised by inflammation of mucous membranes, widespread haemorrhages and oedema (Gard et al 1996). The BT virus causes disease mainly in sheep, occasionally in goats and rarely in cattle and deer. In most other species infections are subclinical (Verwoerd & Erasmus 2004). The EHD virus can infect most wild and domestic ruminants. Clinical signs are seen mainly in deer and rarely in cattle (CFSPH 2006).

BT and EHD viruses are transmitted by *Culicoides* spp. midges, and outbreaks of the disease usually occur in late summer to autumn when midges are most active. Of the 180-odd midge species in Australia, only 6 species of *Culicoides* in northern Australia have been shown to be capable of being infected by bluetongue virus and of playing a role in the ecology of the disease (Gard et al 1996).

The incubation period for BT in susceptible animals is generally 4–8 days, and for EHD in deer is 5 to 10 days (Gard et al 1996). For BT it is considered that the maximum duration of effective viraemia is normally about 50 days in cattle and 20 days in sheep, although most animals are infectious to vectors for a much shorter period (Gard et al 1996).

Apart from one clinical case in a sentinel sheep flock on a research station near Darwin in 1989, there has been no evidence of any clinical disease associated with bluetongue infection of any livestock species in the field in Australia (Gard et al 1996). There have been no clinical cases of EHD in any species in Australia. Seroconversion to EHD virus has been found in sentinel cattle, sheep and one deer in the last 5 years, and there has been occasional virus isolation but no clinical disease (Post DAFF 2008).

In free-ranging black and white African rhinoceros, specific and sometimes high BT antibody titres were recorded in 35-55% of sera tested using the competitive ELISA (Anderson and Rowe 1998, Fischer-Tenhagen et al 2000). Using the less sensitive AGID test, no antibodies were found in the sera of 20 white and 20 black rhinos (Barnard and Botha 1997). Antibody to EHD was found in 19% of rhino sera tested (Fischer-Tenhagen et al 2000).

The predominantly low titres probably reflect continual exposure to the agent, however the high titres ($\geq 1/60$), albeit in small numbers of rhino sera, are analogous to those recorded in post infected or convalescent equines with the closely related orbivirus, African horse sickness virus, and may suggest some virus replication (Fischer-Tenhagen et al 2000). No reports could be found of virus isolation from rhinos.

6.1.5. Hazard identification conclusion

In view of the above, bluetongue and epizootic haemorrhagic disease viruses are classified as potential hazards in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry Assessment

BT and EHD virus could only be introduced into New Zealand by animals that are in the incubation period or viraemic at the time of introduction. The imported rhinos would need to be from a Northern Australian zoo where the *Culicoides* vector is known to be present, and would need to be imported during the season they are known to be active: in summer and for a period of 60 days after the onset of winter (Verwoerd and Erasmus 2004). Therefore the likelihood of introducing an incubating or viraemic animal is low but non-negligible.

6.2.2. Exposure Assessment

A viraemic animal introduced into New Zealand would not be infectious. BT and EHD virus could only be transmitted to other animals in New Zealand by competent insect vectors. Annual surveys reported in the MAF publication *Surveillance* have demonstrated that *Culicoides* spp. are not present in New Zealand.

A typical report shows that no *Culicoides* spp. were found in 15,000 insects trapped and that serological conversion to arboviruses did not occur in sentinel cattle (Motha et al 1997). Since *Culicoides* spp. are the vectors of the disease it is unlikely that New Zealand ruminants would be exposed to the virus. To date, seroconversion to arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped.

In the absence of a competent vector in New Zealand, the exposure assessment is considered to be negligible.

6.2.3. Risk Estimation

Because the exposure assessment is negligible, the risk estimate for BT and EHD virus is negligible, and BT and EHD viruses 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. All weblinks were accessed in November 2008.

Anderson EC, Rowe LW (1998). The prevalence of antibody to the viruses of bovine virus diarrhoea, bovine herpes virus 1, rift valley fever, ephemeral fever and bluetongue and to *Leptospira* spp. in free-ranging wildlife in Zimbabwe. *Epidemiology and Infection* 121, 441-449.

Aradaib I, Ali, N (2004) Current status and future prospects of epizootic haemorrhagic disease of deer -a review. *Veterinarski Arhiv* 74, 63-83.

Barnard BJH, Botha MJ (1997) Antibodies against some viruses of domestic animals in southern African wild animals. *Onderstepoort Journal of Veterinary Research*, 64, 95-110.

CFSPH Center for Food Security and Public Health, Iowa State University (2006) Diseases Caused by the Epizootic Hemorrhagic Disease Virus Serogroup Factsheet available at:
http://www.cfsph.iastate.edu/Factsheets/pdfs/epizootic_hemorrhagic_disease.pdf*

Fischer-Tenhagen C, Hamblin C, Quandt S, Frolich K (2000). Serosurvey for selected infectious disease agents in free-ranging black and white rhinoceros in Africa. *Journal of Wildlife Diseases*, 36(2), 316-323.

Gard G, Kirkland P, Johnson S, Muller M, Curran J, Webster R, Richards B, St George T (1996) Disease strategy for Bluetongue In *AUSVETPLAN* (2nd Ed) ISBN 0 642 24506 1

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

Post L (2008) Principal Veterinary Officer, Department of Agriculture, Fisheries and Forestry (DAFF) email to Somervell R *pers comm*.

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

7. Ross River Fever Virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: Togaviridae Genus: Alphavirus Species: Ross River virus (RRV).

7.1.2. OIE list

Not listed.

7.1.3. New Zealand status

Exotic. Serological studies have shown that RRV has probably been introduced into New Zealand by viraemic travellers on many occasions. Although some local mosquitoes have shown the ability to transmit the virus in the laboratory, there has been no evidence of establishment (Maguire 1994).

7.1.4. Epidemiology

RRV occurs throughout Australia, including Tasmania, and also in Papua New Guinea, the Solomon Islands, New Caledonia, Fiji, American Samoa and the Cook Islands (Aaskov & Dougherty 1994).

RRV is an arbovirus causing polyarthrits/arthritis in humans and laboratory mice. It is often accompanied by a maculopapular rash and low-grade fever. The incubation period is from 3-21 days. Symptoms usually resolve within a month, and there is no evidence that infection with RRV can lead to chronic disease (Harley et al 2001).

Serological evidence indicates RRV may also be associated with a condition in horses involving muscle and joint stiffness, limb oedema and nervous signs. Experimental inoculation of horses has only resulted in a very mild clinical syndrome (Kay & Aaskov 1989).

Positive antibody titres to RRV in the absence of clinical signs (subclinical infection) occurs in a broad range of marsupials, mammals and birds (Aaskov & Dougherty 1994). The role of horses in RRV transmission has been investigated through experimental inoculation of 11 horses. Viraemia in ten was low or undetectable, and in one was relatively high and lasted 5 days (Kay & Aaskov 1989).

A wide variety of mosquito species are capable of transmitting RRV, at least 13 different species across six genera in Australia, although efficiency varies considerably. The most common vector species in Australia *Culex annulirostris* and *Aedes vigilax* don't occur in New Zealand, but several species of the same genera do. One species in New Zealand, *Aedes australis* has been shown to be capable of transmitting the virus under laboratory conditions (Maguire 1994).

There are no reports of clinical signs associated with RRV infection or virus isolation from rhino, but white rhino serosurveyed in Australia had positive antibody titres for the virus. It was presumed this simply represented past exposure to the virus but it is unknown how long rhino might remain viraemic, or if they can remain persistently infected (Portas 2009).

A variety of serological tests are used to diagnose RRV infection in people including: HI, ELISA, CF, and VNT. Virus isolation is possible, but considered to be too slow and expensive for routine diagnostic use (Aaskov & Dougherty 1994). The VNT has been used but not validated in rhino, and a PCR is now available in Australia (Portas 2009).

7.1.5. Hazard identification conclusion

Because seropositive rhino have been detected in Australia, and New Zealand has potentially competent mosquito vectors, Ross River Fever Virus is classified as a potential hazard in the commodity.

7.2. RISK ASSESSMENT

7.2.1. Entry Assessment

RRV could only be introduced into New Zealand by animals that are in the incubation period or viraemic at the time of introduction. Because the time course of infection is unknown in rhino, the likelihood of introducing an incubating or viraemic animal is low but non-negligible.

7.2.2. Exposure Assessment

A viraemic animal introduced into New Zealand would not be directly infectious. RRV virus could only be transmitted to other animals or humans in New Zealand by competent insect vectors. One mosquito species in New Zealand has been shown to be capable of transmitting the virus under laboratory conditions (Maguire 1994). The potential for other competent vector species in New Zealand is unknown.

The likelihood of a competent vector mosquito biting a viraemic imported rhino and then transmitting RRV to susceptible animals or humans is very low but non-negligible.

7.2.3. Consequence assessment

The significance of importing rhino infected with RRV would probably be related only to its zoonotic potential. The number of potentially infected humans from Australia far exceeds the number of rhino imported annually.

Serological studies have shown that RRV has probably been introduced into New Zealand by viraemic travellers on many occasions. There has been no evidence of local spread or establishment of virus from the imported cases (Maguire 1994).

There are currently no risk management measures applied to importation of horses potentially infected with RRV from Australia, and no incursions have been reported.

Therefore, the likelihood of significant consequences is negligible.

7.2.4. Risk Estimation

Because the consequence assessment is negligible, the risk estimate for RRV is negligible, and the virus 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. All weblinks were accessed in November 2008.

Aaskov JG, Doherty RL (1994) Arboviral Zoonoses of Australasia. In: Beran GW (ed) *Handbook of Zoonoses, Second Edition, Section B; Viral*. CRC Press 289-304. .

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

Kay B, Aaskov J. Ross River Virus (Epidemic Polyarthrits). In: Monath TP (ed) *The Arboviruses: Epidemiology and Ecology. Volume IV*. CRC Press 93-112.

Maguire T (1994). Do Ross River and Dengue viruses pose a threat to New Zealand? *New Zealand Medical Journal*, 24(4),18-9.

Portas T (2009) Australia Zoo, Veterinary Services Manager, draft review to Somervell R *pers comm*.

8. Bovine Herpes Viruses

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agents

Family: Herpesviridae Subfamily: Alphaherpesvirinae Genus: Varicellovirus

Bovine herpesvirus 1 (BHV-1) is associated with several diseases in cattle: infectious bovine rhinotracheitis (IBR), infectious pustular vulvovaginitis (IPV), balanoposthitis, conjunctivitis, abortion, encephalomyelitis, and mastitis. Only a single serotype of BHV-1 is recognized; however, three subtypes of BHV-1 have been described: BHV-1.1 respiratory subtype, BHV-1.2 genital subtype, and BHV-1.3 encephalitic subtype. BHV-1.3 has been reclassified as a distinct herpesvirus designated BHV-5 (Merck 2008).

Subtype 1.2 strains can be further classified as BHV-1.2a and BHV-1.2b strains. Some subtype 1.1 and 1.2a strains are abortifacient, as shown by association with clinical cases of abortion and by experimental infection of pregnant heifers (Miller et al 1991). Subtype 1.2b strains are associated with respiratory and genital infections but not with abortions (Miller et al 1991).

Table 3. Bovine herpesviruses

Type	Syndrome			
	IBR	IPV/IPB	Abortion	Encephalitis
BHV 1.1	+	-	+	-
BHV 1.2a	+	+	+	-
BHV 1.2b	+	+	-	-
BHV5	-	-	-	+

8.1.2. OIE list

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

8.1.3. New Zealand status

Only BHV1.2b has been isolated in New Zealand (Wang et al 2006). Abortions have not been seen in New Zealand (Fairley 1996; Horner 1990). An attempt to cause abortion by experimental infection with the New Zealand strain of the virus was unsuccessful (Durham et al 1975). However, at the present time identification of abortifacient strains of the virus from either subtype 1 or 2 strains would require experimental infection of pregnant cows. A more pragmatic approach is to regard BHV1.1 and BHV 1.2a as exotic organisms. Abortifacient strains are classified on the unwanted organisms register as unwanted notifiable organisms.

8.1.4. Epidemiology

IBR/IPV has a world-wide distribution. The virus is endemic in New Zealand and serological surveys have shown that it occurs very widely (Neilson and Grace 1988). Both the IBR and the IPV syndrome have been described. However, in the vast majority of

cases there are no or only mild clinical signs (Vermunt and Parkinson 2000). Transmission can occur in the absence of visible lesions.

After an incubation period of 2-4 days, acute infections are of short duration and virus is excreted in nasal secretions for up to 14 days after infection (Kramps 2008). Viraemia is hard to detect (Babuik et al 2004). Virus spreads to the conjunctiva and trigeminal ganglion by neuronal axonal transport (Kramps 2008). Many animals become chronically infected latent carriers of the virus in their trigeminal or sacral ganglia, and may excrete the virus periodically when they are stressed (Babuik et al 2004). Semen may be infected with virus and insemination with such semen causes infection in recipient females (van Oirschot 1995).

BHV-5 associated with encephalitis has been described in Australia (Brake and Studdert 1985) but not in New Zealand.

Serosurveys of free-ranging rhinos in Africa have found no (Anderson and Rowe 1998) or low 3-6% prevalence with low titres of antibody to BHV-1 (Barnard and Botha 1997, Fischer-Tenhagen et al 2000). The low prevalence and low titres probably reflect cross reactions with a rhino species specific herpes virus rather than a susceptibility to BHV-1 infection (Fischer-Tenhagen et al 2000).

Serological tests are not specific for sub-types of herpes viruses and it is not possible to distinguish between the antibodies induced by BHV1.2a, BH1.1 or BHV5 strains from other strains of herpes virus in rhino species. In the absence of virus isolation susceptibility of rhinoceroses to infection with herpes viruses remains unclear.

8.1.5. Hazard identification conclusion

Non- abortifacient strains of BHV-1 are present in New Zealand. Clinical cases, abortions, or virus isolation of any bovine herpes virus has not been reported in rhinos.

The low prevalence and low titres of antibody to BHV-1 in wild rhinos are probably due to cross reactions. Therefore, bovine herpes viruses are not classified as potential hazards in the commodity.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

Anderson EC, Rowe LW (1998). The prevalence of antibody to the viruses of bovine virus diarrhoea, bovine herpes virus 1, rift valley fever, ephemeral fever and bluetongue and to *Leptospira* spp. in free-ranging wildlife in Zimbabwe. *Epidemiology and Infection* 121, 441-449.

Babuik TA, Van Drunen Littel-van den Hurk S, Tikoo SK (2004). Infectious bovine rhinotracheitis / pustular vulvovaginitis and infectious pustular balanoposthitis. In: Coetzer JAW, Tustin RC, (eds). *Infectious Diseases of livestock*. Oxford University Press, Oxford, 875-86.

Barnard BJH, Botha MJ (1997). Antibodies against some viruses of domestic animals in southern African wild animals. *Onderstepoort Journal of Veterinary Research*, 64, 95-110.

- Brake F, Studdert MJ (1985).** Molecular epidemiology and pathogenesis of ruminant herpesviruses including bovine, buffalo and caprine herpesviruses 1 and bovine encephalitis herpesvirus. *Australian Veterinary Journal*, 62(10), 331-4.
- Durham PJK, Forbes-Faulkner JC, Poole WSH (1975).** Infectious bovine rhinotracheitis: Experimental attempts at inducing bovine abortion with a New Zealand isolate. *New Zealand Veterinary Journal*, 23, 93-4.
- Fairley RA (1996).** Respiratory diseases of New Zealand cattle. *Surveillance*, 23(4), 15-6.
- Fischer-Tenhagen C, Hamblin C, Quandt S, Frolich K (2000).** Serosurvey for selected infectious disease agents in free-ranging black and white rhinoceros in Africa. *Journal of Wildlife Diseases*, 36(2), 316-323.
- Horner GW (1990).** Infectious bovine rhinotracheitis in New Zealand. *Surveillance*, 17(2), 25-6.
- Kramps JA (2008).** Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. OIE, Paris, 752-767.
- Miller JM, Whetstone CA, Van der Maaten MJ (1991).** Abortifacient property of bovine herpesvirus type 1 isolates that represent three subtypes determined by restriction endonuclease analysis of viral DNA. *American Journal of Veterinary Research*, 52(2), 458-61.
- Neilson FJA, Grace PJ (1988).** Infectious bovine rhinotracheitis is widespread in New Zealand. *Surveillance*, 15(2), 29.
- Merck (2008).** Bovine Herpesvirus 1 (Infectious bovine rhinotracheitis virus, Infectious pustular vulvovaginitis, and associated diseases) Chapter online in *The Merck Veterinary Manual* 9th Ed. Available at: <http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/121212.htm&word=bovine%2cherpes%2cvirus>
- van Oirschot JT (1995).** Bovine herpesvirus in semen of bulls and the risk of transmission: a brief review. *Veterinary Quarterly*, 17(1), 29-33.*
- Vermunt JJ, Parkinson TJ (2000).** Infectious diseases of cattle in New Zealand. Part 2- adult animals. *Surveillance*, 27(3), 3-9.
- Wang J, Horner GW, O'Keefe JS (2006).** Genetic characteristics of bovine herpesvirus 1 in New Zealand. *New Zealand Veterinary Journal*, 54(2), 61-66.

9. Anthrax (*Bacillus anthracis*)

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.

9.1.4. Epidemiology

Anthrax is a bacterial disease of most warm-blooded vertebrates including humans. New Zealand has been free from the disease for over 50 years (Gill 1992).

The Northern Territory, South Australia and Tasmania are considered to be free of anthrax. Anthrax is uncommon in Australia and clinical cases of the disease are seen only sporadically, with cases occurring on about 6 to 12 premises per year, with usually only a small number of cases on each affected farm (eg: 1-3 cattle or 5-20 sheep on average). Cases in central New South Wales tend to be in sheep and occur predominantly between October and March. In northern and north-eastern Victoria, virtually all incidents and outbreaks occur in cattle, with cases occurring relatively evenly throughout the year. Western Australia has only recorded cases in a localised, isolated area in the southwest of the state. The remainder of Western Australia is considered to be free of anthrax. Two properties in southern Queensland linked by cattle movements suffered cases in 2002. The remainder of Queensland is still considered to be free of anthrax (AHA 2005).

B. anthracis is a spore forming, gram positive aerobic rod that can survive in the spore state in suitable soils for many decades. The pathogen only multiplies in animals and if infection results in bloody exudates from nose, mouth or anus; or if a carcass is opened it sporulates resulting in contamination of soil and the environment. Sporulation requires oxygen, so in unopened carcasses the organism does not sporulate and is destroyed by putrefaction (De Vos & Turnbull 2004). The disease is not directly transmissible from animal to animal and infection is believed to be associated with ingestion of 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. Infection through skin wounds and abrasions may also occur and is a common route of infection for humans. 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 (De Vos & Turnbull 2004).

The incubation period probably varies from 1-14 days and in the peracute form in susceptible species the course of the disease is only a few hours (De Vos & Turnbull 2004). In the acute form of the disease, death usually occurs within 48 hours (Blood & Radostits 1989). Sub-acute and chronic forms of the disease occur in less susceptible animals such as pigs and carnivores (De Vos & Turnbull 2004).

Confirmed infection of rhinos has only been recorded in Indian (*Rhinoceros unicornis*) and black (*Diceros bicornis*) rhinoceros species (Gates et al 2001). The death of one white rhino in Kenya was strongly suspected but not confirmed to be caused by anthrax (Promed 2001).

Although antibiotics may be effective if started early, the course of disease is usually rapid and infections are often fatal (CFSPH 2007). Efficient live spore vaccines are available for control of the disease and have been used for over 3 decades in black rhinoceros in Namibia. Effectiveness was evaluated by inoculating mice with vaccinated rhino sera. Protection was conferred on 80-100% of mice by sera from three of four vaccinated rhinos, but no protection was conferred by serum from the fourth rhino despite having antibody profiles in line with other vaccinated animals (Turnbull et al 2004).

9.1.5. Hazard identification conclusion

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

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Anthrax could be introduced if rhinos are imported from zoos in infected zones of Australia that are in the incubation period of the disease, up to 14 days. Bloody exudates associated with disease, and death after importation followed by the carcass being opened, would result in the organism contaminating the environment (particularly soil) with spores that can survive for many years. Therefore, the likelihood of entry is considered low but non-negligible.

9.2.2. Exposure assessment

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. Although this would be within a zoo containment facility, any animals accessing the area in future (including wild rodents which can leave the facility) could be infected. Therefore the likelihood of exposure is considered to be non-negligible.

9.2.3. Consequence assessment

If the organism was introduced into the environment, animals that come into contact with and ingest infected soil or water could become infected and again contaminate the environment during the course of disease or after death. The disease could thus become established and lead to the deaths of animals and the need for vaccination to control the disease.

Since anthrax is a zoonotic organism, if 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.

In view of the above, the consequences of introducing *B. anthracis* are considered to be non-negligible.

9.2.4. Risk estimation

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

9.3. RISK MANAGEMENT

9.3.1. Options

The OIE *Terrestrial Animal Health Code* states that “there is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs” (OIE 2008). Since the incubation period is up to 14 days, the disease generally runs a short course, and there are not long term carriers of the disease, pre-export isolation is an efficient method to prevent introducing the disease.

A pre-export isolation period of 20 days as recommended in the *Code* could be imposed on animals to be imported. In addition, since effective vaccines are available all animals could be vaccinated before shipment.

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

- Rhinos could be certified as being born or permanently resident in establishments located in anthrax free zones of Australia.
- Rhinos could be held in isolation for at least the 20 days prior to shipment in an establishment where no case of anthrax has ever occurred.
- Rhinos could be vaccinated against anthrax, not less than 20 days and not more than 6 months prior to shipment.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

AHA Animal Health Australia (2005). Disease strategy for Anthrax version 3.2 In: *AUSVETPLAN* (3rd edition) ISBN 0 642 245061

Blood DC, Radostits OM (1989). Anthrax. *Veterinary Medicine*. Balliere Tindall, London, 592-6.

CFSPH Center for Food Security and Public Health, Iowa State University (2007) Anthrax Factsheet Available at: <http://www.cfsph.iastate.edu/Factsheets/pdfs/anthrax.pdf>*

De Vos V, Turnbull PCB (2004). Anthrax. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Oxford University Press, Oxford, 1788-818.

Gates C, Elkin B, Dragon D (2001) Anthrax. In: Williams E S, Barker I K (eds) *Infectious Diseases of Wild Mammals (3rd Edition)* Iowa State University Press, USA, 396-412

Gill J (1992). Anthrax - still history after all these years. *Surveillance*, 20(121-2).

OIE (2008). Anthrax. Chapter 8.1. In: OIE *Terrestrial Animal Health Code* Available at:
http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.1.htm

ProMED mail (2001) Anthrax, cattle rhinoceros?-Kenya archive no. 20010608.1122
<http://www.promedmail.org>*

Turnbull PCB, Tindall BW, Coetzee JD, Conradie CM, Bull RL, Lindeque PM, Huebschle OJB (2004) Vaccine-induced protection against anthrax in cheetah (*Acinonyx jubatus*) and black rhinoceros (*Diceros bicornis*) *Vaccine* 22(25-26) 3340-3347.

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.*

10. Salmonellosis (*Salmonella* spp.)

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2004). Most of the isolates that cause disease in humans and other mammals belong to the species enterica and the subspecies enterica and if correct naming conventions are used, the names such as Dublin and Typhimurium, which do not have species status, should not be italicised. The correct name for the serovar typhimurium is *Salmonella enterica* subsp. *enterica* serovar Typhimurium. However, in the following discussion for the sake of simplicity names are italicised and abbreviated as though the serovar had species status e.g. *Salmonella typhimurium*.

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

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

10.1.2. OIE list

General salmonellosis is not a listed disease in the OIE *Terrestrial Animal Health Code*. However, *Salmonella gallinarum*, *pullorum*, *enteritidis* and *typhimurium* in poultry are listed, and in the OIE *Manual of Diagnostic Tests and Vaccines* salmonellosis is included in the section “Diseases not covered by List A and List B”.

10.1.3. New Zealand status

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

10.1.4. Epidemiology

Salmonellosis can be found worldwide but serovars vary in their distribution.

Information in this section relates mainly but not exclusively to *Salmonella typhimurium* and *S. dublin* which commonly infect livestock.

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

S. dublin has not been isolated in New Zealand animals. It was isolated from one human case in 2007 (ESR 2007). In Australia, *S. dublin* occurs most commonly in cattle but also in sheep.

Salmonella typhimurium is endemic in New Zealand in both animals and humans but DT104 has only been isolated from humans in very low numbers for many years (ESR). This suggests that *S typhimurium* DT104 has not become established in the New Zealand animal population. *S typhimurium* DT 104 also occurs in Australia at a low prevalence related mostly to imported cases in humans. It is postulated that the situation may be similar to NZ due to our shared geographically isolated island status (Helms et al 2005).

Salmonella spp. are mainly transmitted by the faecal-oral route. They are carried asymptotically in the intestines or gall bladder of many animals, and are continuously or intermittently shed in the faeces. They can also be carried latently in the mesenteric lymph nodes or tonsils; these bacteria are not shed, but can become reactivated after stress. Vertical transmission occurs in birds within eggs, and can also be transmitted *in utero* in mammals (CFSPH 2005).

Excreted organisms contaminate the environment and become a source of infection via fomites (Blood et al 1994). *Salmonella* spp. can survive for long periods in the environment, particularly where it is wet and warm. *S. typhimurium* and *S. dublin* have been found to survive for over a year in the environment (CFSPH 2005).

Factors such as infecting dose, the particular strain and species, and various stress factors influence the outcome of infection (Fenwick & Collett 2004). Young animals are more often affected by the disease than adults and may die after a short bacteraemia. The incubation period is variable but the organisms may be found in the bloodstream of newborn calves within 15 minutes of ingestion (Blood et al 1994). The intestine is initially infected and inflammation of the gut is the primary lesion. Initial infection may be followed by invasion of the gut and mesenteric lymph node followed by bacteraemia and dissemination to many organs. In the case of pregnant animals abortion due to *S. dublin* may occur. Animals that recover from *S. dublin* infections frequently become carriers and may remain carriers for life, shedding organisms sporadically in their faeces. Animals infected with *S. typhimurium* may be carriers of infection for 3-4 months.

For humans, most *Salmonella* infections are acquired by handling or consuming contaminated food products, particularly foods of animal origin. Infections also are acquired by direct and indirect contact with farm animals, reptiles, and occasionally pets.

Various species of salmonella have been isolated by faecal culture from both asymptomatic and clinically ill black and white rhinoceros, including *S. typhimurium* and *S. dublin* in captive black rhinos from the USA (Kenny 1999). *S. typhimurium* was found in 2 black rhino and was reported as 'the pathogenic strain found in 60-70% of salmonella positive equids'. *S. dublin* was also found in 2 black rhinos and was reported as 'the pathogenic strain in cattle' (Kenny 1999). A *Salmonella* herd prevalence of 2.4% positive cultures and 2.6% positive PCRs was reported in asymptomatic captive black rhinos (Miller et al 2008). The herd contained only 6 animals, but a total of 550 cultures and 464 PCRs were performed. None of the animals shed *S. typhimurium* or *S. dublin* during the 3 year study period. There were no reports found of these *Salmonella* species occurring in white rhinos, but potential infection with other *Salmonella* species exotic to New Zealand cannot be ruled out.

Carriers of infections can be detected by culturing faeces samples but because excretion is intermittent repeated sampling and culture is necessary (Davies 2004). Serology may be useful

but is best applied on a herd basis (Davies 2004; Veling et al 2002). It has also been used for the identification of individual bovine carriers but its validity is influenced by age of the animal and is most valid for animals aged over 100 days of age (Nielsen et al 2004a/b). No practical method exists for detecting individual carrier animals (Hansen et al 2006).

Treatment of infected rhinos suffering enteritis and sepsis has been attempted with parenteral antibiotics and fluids but has not often been successful (Miller 2003).

10.1.5. Hazard identification conclusion

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

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Animals infected with *Salmonella* spp. may carry the organism for long periods and excrete the organism intermittently in their faeces. Therefore the likelihood of entry of the agent/s into New Zealand is non-negligible.

10.2.2. Exposure assessment

The requirement that rhinos be kept in containment facilities will significantly limit the exposure of both people and other animals to any associated *Salmonella*. However, undetected carrier animals would be moved into groups of susceptible rhinos and would excrete the organism intermittently in their faeces. Therefore they would be likely to infect other rhinos, and zoo staff could be occupationally exposed. Enclosures are also often 'open range' and accessible by wildlife. Drainage run-off or waste material removed from enclosures is also likely to contribute to potential exposure.

The likelihood of exposure of New Zealand animals and humans to the organisms is therefore assessed to be low but non-negligible.

10.2.3. Consequence assessment

The introduction of rhinos with exotic salmonellae is likely to result in infection of animals in contact with them. These newly infected animals could become carriers and excretors of organisms and potentially infect other animals and people. The introduction and establishment of any new *Salmonella* spp. could result in spread of the organisms in New Zealand and the establishment of production limiting diseases of livestock.

The establishment of *S. typhimurium* DT104 in animal populations would constitute a source of infection and be of particular concern to human health because of its resistance to antibiotics (Hogue et al 1997). *S. dublin* is also a zoonotic organism that could cause disease in people.

There would be no particular consequences for the environment other than possibly causing sporadic cases of salmonellosis in wild or feral animals. An outbreak of a new phage type of *S typhimurium* (DT160) occurred in sparrows and in humans in 2001. Infection was associated with several hundred deaths in sparrows (Alley et al 2002). The

outbreak was self limiting and did not have any lasting effect on the sparrow population. However following this outbreak isolation of *S. typhimurium* DT160 from humans occurred frequently and it is now endemic in humans in New Zealand (ESR). *Salmonella* infections can establish in wild bird populations and be associated with sporadic mortalities (Pennycott 2001). *S typhimurium* DT 160 and DT195 have been isolated and caused clinical signs in silvereye, kaka, kakariki and hihi (Alley 2007). However, the effects that introducing new *Salmonella* spp. might have on native birds cannot be predicted.

In conclusion, there is a low likelihood that introduction of infected rhinos could lead to the establishment of new *Salmonella* spp. that have the potential to cause disease in humans and animals. Therefore the consequences are non-negligible.

10.2.4. Risk estimation

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

10.3. RISK MANAGEMENT

10.3.1. Options

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

Although animals to be imported must be certified as clinically healthy, and in particular would not show any signs of diarrhoea, carriers of *Salmonella* spp. are unlikely to show signs of infection. Animals could be held in pre-export quarantine and faecal samples could be cultured for *Salmonella* spp. However since carriers may excrete organism intermittently they would need to be cultured on more than one occasion.

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

- Since many *Salmonella* serovars occur in New Zealand and because the small numbers of imported zoo animals are not regarded as important in the epidemiology of *Salmonellosis*, clinically healthy rhinos could be imported without restrictions.
- Rhinos could be required to have originated from premises where outbreaks of salmonellosis due to *S dublin* or *S typhimurium* DT104 have not been confirmed by laboratory testing in the last 3 years.
- Rhinos could be held for at least 3 weeks in pre-export quarantine. Faecal samples from quarantined animals could be cultured on at least 2 occasions with an interval of at least 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 MAF.

Where pathogenic *Salmonella* spp. exotic to New Zealand are isolated, the animals could be considered ineligible for importation for the remainder of its life (unless the organism is no longer considered exotic to New Zealand). Where *Salmonella* spp. that are endemic to

New Zealand are isolated it could be at the discretion of the importer of the animals to decide whether to proceed with the importation.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

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.

Alley MR (2007). Personal communication by Email to H J Pharo.

Blood DC, Radostits OM, Gay CC (1994). Diseases caused by Salmonella spp. In: *Veterinary Medicine*. Balliere Tindall, London, 643-56.

CFSPH Center for Food Security and Public Health, Iowa State University (2005) Salmonellosis Factsheet http://www.cfsph.iastate.edu/Factsheets/pdfs/Nontyphoidal_Salmonellosis.pdf*

Davies R (2008). Salmonellosis. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. OIE, Paris, 1267-83.

Environmental and Science Research laboratory (ESR) Database of the enteric reference laboratory. http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php*

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

Hansen KR, Nielsen LR, Lind P (2006). Use of IgG avidity ELISA to differentiate acute from persistent infection with Salmonella Dublin in cattle. *Journal of Applied Microbiology* 100(1) 144-52.

Helms M, Ethelberg S, Molbak K, (2005). International *Salmonella typhimurium* DT104 infections, 1992-2001 *Emerging Infectious Diseases* 11(6) 859-867

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>.

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. *The Veterinary Record*, 150, 649-54.

Miller RE (2003). Rhinocerotidae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* Saunders Elsevier Science, USA, 558-569

Miller M, Schille B, Pancake C (2008). Salmonella surveillance in a herd of asymptomatic captive black rhinoceros (*Diceros bicornis*) using faecal culture and PCR. *Journal of Zoo and Wildlife Medicine* 39(1) 56-60.

Nielsen LR, Toft N, Ersboll AK (2004a). 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.

Nielsen LR, Toft N, Ersboll AK (2004b). 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.

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

Veling J, Barkema HW, van der Schans J, van Zijderveld F, Verhoeff J (2002). Herd-level diagnosis for Salmonella enterica subsp. enterica serovar Dublin infection in bovine dairy herds. *Preventive Veterinary Medicine*, 53(1-2), 31-42.*

11. Leptospirosis (*Leptospira* spp)

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Before 1989 in the accepted taxonomic scheme, all pathogenic serovars belonged to the species *Leptospira interrogans* which contained more than 200 serovars in 23 serogroups. More recently the genus has been re-organised and pathogenic leptospire are now identified in several species of *Leptospira* (CFSPH 2005). However for the purposes of this risk analysis, serovars are written as if they were single species e.g. *Leptospira hardjo*, *L. pomona* etc.

11.1.2. OIE list

Listed.

Since 2004 the *Code* has not had a chapter on leptospirosis, only a statement that it is “under study”.

11.1.3. New Zealand status

L. hardjo, *L. pomona*, *L. balcanica*, *L. copenhageni*, *L. ballum*, and *L. tarrasovi* have been isolated from animals in New Zealand (Midwinter, 1999). Single isolations of *L. australis* and *L. canicola* have been reported from humans (Thompson 1980; Cheresky et al 1993). In humans, serological diagnosis indicates that five of the species endemic in farm animals infect humans but *L. balcanica*, which is associated with possums, has not been diagnosed in humans (ESR 2003). A serosurvey of 8,730 dogs throughout New Zealand found only one weak reaction to *L. canicola*, and it is concluded that this serovar is not present (Hilbink et al 1992).

Other *Leptospira* spp. are classified by MAF as ‘other exotic organisms’

11.1.4. Epidemiology

Leptospirosis occurs world-wide but the endemic serotypes that occur in each country differ. It 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. *L. hardjo* for example infects most cattle in an endemic situation but only causes occasional cases of disease in cattle. However, it may be responsible for causing sporadic cases of disease in other species such as humans (accidental hosts). In maintenance hosts, *Leptospira* may localise in the kidneys and the animals may continue to excrete the organism in their urine for years. Cattle can remain carriers of *L. hardjo* for at least 450 days (Hunter 2004). In New Zealand the prevalence of the disease in humans is relatively high for a temperate climate country and *L. hardjo* accounts for nearly half the cases (Thornley et al 2002). *L. hardjo* is also the most common serovar in Australia. (VGHI 2008) Of 230 serovars identified, only 22 have been isolated in Australia (AQIS 2000).

Leptospirosis can be transmitted either directly between hosts or indirectly in the environment. Direct transmission occurs through contact with infected urine, venereal and placental transfer, bite wounds or ingestion of infected tissues. The organisms usually enter the body through mucous membranes or abraded skin (CFSPH 2005). Indirect transmission occurs through exposure to water, soil, food or bedding contaminated with infected urine. Diseased animals shed more organisms and are more important sources of infection than chronic carriers (Horsch 1989). In accidental hosts the incubation period may be from 2-16 days and is followed by a period of bacteraemia. A variety of signs may be shown by diseased animals including abortion, haemolytic anaemia, icterus, and nephritis.

Infection by *Leptospira* is common in a wide range of wild mammals, but disease has rarely been reported in free-ranging wildlife (Leighton and Kuiken 2001). Crowding of animals enhances direct spread of leptospirosis through increased contact with infective urine, so infection is more often reported in captive rhinos. Leptospirosis has been associated with cases of haemolytic anaemia and abortion in several species of captive rhino (Miller 2003). Antibody titres to *L. bratislava*, *L. copenhageni*, *L. grippityphosa* and *L. tarassovi* were detected in sera from asymptomatic free-ranging black and white African rhinos collected between 1987 and 1997 (Fischer-Tenhagen et al 2000). The prevalence estimates for positive microagglutination tests to the 4 serovars ranged from 3.3-8.5%. *L. grippityphosa* was isolated from 2 captive black rhinos in Pittsburgh Zoo with gastrointestinal signs (Neiffer et al 2001).

The disease can be diagnosed by the isolation of the organism, but because this is a slow process (taking up to 26 weeks dependent on serovar) it is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low titre indicating resolution or a chronic infection. The microscopic agglutination test is still the most commonly used test and can be used on a variety of animal species without modification. A number of variations of commercial ELISAs are also available but these generally lack serovar specificity (Bolin 2004).

Leptospirosis is seldom the cause of economically serious disease in animals and is mainly of concern because it is a zoonotic disease that occasionally causes serious disease in humans (Thornley et al 2002). *Leptospira* spp. are sensitive to several antibiotics (Murray & Hospenthal 2004).

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

11.1.5. Hazard identification conclusion

A range of serovars have been implicated in subclinical and clinical leptospiral infections in rhinos. *Leptospira* spp. other than the 6 endemic serovars are exotic, zoonotic organisms and are classified as potential hazards in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Clinical signs in rhinos vary from no noticeable disease to gastroenteritis or haemolytic anaemia. Acutely infected animals or chronic carriers may excrete the organism in urine. Therefore the likelihood of entry is non-negligible.

11.2.2. Exposure assessment

Infected rhinos are proportionally much less of a hazard than domestic livestock or pets as a human public health risk in New Zealand. However, carrier rhinos shed the organism in their urine and therefore have the potential to infect zoo staff and wildlife accessing their enclosures. Venereal transmission of the organism also occurs, so exposure of other rhinos through breeding programmes is also possible. Drainage run-off or contaminated waste material removed from enclosures would also contribute to potential exposure.

The likelihood of exposure of New Zealand animals and humans to the organisms is therefore assessed to be low but non-negligible.

11.2.3. Consequence assessment

Introduction of new serovars of *Leptospira* are unlikely to have a significant impact on the New Zealand animal population. Sporadic cases of disease may occur, but the economic consequences would be negligible.

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 *L. balcanica* is common in its maintenance host the brush tailed possum, but infections of humans have not occurred despite the close contact between possums and possum hunters (Anonymous 2004).

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

The establishment of new *Leptospira* serovars is likely to cause sporadic cases of disease in humans. Therefore, the consequences of establishment are considered to be low but non-negligible.

11.2.4. Risk estimation

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

11.3. RISK MANAGEMENT

11.3.1. Options

In its report in 2003, the OIE *Code* commission noted the ubiquity of the organism, the absence of any meaningful control programmes, and that there are no effective treatments; thus proposing deletion of the chapter in 2004 (OIE 2003).

Although animals to be imported must be certified as clinically healthy, carriers of *Leptospira* spp. are unlikely to show any signs of infection. Because of the occurrence of long term carriers of infection, quarantine alone is not an effective option.

Testing urine samples by culture or PCR while animals are held in pre-export quarantine is problematic because isolation of organisms is slow (may take up to 26 weeks dependent on serovar) or they may fail to grow altogether as the organism is relatively fastidious. Selection of primers for PCR that will recognize all serovars has not yet been achieved.

Studies of captive black rhinos found that vaccination elicited a response similar to domestic hoofstock, however about 5% of the vaccinated animals exhibited anaphylactic-like reactions (Miller 2003).

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

- Since many *Leptospira* serovars occur in New Zealand, and because the small numbers of imported zoo animals are not regarded as important in the epidemiology of leptospirosis, so clinically healthy rhinos could be imported without restrictions.
- During pre-export quarantine, rhinos could be serologically tested using the standard microscopic agglutination test (MAT). This test will identify which sero-groups are present, but not which serovars. Cross-reactivity between serovars confounds serovar-specific serological diagnosis. However, negative serology may not provide a strong assurance that the animal is not currently infected, as animals can test negative in the early stages of infection, or can fail to sero-convert and therefore be actively infected while testing negative. A negative test (50 % agglutination) at a 1:200 titre may provide the most appropriate interpretation (Greene et al 2006).
- Negative serology to a panel of antigens representing a wide range of serogroups could be required, even though this measure may mean rhinos infected with serovars endemic to New Zealand could be excluded.
- Rhinos could be allowed to be imported after completing antibiotic treatment for eliminating potential carriers of the organism. Oral ampicillin has been used effectively in rhinos (Miller 2003).

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

AQIS (2000) A Scientific Review of Leptospirosis and Implications for Quarantine Policy-Precis
http://www.daff.gov.au/_data/assets/pdf_file/0003/43086/00-030a.pdf

- Anonymous (2004).** Notifiable and other diseases in New Zealand. Annual Report 2003, Institute of Environment and Science Research, New Zealand, 26-7.
http://www.surv.esr.cri.nz/PDF_surveillance/AnnSurvRpt/2003AnnualSurvRpt.pdf.
- Bolin CA (2008)** Leptospirosis. In: OIE (ed). *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. OIE, Paris, 251-64.
- CFSPH Center for Food Security and Public Health, Iowa State University (2005).** Leptospirosis Factsheet <http://www.cfsph.iastate.edu/Factsheets/pdfs/leptospirosis.pdf>*
- Chereshky, A; Cameron, G; Marshall, R B (1993).** A case of human *Leptospira canicola* in New Zealand. *New Zealand Veterinary Journal* 41: 101.
- ESR (2003).** Institute of Environmental and Science Research. *Notifiable and Other Diseases in New Zealand*. Annual report 2003; New Zealand.
http://www.surv.esr.cri.nz/PDF_surveillance/AnnSurvRpt/2003AnnualSurvRpt.pdf
- Greene, C E; Sykes, J E; Brown, C A; Hartmann, K (2006)** Leptospirosis. In Greene, C E (ed) *Infectious Diseases of the Dog and Cat*. Elsevier; St. Louis; USA; pp 403-417.
- Hilbink F, Penrose M, McSporron K (1992).** Antibodies in dogs against *Leptospira interrogans* serovar *copenhageni ballum* and *canicola*. *New Zealand Veterinary Journal* 40 123-125.
- Horsch F (1989).** Leptospirosis. In: Blaha T (ed). *Applied Veterinary Epidemiology*. Elsevier Science Publishers, Amsterdam, 95-102.
- Hunter P (2004).** Leptospirosis. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Oxford University Press, Oxford, 1445-56.
- Leighton FA, Kuiken T (2001)** Leptospirosis In: Williams ES, Barker IK (eds) *Infectious Diseases of Wild Mammals*. Iowa State University Press, USA, 498-502
- Midwinter A (1999).** Spirochaetes in New Zealand. *Surveillance*, 26(3), 10-2.
- Miller RE (2003)** Rhinocerotidae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* Saunders Elsevier Science, USA, 558-569
- Murray CK, Hospenhal DR (2004).** Determination of susceptibilities of 26 *Leptospira* sp. serovars to 24 antimicrobial agents by a broth microdilution technique. *Antimicrobial Agents Chemotherapy*, 48(10), 4002-5.*
- Neiffer DL, Klein EC, Wallace-Switalski C (2001)** *Leptospira* Infection in Two Black Rhinoceros *Journal of Zoo and Wildlife Medicine* 32(4), 476-486*
- OIE, 2003.** Report of the meeting of the bureau of the OIE Terrestrial Animal Health Standards Commission 30th June to 4th July 2003.
- OIE, 2007.** Report of the meeting of the OIE Terrestrial Animal Health Standards Commission, September 2007, OIE, Paris. http://www.oie.int/downld/SC/2007/A_TAHSC_September%202007_introduction.pdf*
- Thompson A (1980).** The first New Zealand isolation of *Leptospira interrogans* serovar *australis*. *New Zealand Medical Journal*, 91(651), 28.
- Thornley CN, Baker MG, Weinstein P, Maas EW (2002).** Changing epidemiology of human leptospirosis in New Zealand. *Epidemiology and Infection*, 128(1), 29-36.*
- VGHI Victorian Government Health Information (2008)** Leptospirosis
<http://www.health.vic.gov.au/ideas/bluebook/leptospirosis>*

12. Babesiosis (*Babesia* spp.)

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agents

Only 2 *Babesia* spp. that may be associated with rhinos are known to be present in Australia:

Babesia bigemina
Babesia bovis

12.1.2. OIE list

Bovine babesiosis is listed under the category of “cattle diseases”.

12.1.3. New Zealand status

Babesia spp. are listed as unwanted notifiable organisms.

12.1.4. Epidemiology

Bovine babesiosis is the major tick-borne disease of cattle in Australia and is caused by the protozoan parasites *Babesia bovis* and *Babesia bigemina*. The cattle tick, *Boophilus microplus*, is the only vector for bovine babesiosis in Australia and the infection is widespread throughout the tick's endemic area (AHA 2005).

Babesiosis is a serious disease characterised by high morbidity and mortality in naïve cattle when they are introduced into tick infested areas. The severity of infection depends on the species of *Babesia* and the animal's age. Clinical signs are a result of red blood cell lysis as the organism parasitises these cells. Typical disease signs are fever and haemolytic anaemia accompanied by haemoglobinuria, from which the common name of “redwater” is derived.

After recovery from infection cattle develop a lasting immunity which is not dependent on persistent infection. Infected animals remain carriers for long periods. The persistence of infection is variable depending on the species of *Babesia* and the species of cattle. Cattle infected with *B. bovis* generally remain infective for ticks for up to two years while those infected with *B. bigemina* are infective for ticks for only 4-7 weeks (De Vos et al 2004).

Although some *Babesia* spp. commonly found in domestic animals have been reported from wild ungulates and carnivores closely related to their domestic counterparts, this would appear to be the exception to the rule. Exceptions may occur where wild animals are held in captivity. Under these conditions, infection of wild animals with *Babesia* spp. from domestic animals may well prove fatal. Indications are that wild animals have their own *Babesia* spp., with varying degrees of host specificity, and that endemic stability generally prevails (Penzhorn 2006).

Babesia spp. in black rhinoceros were first recorded in Kenya in 1967, and in 2003 they were characterised and named *Babesia bicornis* (Nijhof et al 2003). Similar *babesia* parasites have been reported in white rhinos, but the species is yet to be confirmed.

A number of drugs have been used for the treatment of the disease. Imimidocarb is a useful drug that has a prophylactic effect that lasts from 4-8 weeks (De Vos et al 2004) and is recommended by OIE for the treatment of cattle for international trade (OIE 2008).

Examination of blood smears is used for the diagnosis of acute infections but in persistent infections the number of parasites in the blood is too low to be reliably detectable by this means. In cattle, PCR tests are available, (De Vos et al 2004) and an ELISA is available for the diagnosis of *B. bovis* but not for *B. bigemina*. An indirect fluorescent antibody test is widely used for both *B. bigemina* and *B. bovis* but cross reactions occur between different *Babesia* spp. (De Vos et al 2004).

12.1.5. Hazard identification conclusion

Babesia spp. are exotic unwanted organisms that cause serious disease in cattle. However, *Babesia* spp. of cattle are not known to infect species other than cattle, African buffalo, and possibly some antelope species (Worthington and Bigalke 2001).

Babesia bicornis identified in black rhinos is not known to occur in Australia, and has not yet been confirmed in white rhinos. This species has not been found in animals other than rhino so may be host specific (Penzhorn 2006). *Babesia* spp. are therefore not considered to be potential hazards in the commodity.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

AHA Animal Health Australia (2005) Bovine Babesiosis
<http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/bovb.cfm>*

De Vos AJ, De Waal DT, Jackson LA. (2004). Bovine babesiosis. In: Coetzer JAW, Tustin RC (eds). *Infectious Diseases of Livestock*. Oxford University Press, Oxford, 406-24.

Kuttler KL (1998). Babesiosis. Foreign Animal Diseases. "The Gray Book".
http://www.vet.uga.edu/vpp/gray_book/FAD/index.htm.*

Nijhof AM, Penzhorn BL, Lynen G, Mollel JO, Morkel P, Bekker CPJ, Jongejan F (2003) *Babesia bicornis* sp. nov. and *Theileria bicornis* sp. nov.: Tick-borne parasites associated with mortality in the Black Rhinoceros (*Diceros bicornis*) *Clinical Microbiology* 41(5), 2249-2254

OIE (2008). Bovine babesiosis. In: OIE (ed). Terrestrial Animal Health Code Chapter 11.2
http://www.oie.int/eng/normes/mcode/en_chapitre_1.11.2.htm OIE, Paris.

Penzhorn BL (2006). Babesiosis of wild carnivores and ungulates *Veterinary Parasitology* 138 (1,2) 11-21.

Worthington RW, Bigalke RD (2001). A review of the infectious diseases of African wild ruminants. *Onderstepoort Journal of Veterinary Research*, 68, 291-323.

13. Internal Parasites

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agents

The following internal parasites were identified in the preliminary hazard identification as being able to infect rhinos, and potentially present in Australia:

Nematodes: multiple genera including- *Kiluluma* spp, *Khalilia* spp, *Murshidia* spp, *Quilonia* spp, *Oxyuris* spp.

Cestodes: *Anoplocephala* spp.

13.1.2. OIE list

No parasites that may infect rhinos are listed.

13.1.3. New Zealand status

Of the internal parasites that may infect rhinos only the trematode

Fasciola gigantica is listed as unwanted. This parasite is not present in Australia.

13.1.4. Epidemiology

Internal parasites belong to three basic groups:

- Cestodes (tapeworms)

- Trematodes (flukes)

- Nematodes (mainly intestinal parasites)

The diversity of helminth species is extensive. At least 40 known species have been reported in rhinos and most of these are nematodes. Several strongylid nematode genera predominate, and the most abundant species is the small pinworm *Probstmayria vivipara* (Penzhorn et al 1994). *P.vivipara* is endemic in New Zealand horses (McKenna 1997). It is generally considered non-pathogenic as heavy infections may occur without clinical signs (Taylor et al 2007).

Of the other strongylid genera identified as aetiological agents, most were recovered from free-living black and white rhinos in Central and South Africa (Penzhorn et al 1994). In addition *Oxyuris karamoja* and *Kiluluma* spp. have been found in captive white rhinos in Australia (TWPZ 2008). *Oxyuris* spp. have also been found in captive white rhino in New Zealand, but were not identified to species level. They are seen infrequently in captivity and are considered a normal part of gut flora, so are not treated for (Kudeweh 2009). *Oxyuris equi* has been found to infect rhinos but this species is endemic in New Zealand horses (McKenna 1997). There are 11 possible species of *Kiluluma*, and this nematode was also found in one import of rhinos from Australia into New Zealand and infection was eliminated (Kudeweh 2009).

Tapeworms are commonly seen in captive rhinos, but are usually asymptomatic (Miller 2003). There are no reports of cestodes from captive Australasian white rhino.

The cestodes *Anoplocephala diminuta*, and *A. gigantea* were recovered from the white and black rhinos in Africa (Penzhorn et al 1994) and *A. latissima* and an unknown species were recovered from a captive Indian rhino in Japan (Matsuo and Sumiya 2005).

The apparent absence of trematodes in captive Australasian rhino populations may be due to lack of a suitable snail intermediate host. Certain nematode species present in the wild may also be absent in regional captive populations for this reason.

Gyrostigma pavesii (stomach bot) was found in white rhino in Australia following the 1999 importation from South Africa. All animals were treated and no further cases were noted (Portas 2009). There have been no reports of this parasite in New Zealand rhino (Kudeweh 2009).

As comprehensive surveys of intestinal parasites present in New Zealand zoo animals have not been undertaken, it is likely that those parasites present in Australian captive rhinos are also present in the New Zealand captive rhino population. The founding members of each population were from the same South African import shipment in the late 1990's. Very little is known about the lifecycles of these rhino-specific parasites, and they often appear to be non-pathogenic as incidental findings.

Anthelmintic resistance of parasites is a major problem that occurs world-wide. Introduction of resistant strains of endemic parasite species, that are resistant to an anthelmintic type for which resistance does not occur in New Zealand should be considered to be a biosecurity risk.

Anthelmintic resistance to the commonly-used anthelmintics for nematode control is widespread in New Zealand ruminants and has been reported in the small strongyles of horses, (Taylor et al 2007) so it is possible that these strongyle species in captive rhinos may be resistant strains not already present in New Zealand.

Internal parasite infections are diagnosed 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 live animal Import Health Standards and those of our trading partners. No other practical methods are available for this purpose. Identification of parasites to species level as part of a quarantine procedure is often not possible and the criterion generally used for imported animals is that they should be entirely free from all parasite eggs in the standard egg flotation method used when examining faeces from imported animals.

In rhino, this method should probably be supplemented with a scraping or sticky-tape preparation from the perineum; as experience in Australia has found this to be much more reliable for detecting *Oxyuris karamoja*, which has not been identified via faecal floatation even in heavily infested animals (Portas 2009).

13.1.5. Hazard identification conclusion

The internal parasites that have been reported in rhinos are too numerous to be considered individually. Most are species specific, but as surveys of parasites already present in New Zealand rhinos have not been undertaken, it is not possible to say which are, or are not already here. There have been no reports of rhino-specific parasites establishing in other potential hosts (for example horses) in New Zealand and so the actual risk to biosecurity is also unknown.

However, given the wide range of internal parasites potentially present in rhinos and the uncertainty regarding their significance, they are considered to be potential hazards in the commodity.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

New species of parasites are likely to be introduced with infested/carrier animals that show no clinical signs. Therefore the likelihood of entry in imported rhinos is considered to be non-negligible. However, there is no evidence that anthelmintic resistant parasites exist in rhinos.

13.2.2. Exposure assessment

Imported rhinos will be integrated with New Zealand captive rhinos and would shed eggs and larvae of internal parasites on pasture within enclosures. There is no evidence that rhino internal parasites can infect non-rhino species, but existing rhinos in zoo enclosures are likely to be exposed to the eggs/larvae. The likelihood of exposure is therefore considered to be non-negligible.

13.2.3. Consequence assessment

New parasites could be introduced and become established in New Zealand. The parasites covered in this section are generally not considered to be significant pathogens for rhinos, or other animals. Therefore the health consequences for potentially affected species are likely to be minimal. There have been no reports of parasite species present in rhinos infecting humans.

The potentially anthelmintic resistant endemic strains of strongyles that could infect rhinos are primarily pathogens of horses. *P. vivipara* is not associated with clinical signs, and *O. equi* within the intestine rarely causes clinical signs. Heavy infestations of *O. equi* however, can result in widespread small erosions of the intestinal mucosa and an associated inflammatory response. Perineal irritation and intense pruritis caused by adult females during egg laying, can cause the affected animal to rub on solid objects creating bare patches, inflammation and scaling of the skin over the rump and tail head. The intense itching often leads to restlessness and impaired feeding, causing some loss of condition (Taylor et al 2007). The lifecycle is direct, and horses are only infected by ingestion of embryonated eggs. As rhinos and horses will not be in direct contact, or share the same pasture, exposure would only be possible by the transfer of eggs on a fomite or person. The likelihood of this occurring is considered to be extremely low.

Anthelmintic resistance has not been reported in *A. perfoliata* (Reinemeyer et al 2006).

Given the wide range of poorly characterised exotic internal parasites that can infect rhinos, it is possible that potentially affected species have not yet been exposed, and so susceptibility and therefore consequences are unknown. Given this uncertainty, the consequences of introduction are considered likely to be very low, but non-negligible.

13.2.4. Risk estimation

Since entry, exposure, and consequence assessments are non-negligible, the risk estimate for exotic parasites or anthelmintic resistant strains of endemic parasites is non-negligible

and they are classified as hazards in the commodity. Therefore, risk management measures can be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

One or a combination of the following measures could be considered in order to mitigate the risk of importing exotic endoparasites, or anthelmintic resistant endemic parasites. These measures could integrate well with the recommendations for preventing the importation of exotic ticks.

- Rhinos could be treated with an endoparasiticide effective against nematodes and cestodes 7-10 days prior to entering pre-export isolation.
- Rhinos could be held in quarantine for a period of 30 days in premises with an impervious washable floor or on an impervious pad. While in quarantine soiled bedding could be removed at least every 10 days and floors could be washed by high pressure hosing or steam cleaning (note that this measure would need to be combined with a treatment option in order to be effective).
- Within 3 days of export to New Zealand animals could again be treated with an endoparasiticide.
- Rhinos could be treated with an endoparasiticide within 48 hours after entering pre-export isolation. The efficacy of the endoparasiticide could be checked 7-14 days after the endoparasite treatment by examining faeces samples from the treated cattle by the faecal floatation concentration/sedimentation method (Egweg and Slocombe 1982) AND
- by examination of a perineal scraping or sticky-tape preparation; and be required to give a zero roundworm and tapeworm egg count.
- Treatments and testing could be repeated on animals that have positive egg counts until they give a zero roundworm and fluke egg count, the anthelmintic type should be changed as necessary.

In the case of surviving parasites larval cultures could be made, the parasites identified, and MAF notified of the results. Where pathogenic endoparasite spp. exotic to New Zealand are identified, the animals could be considered ineligible for importation until treatment has been demonstrated to be effective (or the organism is no longer considered exotic to New Zealand). Where endoparasite spp. identified are demonstrated to be non-pathogenic and/or species specific the biosecurity risk may be determined to be negligible, and the animals may be considered eligible for import.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 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.

Kudeweh S T (2009) Hamilton Zoo, Team Leader Mammals, email *pers comm* to Somervell R

Matsuo K, Sumiya G (2005). Anoplocephala sp. from the Indian Rhinoceros (*Rhinoceros unicornis*) in Higashiyama Zoo, Japan. *Japanese Journal of Zoo and Wildlife Medicine* 10(2) 95-98.*

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

Miller RE (2003) Rhinocoridae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* Saunders Elsevier Science, USA, 558-569

Penzhorn BL, Krecek RC, Horak IG, Verster AJM, Walker JB, Boomker JDF, Knapp SE, Quandt SKF (1994) Parasites of African Rhinos: A Documentation: In Penzhorn BL et al *Proceedings of a Symposium on Rhinos as Game Ranch Animals*. Onderstepoort, Republic of South Africa. 168-175

Portas T (2009) Australia Zoo, Veterinary Services Manager, draft review to Somervell R *pers comm*..

Reinemeyer CR, Hutchens DE, Eckblad WP, Marchiondo AA, Shugart JI (2006) Dose confirmation studies of the cestocidal activity of pyrantel pamoate paste in horses. *Veterinary Parasitology* 138(3-4), 234-239

Taylor MA, Coop RL, Wall RL (2007). *Veterinary Parasitology* 3rd ed. Blackwell Publishing; Oxford

TWPZ Taronga Western Plains Zoo (2008) . B Bryant Senior Veterinarian *pers comm*.to R Somervell.

14. Ticks

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agents

World-wide there are around 170 species of Argasidae or soft ticks and 650 species of Ixodidae or hard ticks (Allan 2001).

14.1.2. OIE list

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

14.1.3. New Zealand status

There are nine tick species in New Zealand, most of which are found on wild birds (Heath 1977). The cattle tick *Haemaphysalis longicornis* is the only one of economic importance to livestock and agriculture (Loth 2004).

All exotic ticks are notifiable under the Biosecurity Act 1993.

14.1.4. Epidemiology

Ticks are blood-feeding external parasites of mammals, birds and reptiles. Ticks have many susceptible hosts and they are important vectors of disease-causing agents for humans and animals throughout the world (Loth 2005). A broad range of organisms can be carried by ticks including bacteria, rickettsiae, protozoa and viruses. Some species of tick inject neurotoxins into their host while feeding causing paralysis and death in animals and humans. Blood taken up by the tick remains largely undigested, existing as a food reserve which is gradually consumed. Pathogens in the blood may survive for long periods in this environment (Grattan-Smith 1997).

An infected tick may carry a particular pathogen for life. A female tick can transmit some blood-borne pathogens to her eggs by transovarial transmission (through the eggs to the next generation of larvae) while other pathogens may only be transmitted transstadially (between development stages). Some pathogens can be transmitted transovarially and transstadially. For multi-host ticks, where each subsequent life stage must find a host and feed, it is possible to transmit tick-borne organisms to multiple hosts.

Argasid ticks have soft leathery bodies and feed for 5-25 minutes. The Ixodidae ticks are characterized by a hard body plate and a prolonged feeding time (Grattan-Smith et al 1997). For example *Rhipicephalus sanguineus* may take up to 21 days to engorge (Soulsby 1969).

There are approximately 75 species of tick in Australia, the majority of which are Ixodidae- hard ticks (MedEnt 2003). *Ixodes holocyclus* has been found on all ruminants and horses in Australia, and *Ixodes australiensis* and *Boophilus microplus* have also been recorded on cattle (TAG 2005). *Ixodes holocyclus* is a three-host paralysis tick, and it's distribution is roughly within a 20-kilometre band along the eastern coastline of Australia (MedEnt 2003). *Ixodes australiensis* is found in Western Australia and Tasmania (TAG 2005). *Boophilus microplus* is found in QLD and NSW. It is a notifiable to the Department

of Agriculture in NSW, where planned control programs, quarantine areas and regulations for livestock movement keep the number of infested properties low (TAG 2005).

Common sites of tick attachment on rhinos are skin folds in the perineal region, in and around the ears, and around the eyes (Penzhorn et al 1994). Neither *Ixodes* or *Boophilus* tick spp. have been reported on rhinos. In Australian zoo's external parasites 'are not seen' on captive white rhinos (TWPZ 2008). This is despite regular inspection of typically affected areas on the rhinoceros such as the ears and perineum during routine blood collection (Portas 2009).

However, infestation with ticks is commonly noted on rhinos (Miller 2003) with 18 different Ixodid tick species recovered from 6 black and 3 white free-living rhinos in South Africa and Zimbabwe (Knapp et al 1997). Ten of the reported 18 species were present on white rhinos. Many of the species were considered 'accidental' parasites, and none of the tick species recorded occur in Australia or New Zealand.

In Australia, rhino have remained tick free while free-ranging macropods, reptiles and echidnas in the local area have been heavily parasitized by a range of ticks. It is possible that the ticks present in Australia lack suitable mouth parts to parasitise the thick dermis of the rhinoceros. (Portas 2009)

14.1.5. Hazard identification conclusion

Apart from one species, all ticks known to infest rhinos are exotic to New Zealand. Many species are vectors of zoonotic diseases and can also cause production losses associated with parasitism of animals. Ticks have the potential to parasitise all mammals, birds and reptiles. Importing the one species of tick endemic to NZ is also hazardous as any tick could harbour exotic pathogenic agents. Ticks are therefore considered to be potential hazards in the commodity.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

Rhinos have the potential to carry exotic tick species present in Australia. Zoo animals in general are not considered a significant pathway for the introduction of exotic ticks, mainly due to small volumes of animals imported, and reduced exposure in captivity; particularly where premises are not situated within known tick distribution zones. Pre-export inspection is meticulous, but in some cases small tick larvae may be almost impossible to detect. Therefore the likelihood of introducing exotic tick species is considered to be low but non-negligible.

14.2.2. Exposure assessment

Ticks can survive for long periods and have many susceptible hosts. Ticks carried by imported rhinos could leave their hosts and complete their lifecycle by infesting other rhinos, humans, or wildlife that may access their enclosures.

As a similar climate exists in regions of the North Island of New Zealand, to the climate in the distribution zone of *Ixodes holocyclus*, it is reasonable to conclude that it could establish itself here (Loth 2005). It is thought that all ticks could establish in New Zealand, except for those adapted to the most arid climates.

The likelihood of exposure is therefore considered to be low but non-negligible.

14.2.3. Consequence assessment

The major consequences of exotic tick establishment are the direct effects of parasitism and toxicity; the possible introduction of exotic tick-borne diseases; and the increased risk of introduced exotic diseases being able to establish in New Zealand if suitable tick vectors are established here.

The parasitic effects of ticks in sufficient numbers can include anaemia as a result of blood ingestion, debilitation and skin disease associated with hypersensitivity and bacterial pyoderma (Irwin & Jefferies 2004). *I holocyclus*, the Australian paralysis tick is one of the most toxic of all the worlds paralysing ticks. It is the cause of paralysis and death in pets, domestic animals, mice and humans (Grattan-Smith 1997). Infestation of New Zealand ruminants would result in associated production losses as well as expenses incurred to control ticks.

Ticks present in Australia are capable of carrying and transmitting *Rickettsia australis*, which causes Rickettsial Spotted Fever (also known as Queensland Tick Typhus) and *Rickettsia honei* which causes Flinders Island Spotted Fever in animals and people. It is speculated but uncertain whether Australian ticks can carry and transmit *Borrelia burgdorferi*, so this potential risk is described as ‘Lyme-like’ disease (TAG 2005). It is not known if rhinos would be capable of being infected with and/or transmitting these diseases, but if the tick became established in NZ it would be likely to infest and/or infect other animal species.

If new tick species were to become established in New Zealand the likelihood of exotic tick-borne diseases establishing here at some point in the future will be increased. The absence of tick borne diseases in New Zealand may be attributable to the limited vector potential of *H. longicornis*.

The effects on the health of humans and animals may be severe. If an exotic tick were to establish, eradication would be difficult and expensive. The consequences are therefore assessed to be non-negligible

14.2.4. Risk estimation

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

14.3. RISK MANAGEMENT

14.3.1. Options

One or a combination of the following measures could be considered in order to mitigate the risk of importing exotic tick species:

- Rhinos could be treated with an acaricide 7-10 days prior to entering pre export isolation (PEI).
- Rhinos could be treated during the 48 hours immediately prior to entering PEI 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 backpack spray unit).

- Rhinos 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 could be considered suitable. The animals could be fed rations that are inspected and determined to be free from potential contamination with ticks, tick eggs, larvae or nymphs. (note that this measure needs to be combined with a treatment option in order to be effective)
- Rhinos could have all the bedding on which they are housed removed every ten days during the quarantine period and, at this time, the walls and floor could be thoroughly cleaned, and sprayed with an acaricide.
- Rhinos could be meticulously inspected for ticks and other ectoparasites, at least 10 days after entering PEI. If still infested, the treatment could be repeated and animals inspected again at least 10 days later. Treatments and inspections could be repeated until the animals are found to be free from evidence of ticks. The ectoparasiticide could be altered if the previously used treatment has not been effective.
- Rhinos could be treated with an acaricide within the 3 days prior to shipment.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

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

Grattan-Smith, P J; Morris, J G; Johnston, H M; Yiannikas; et al. (1997). Clinical and neurophysiological features of tick paralysis. *Brain : A Journal of Neurology* 120 (Pt 11): 1975-1987.

Heath, A C G (1977). Zoogeography of the New Zealand tick fauna. *Tuatara* 23(1): 26-38

Irwin, P J; Jefferies, R (2004). Arthropod-transmitted diseases of companion animals in Southeast Asia. *Trends in Parasitology* 20(1): 27-34.

Knapp SE, Krecek RC, Horak IG, Penzhorn BL (1997). Helminths and Arthropods of Black and White Rhinoceroses in Southern Africa. *Journal of Wildlife Diseases* 33(3) 492-502

Loth, L (2004). *Review of tick investigations performed by MAF NCDI and development of standard operating procedures*. MAF NCDI; Wellington, New Zealand.

Loth, L (2005). Review of exotic tick interceptions in New Zealand since 1980 *Surveillance* 32(3): 7-9.

MedEnt (2003). Ticks *University of Sydney Department of Medical Entomology*
<http://medent.usyd.edu.au/fact/ticks.htm>

Merial (2008). Tick paralysis prevention. Available at:
http://au.merial.com/pet_owners/dogs/tick_paralysis1.asp*

Miller RE (2003). Rhinoceridae In: Fowler ME, Miller RE (eds) *Zoo and Wild Animal Medicine* Saunders Elsevier Science, USA, 558-569

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

Penzhorn BL, Krecek RC, Horak IG, Verster AJM, Walker JB, Boomker JDF, Knapp SE, Quandt SKF (1994). Parasites of African Rhinos: A Documentation: In Penzhorn BL et al *Proceedings of a Symposium on Rhinos as Game Ranch Animals*. Onderstepoort, Republic of South Africa.168-175

Portas T (2009) Australia Zoo, Veterinary Services Manager, draft review to Somervell R *pers comm.*.

Soulsby, E J L (1969) Suborder: *Ixodoidea* (Ixodides). In Soulsby, E J L (ed) *Helminths, Arthropods, and Protozoa of Domestic Animals*. Bailliere, Tindall and Cassell; London; pp 465-495.

TAG (2005) Ticks of Australia *Tick Alert Group Support* <http://www.tickalert.org.au/ticksaust.htm>*

TWPZ Taronga Western Plains Zoo (2008). B Bryant Senior Veterinarian *pers comm.* to R Somervell.

15. Weed Seeds

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

All plant seeds and plant material.

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Organisms of concern are all exotic plants and plant seeds.

15.1.4. General considerations

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 lemmings burrow that was dated as 10,000 years old germinated within 48hours when placed in favourable conditions (Anonymous undated). 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.

Weeds and weed seeds could be found attached to the sparse hair, or within the deep skin folds of imported rhinos. Large seed heads and pieces of plant material would be easily visible and could be removed before shipment but small seeds may not be visible.

Weed seeds can survive passage through an animal's digestive system and be passed out in the faeces (Katovich undated).

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

15.1.5. Hazard identification conclusion

It is concluded that weed seeds could be introduced on animal's hair, within skin folds, or in their faeces. Therefore weed seeds are considered to be potential hazards in the commodity.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

As seeds and plant material could be introduced attached to animal's hair, within skin folds and in faeces, the likelihood of entry in the commodity is non-negligible.

15.2.2. Exposure assessment

Weed seeds could become detached from animal's hair, dislodged from skin folds, or released 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 non-negligible.

15.2.3. Consequence assessment

As a result of the release of exotic weed seeds, exotic weeds could be introduced and become established with subsequent deleterious effects on the environment and the economy.

This could include out-competing native flora and over-running pasture thereby reducing biodiversity and stock grazing areas. The cost and resources required to control can be significant, as seen with invasive exotic weeds already present in New Zealand.

15.2.4. Risk estimation

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

15.3. RISK MANAGEMENT

15.3.1. Options

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

- The rhinos could be thoroughly washed and then inspected for contaminating plant material immediately prior to entering pre-export quarantine.
- The measures appropriate to control the introduction of ticks would also 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 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 the animals body surface.
- Feeding of rations that are inspected and determined to be free from potential contamination with weed seeds will ensure that the animals do not ingest new burdens of weed seeds.
- 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. 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 (Ministry of Agriculture and Forestry 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. Dung produced during transport could be safely disposed of, either enroute or on arrival in New Zealand.

References

References marked * have been sighted as summaries in electronic media. All weblinks were accessed in November 2008.

Anonymous (undated). Germination, Dormancy and life-span of seeds. In encyclopaedia Britannica. Britannica online. <http://britannica.com/eb/article-75927?hook+606897>

Barton K, Williams PA (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>

Ministry of Agriculture and Forestry (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>