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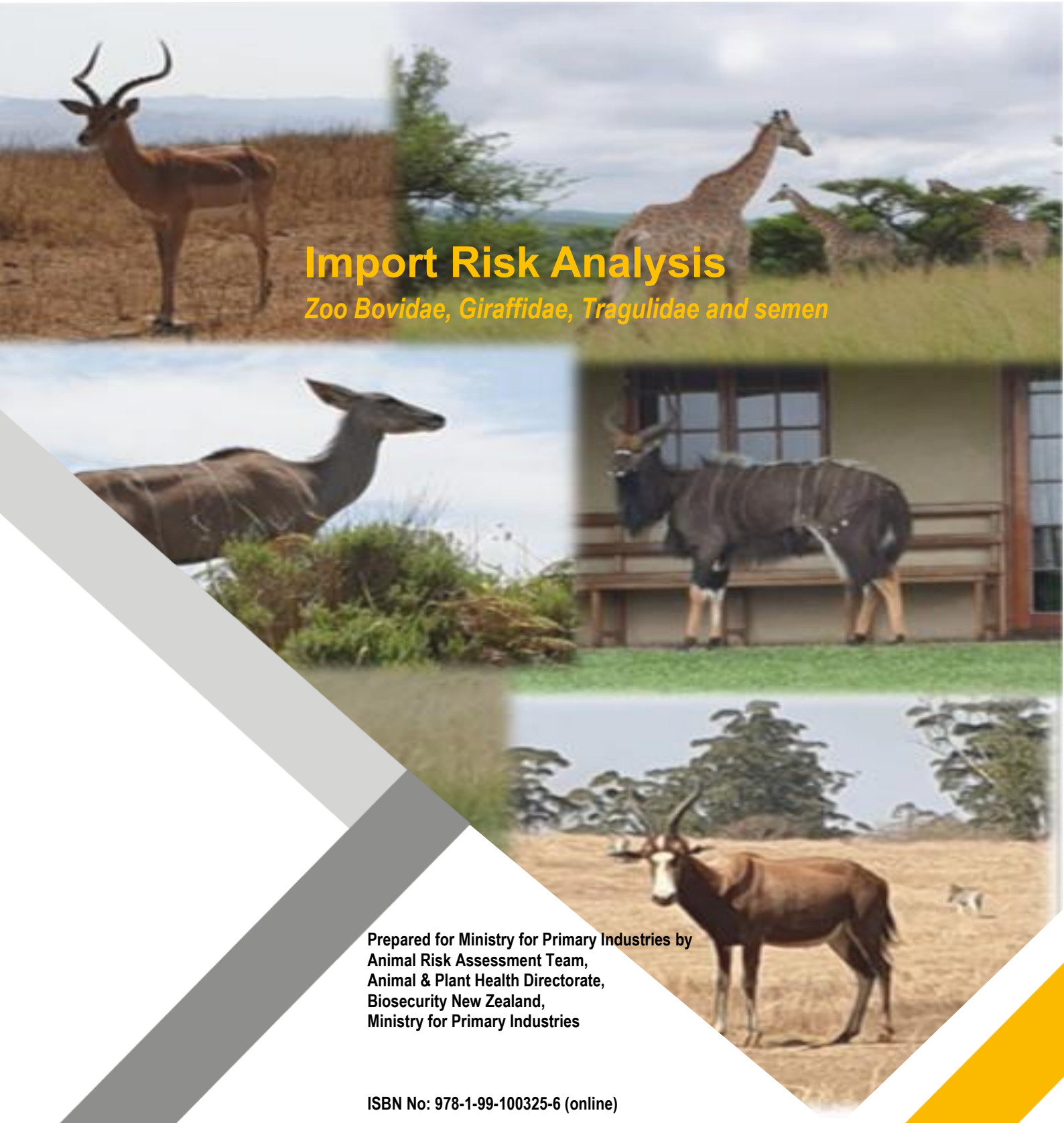

Import Risk Analysis

Zoo Bovidae, Giraffidae, Tragulidae and semen

Prepared for Ministry for Primary Industries by
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Import Risk Analysis: Zoo Bovidae, Giraffidae, Tragulidae and semen

16 March 2021

Approved for IHS Development

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New Zealand is a member of the World Trade Organization and a signatory to the Agreement on the Application of Sanitary and Phytosanitary Measures ("The Agreement"). Under the Agreement, countries must base their measures on an International Standard or an assessment of the biological risks to plant, animal or human health.

This document provides a scientific analysis of the biosecurity risks associated with live zoo (captive wild) Bovidae, Giraffidae, Tragulidae and their semen. It assesses the likelihood of entry, exposure, establishment or spread of agents associated with these commodities and assesses the potential impacts of these organisms should they enter and establish in New Zealand. The document has been internally and externally peer reviewed and is now released publicly. Any significant new science information received that may alter the level of assessed risk will be included in a review, and an updated version will be released.

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Table of contents

Index of figures and tables

Acronyms and abbreviations

Term/Acronym	Definition
ACE	antigen-capture ELISA
AGID	agar gel immunodiffusion
AI	artificial insemination
AlHV-1	*Alcelaphine gammaherpesvirus 1*
ASMP	Australasian species management program
BA	*Bacillus anthracis*
BHV 1	*Bovine alphaherpesvirus 1*
BPAT	buffered plate agglutination test
BSE	bovine spongiform encephalopathy
BST	brucellin skin test
BT	bluetongue
BTB	bovine tuberculosis
BTV	*Bluetongue virus*
BVD	bovine viral diarrhoea
BVDV	*Bovine viral diarrhoea virus*
Captive wild (animal)	means an animal that has a phenotype not significantly affected by human selection but that is captive or otherwise lives under direct human supervision or control, including zoo animals and pets.[1]
CATT	card agglutination test
CBPP	contagious bovine pleuropneumonia
CCHF	Crimean Congo haemorrhagic fever
CCHFV	*Crimean Congo haemorrhagic fever virus*
CCPP	contagious caprine pleuropneumonia
C-ELISA	competitive ELISA
CFT	complement fixation test
CPXV	*Cowpox virus*
CTST	comparative tuberculin skin test
DFAT	direct fluorescent antibody test
dRIT	direct rapid immunohistochemistry test
EAE	enzootic abortion of ewes
ECF	East Coast fever
EDTA	ethylenediaminetetraacetic acid
EFSA	European Food Safety Authority
EHD	epizootic haemorrhagic disease
EHDV	*Epizootic haemorrhagic disease virus*
ELISA	enzyme-linked immunosorbent assay
FAO	Food and Agriculture Organization
FPA	fluorescence polarisation assay
FMD	Foot and mouth disease
FMDV	*Foot and mouth disease virus*
HCT	haematocrit centrifugation technique
HS	haemorrhagic septicaemia
ICTV	International Committee on Taxonomy of Viruses
IBR/IPV	infectious bovine rhinotracheitis/infectious pustular vulvovaginitis
IFAT	immunofluorescent antibody titre test
IHS	import health standard
IPB	infectious pustular balanoposthitis
IRA	import risk analysis
KNP	Kruger National Park
LSD	lumpy skin disease
LSDV	*Lumpy skin disease virus*
Ma	*Mycoplasma agalactiae*
Mccp	*Mycoplasma capricolum* subsp. *capripneumoniae*
MCF	malignant catarrhal fever

[1] As per the Terrestrial Animal Health Code (2019) glossary. https://www.oie.int/index.php?id=169&L=0&htmfile=glossaire.htm

Term/Acronym	Definition
MCFV	*Malignant catarrhal fever virus*
MmmSC	*Mycoplasma mycoides* subsp. *mycoides* Small Colony
MPI	Ministry for Primary Industries
NSD	Nairobi sheep disease
NSDV	*Nairobi sheep disease orthonairovirus*
NWS	new world screw-worm fly
OIE	World Organisation for Animal Health
OIE Code	Terrestrial Animal Health Code of the OIE (World Organisation for Animal Health)
OWS	old world screw-worm fly
PI	persistently infected
PMSGBC	Prince Mohammed Al-Sudairi Gazelle Breeding Centre
PPE	personal protective equipment
PPR	peste des petits ruminants
PPRV	*Peste des petits ruminants virus*
PrP	prion protein
PRNT	plaque reduction neutralisation test
RT-PCR	reverse transcription polymerase chain reaction
RBT	rose bengal test
RPCP	ruminant protein control programme
RSA	Republic of South Africa
RVF	Rift Valley fever
RVFV	*Rift Valley fever virus*
SAT	Southern African Territories
SIT	sterile insect technique
SOP	standard operating procedure
SRM	specified risk material
TSE	transmissible spongiform encephalopathy
TST	tuberculin skin test
UAE	United Arab Emirates
UK	United Kingdom
USA	United States of America
USDA	United States Department of Agriculture
VI	virus isolation
WA-MCF	wildebeest-associated malignant catarrhal fever
WAHIS	World Animal Health Information Database
Wild (animal)	means an animal that has a phenotype unaffected by human selection and lives independent of direct human supervision or control.[1]
Wildlife	means feral animals, captive wild animals and wild animals.[1]
WTO	World Trade Organization

1 Executive summary

This document is a qualitative biosecurity import risk analysis for zoo Bovidae, Giraffidae, Tragulidae and their semen from Australia, Canada, Europe, Japan, Singapore, the Republic of South Africa, United Arab Emirates and the United States of America conducted by the Ministry for Primary Industries to facilitate a trade request by the Zoo and Aquarium Association of Australasia.

The trade of the above commodities will not only strengthen relationships between trading partners but also support the mission of the International Union for Conservation of Nature in promoting the conservation of biodiversity.

The methodology for this risk assessment follows the *Biosecurity New Zealand Risk Analysis Procedures - Version 1* (Biosecurity New Zealand 2006), the *Terrestrial Animal Health Code 2019* (Section 2 Risk Analysis) and the *OIE Handbook on Import Risk Analysis for Animals and Animal Products* (Volume 1, 2010).

From a preliminary list of hazards (approximately 100) of biosecurity concern that could be associated with the commodities and introduced into the country, 37 hazards were identified as requiring further assessment.

They are as follows:
Viruses:
Bluetongue virus
Alcelaphine gammaherpesvirus 1
Bovine viral diarrhoea virus
Cowpox virus
Crimean Congo haemorrhagic fever orthonairovirus
Epizootic haemorrhagic disease virus
Foot and mouth disease virus
Bovine alphaherpesvirus 1
Lumpy skin disease virus
Nairobi sheep disease orthonairovirus
Peste des petits ruminants virus
Rabies lyssavirus
Rift Valley fever phlebovirus

Bacteria and mollicutes:
Bacillus anthracis
Brucella abortus
Brucella melitensis
Mycobacterium bovis
Mycoplasma agalactiae
Mycoplasma mycoides subsp. *mycoides* SC[2]
Mycoplasma capricolum subsp. *capripneumoniae*
Mycoplasma bovis
Pasteurella multocida

Rickettsia:
Anaplasma marginale and *A. centrale*
Ehrlichia ruminantium
Coxiella burnetii

Protozoa:
Besnoitia besnoiti
Babesia bigemina, B. bovis and *B. divergens*
Theileria annulata and *T. parva*
Trypanosoma evansi
Trypanosoma brucei brucei, T. b. gambiense, T. b. rhodesiense, T. congolense and *T. vivax*

Parasites:
Cochliomyia hominivorax

[2] Recent taxonomic revision has dropped the designation “Small Colony (SC)".

Chrysomya bezziana

Prions:
Prions causing bovine spongiform encephalopathy

Internal parasites
External parasites
Seeds

Following a risk assessment, 23 hazards in live captive wild ruminants and 11 hazards in semen were assessed to be risks:

Live captive wild ruminants
Alcelaphine gammaherpesvirus 1
Bovine viral diarrhoea virus
Crimean Congo haemorrhagic fever orthonairovirus
Foot and mouth disease virus
Bovine alphaherpesvirus 1
Lumpy skin disease virus
Peste des petits ruminants virus
Rabies lyssavirus
Rift Valley fever phlebovirus
Bacillus anthracis
Brucella abortus
Brucella melitensis
Mycobacterium bovis
Mycoplasma capricolum subsp. *capripneumoniae*
Pasteurella multocida
Anaplasma marginale and *A. centrale*
Coxiella burnetii
Trypanosoma evansi
Trypanosoma brucei brucei, T. b. gambiense, T. b. rhodesiense, T. congolense and *T. vivax*
Internal parasites
External parasites
Seeds

Semen of captive wild ruminants
Bovine viral diarrhoea virus
Foot and mouth disease virus
Bovine alphaherpesvirus 1
Lumpy skin disease virus
Peste des petits ruminants virus
Rift Valley fever phlebovirus
Brucella abortus
Brucella melitensis
Mycobacterium bovis
Coxiella burnetii

The risk indicators for the vast majority of the hazards were assessed as very low to low. The main contributing factors to the low risk indicators included the low volume of trade of commodities, the absence of competent vectors in New Zealand and the negligible risk of a zoonotic potential. A summary of the risk analysis has been included in Appendix B.

The recommended risk mitigation measures included but were not limited to, country freedom declarations, physical examinations, health attestations, diagnostic testing, vaccination, long-term premises freedom, pre-export isolation and treatment.

2 Introduction

The Zoo and Aquarium Association of Australasia has requested the Animal and Plant Health Directorate (Animal Risk Assessment Team) to assess the biosecurity risk of importing zoo Bovidae, Giraffidae, Tragulidae and their semen directly into New Zealand zoos and wildlife parks.

The current antelope import risk analysis (IRA) *Diseases of antelope: Risks of introducing live antelope into zoological gardens – Import risk analysis (May 2000)* is 20 years old and may be outdated in terms of technical content. The IRA also does not cover all other required species.

The New Zealand zoo and aquarium industry is relatively small. Species population sizes can be limited. As a result, member organisations participate in the Australasian Species Management Program (ASMP) through a commitment to house program specimens and by supporting ASMP breeding and transfer recommendations.

Currently, zoos are limited by the number of species they are allowed to import, and there are limited countries from which they are allowed to import these species.

The ability to import the above species from a wider range of countries than currently permitted will aid zoos in their efforts to deliver high-quality species management that ensures sound genetic management and exceptional animal welfare and to meet the needs of their community and members.

For the purposes of this IRA, zoo animals will be referred to as captive wild animals.

3 Scope

This risk analysis is a qualitative assessment limited to the description of the risks due to disease-causing agents associated with the importation of captive wild animal species within the families Bovidae, Giraffidae, Tragulidae and their semen from approved, licensed or registered zoos or wildlife parks in Australia, Singapore, the United States of America (USA), Canada, Europe (not limited to European Union), the Republic of South Africa (RSA), Japan and United Arab Emirates (UAE). These countries are hereafter referred to as approved countries.

Other risk factors that may be of commercial importance to importers (e.g. genetic diseases) have not been considered in the analysis.

4 Commodity definition

The import risk analysis (IRA) considers the families Bovidae, Giraffidae, Tragulidae (under the taxonomic order Cetartiodactyla) and their semen. The IRA is restricted to species that have been listed in Appendix A.

These species are to be imported from the approved countries only.

Exclusions to this list include:

- Caprinae (chamois, goats, sheep, serows and relatives)
- Genera *Bos* (oxen and true cattle)
- Genera *Bubalus* (water buffalo)
- Genera *Syncerus* (African buffalo).

4.1 General considerations

4.1.1 General risk management measures applicable to all imported captive wild ruminant species (off-shore and on-shore)

- The animal must be resident in an approved, licensed or registered zoo or wildlife park in the exporting country since birth or for at least 12 months immediately before export, unless otherwise recommended in risk management measures and approved by MPI.

- The animal(s) was born in captivity and not caught from the wild.

- The premises of origin (zoo or wildlife park) must provide separation from other animal populations, be under veterinary supervision and have a documented health monitoring program that would be effective in monitoring for diseases of biosecurity concern identified in this IRA (e.g. post-mortem records for deceased animals; disease testing programs; health monitoring records, etc.):
    - The required outcome of veterinary supervision is up-to-date and regular knowledge of the animals, their health status, and the general health status of the institution that allows a veterinarian to sign off on these records.
    - The required outcome of separation is a sufficient distance or other barriers to maintain a distinct animal health status with regards to the diseases in this IRA.
    - The required outcome of a health monitoring programme is the regular monitoring, ongoing surveillance, and veterinary oversight to ensure that the health status of animals and an institution is known and monitored over time.

- The animal must be held in pre-export isolation for a period stipulated in the import health standard and isolated from all other animals not eligible for export to New Zealand.

- The animal must be transported to a transitional facility or containment facility in New Zealand that meets the requirements of the "Facility Standard: Zoo Animals Transitional Facilities (December 2018)" (Transitional Facility Std) and the "Standard for Zoo Containment Facilities (April 2018)" (Containment Facility Std), respectively. The animal must be transported in a manner that ensures no direct exposure to animals of a lesser biosecurity status en route and must undergo a period of post-arrival quarantine for a period stipulated in the import health standard.

4.1.2 General risk management measures relevant to semen

- The semen was collected, processed and stored in a hygienic manner.

- The donor animal(s) were resident in an approved, licensed or registered zoo or wildlife park in the exporting country since birth or for at least 12 months immediately before semen collection, unless otherwise approved by MPI.

- The premises of origin (zoo or wildlife park) must provide separation of the donor animal(s) from other animal populations, veterinary supervision and a documented health monitoring programme that would be effective in monitoring for diseases of biosecurity concern identified in this IRA.

- The donor animal(s) was not under quarantine restriction for the collection period or the 90 days immediately prior.

- The donor animal(s) showed no signs of infectious or contagious disease during the collection period and for the 30 days immediately after.

5 Methodology

5.1 General procedures

The methodology used in this risk analysis follows the guidelines described in *Biosecurity New Zealand Risk Analysis Procedures – Version 1* (Biosecurity New Zealand, 2006), the *Terrestrial Animal Health Code 2019* (Section 2 Risk Analysis) and the *Handbook on Import Risk Analysis for Animals and Animal Products* (OIE, 2010). The risk analysis process comprises three main steps: hazard identification, risk assessment and risk management.

5.1.1 Hazard identification

The preliminary list of hazards of biosecurity concern is compiled from the OIE-listed diseases, infections and infestations in force in 2020, published MPI and other risk analyses, and relevant published scientific literature.

For each agent in the preliminary hazard list, several steps are completed. These include formal identification of the agent, its status as agent of an OIE-listed disease, its New Zealand status (present, under a control programme, or exotic), along with an assessment of the relevant aspects of the epidemiology and characteristics of the agent (Figure 1).

Hazard identification concludes with an assessment of whether the agent is identified as a hazard in the commodity. The results of the hazard identification are commonly summarised as a table. All hazards are subjected to risk assessment.

5.1.2 Risk assessment

Risk assessment (refer to Figure 1) consists of:

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

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

The Biosecurity New Zealand Risk Analysis Procedures (Biosecurity New Zealand 2006) defines risk as either negligible, or non-negligible (see below), and proposes risk descriptors to describe the comparative levels of these risk attributes.

Table 1. Description of risk attributes and risk descriptors used in the risk analysis

Risk attributes	**Description**
Negligible	Not worth considering, insignificant
Non-negligible	Worth considering, significant
Risk descriptors	
Very low	Close to insignificant
Low	Less than average, coming below the normal level
Medium	Around the normal or average level
High	Extending above the normal or average level
Very high	Well above the normal or average level

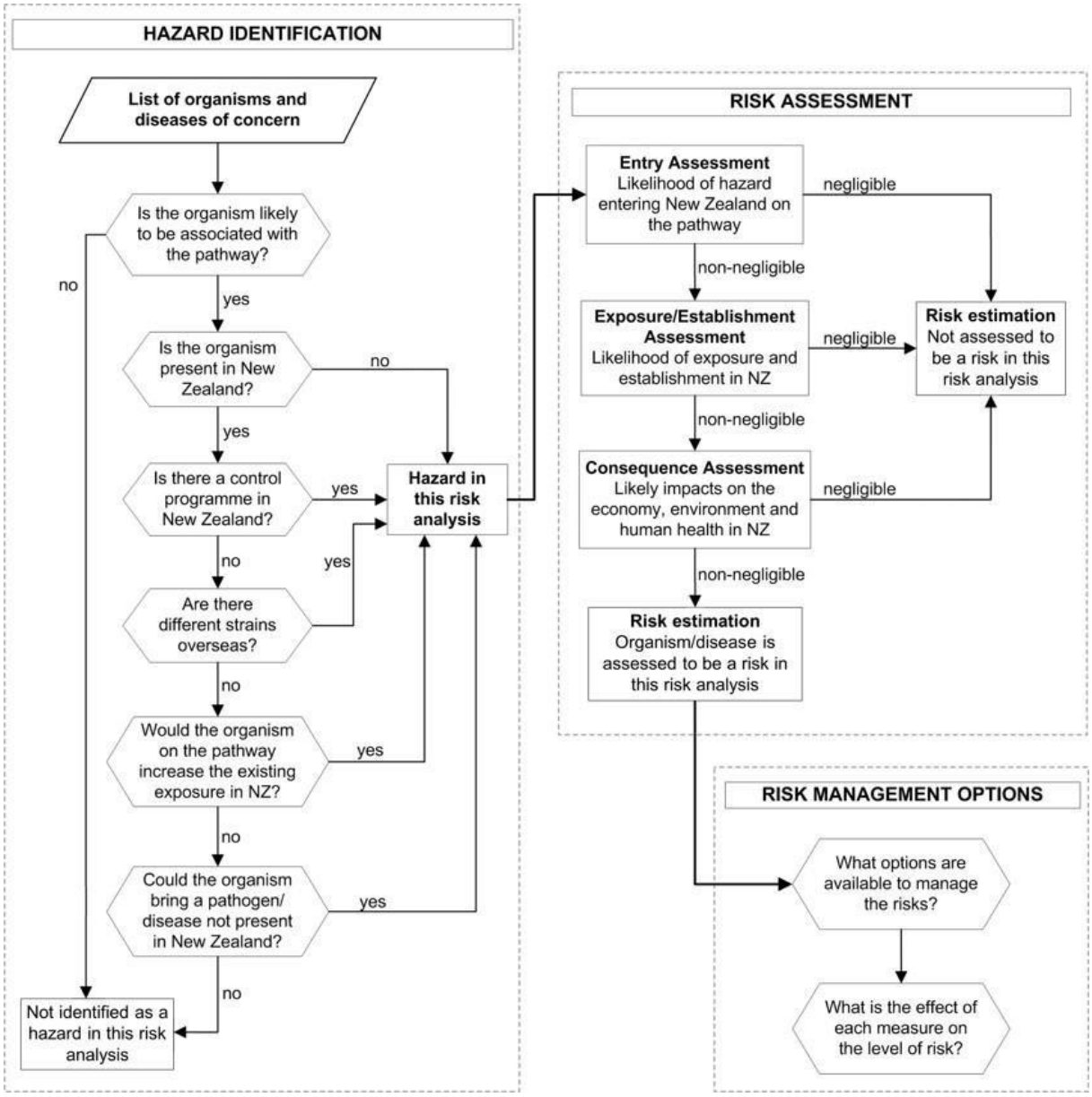

Figure 1. The risk analysis process

5.1.3 Risk management

Risk management identifies the options available for managing that risk, based on the epidemiology of the risk organism. Where the OIE Code lists recommendations for the management of a risk, these are described alongside options of similar, lesser or greater stringency, where available from the scientific literature. In addition to the options presented, unrestricted entry or prohibition may also be considered. 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 is drafted.

As obliged under Article 3.1 of the World Trade Organization (WTO) SPS Agreement (World Trade Organization, 2020), the measures adopted in Import Health Standards (IHSs) will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3. That is, 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 more appropriate based on a scientific risk assessment.

5.1.4 Risk communication

After a draft import risk analysis has been written, MPI analyses the options available and proposes draft measures for the effective management of the identified risks. These are then presented in a draft import health standard (IHS) that is released for public comment with a link to the draft risk analysis.

5.2 Special considerations

Some special considerations are applicable to the risk assessment and risk management processes for this IRA specifically.

Knowledge gaps:
Where there were knowledge gaps or a lack of published studies, research and literature, information from domestic and other wild ruminants has been included. This information is included due to the similarities noted in the epidemiology of certain diseases between species under analysis and domestic and other wild ruminants.

Disease prevalence data was very rarely considered, as this information is not often reported on in captivity. Instead, incidence (case studies and outbreaks) information has been used.

Inferences:
In this IRA, we extrapolated epidemiological scenarios from only domestic Bovidae to species within the scope of this IRA. Inferences were not made from species of the Cervidae family, Caprinae subfamily or other families, as these species were excluded from the scope.

Seroprevalence:
Seroprevalence data has often been used as an indicator of infection in wildlife populations. Various tests are used to obtain this data, each testing different immunological parameters. Thus, not all serological results can be interpreted in the same way. Seropositivity provides information about exposure history to an infectious agent and does not necessarily imply infection (Gilbert et al., 2013).

The immunopathogenesis of some diseases are quite complex and are not fully understood in some species within the scope of this IRA. For the purposes of this IRA; where the only evidence of association of an infectious agent with animals is that of seropositivity (i.e. antibody presence) and there is no documented evidence of clinical disease in these animals or transmission of the agent to other animals, these animals are not likely to play an important role in the epidemiology of the disease.

Vector competency:
Another area with accompanying uncertainty and knowledge gaps is vector competency of vector-borne diseases. New Zealand hosts various arthropod vectors. Some are known to be competent vectors of disease, while the competency of others is uncertain.

Historically, MPI's view was that if there is no published evidence proving vector competency (worldwide), then assumptions, predictions or inferences will not be made to suggest potential vector competency of New Zealand vectors. In compiling this IRA, a similar stance has been taken, as there is no scientific evidence to validate these assumptions.

However, should research in this area bring to light evidence that may contradict, clarify or change the conclusions made regarding vector competency, a review of relevant vector-borne disease chapters would be required.

Speculative events:
The risk assessments do not consider speculative events that could occur in the future, such as the possible geographic range expansion of arthropod pests that could be a result of increased human movement, international trade and global climate change. Conclusions were therefore based on current knowledge and present conditions.

Captive animal collection environment:
The captive animal collection environment is quite different to that of the wild or domestic animal environments. In a well-managed captive facility (i.e. with onsite veterinary supervision, regular and frequent testing for endemic diseases, appropriate separation between captive collections and wild or

domestic animals), animals usually remain free of a large number of endemic diseases, unless these diseases are vector-borne.

The other reason for the fairly low disease risk of these animals is the management and close veterinary and keeper supervision that accompanies captive collections. Welfare and the great value that these animals hold in terms of what they represent (i.e. conservation, awareness, education, research) ensures care is a priority.

Confinement, artificial diets, social stress and proximity to unfamiliar species are all factors that contribute to unique and sometimes novel opportunities for pathogens and parasites. An unnatural host–agent–environment relationship may eliminate risk from diseases recognised in natural ecosystems but may also elevate the risk from other diseases. For example, tuberculosis can perpetuate within the captive environment in the absence of appropriate biosecurity measures.

Country-specific disease sanitary status:
Approved countries (Australia, Singapore, United States of America, Canada, Europe (not limited to European Union), South Africa, Japan and United Arab Emirates) are included as probable countries for the importation of the commodities. The disease sanitary status of each country differs. As a result, these entry assessments identify countries that are free from the disease being assessed and assess entry from these countries as a negligible risk. It is recommended that a country freedom declaration be obtained to give assurance of negligible risk. If this cannot be obtained, a review of the disease risk from that country would be required and risk mitigation measures may need to be implemented (where applicable).

Exposure vs establishment:
The exposure assessments assess the likelihood of susceptible captive wild and domestic (if any) and humans being exposed to the agent, and the likelihood of the agent establishing or spreading. In some instances, e.g. bluetongue, Crimean Congo haemorrhagic fever and heartwater, there is a likelihood of exposure, and thus there would be limited consequences for those susceptible species (i.e. morbidity or mortality). However, due to the absence of further transmission pathways, the agent is very unlikely to establish in New Zealand, therefore exposure was assessed as negligible. For example, if BTV contaminated semen is inseminated into a susceptible animal, it may or may not cause BT. Bluetongue virus cannot spread further via vectors from the inseminated animal or establish, due to the absence of the *Culicoides* spp. vector in New Zealand.

Exposure of captive animals vs animals outside captivity:
The exposure of captive animals (i.e. animals within the zoo) and animals outside captivity could not be assessed as an entity due to the separation and segregation between these environments and animals. Animals within captivity are segregated from each other and animals outside captivity by physical barriers as well as distance (i.e. no direct contact), which limits their likelihood of exposure to diseases.

However, there may be instances where domestic ruminants may be kept at the zoo. These domestic ruminants may also be kept in the same enclosures as captive wild ruminants (i.e. direct contact). Domestic ruminants may be released to the farms they originated from. Therefore, there is a likelihood that domestic ruminants at the zoo could be exposed to hazards that were brought into New Zealand via imported captive wild ruminants. Should these domestic animals be released to New Zealand farms, they could act as a source of infection for other susceptible domestic animals.

Animals outside captivity could also become infected via vertebrate or invertebrate vectors, airborne transmission and fomites.

Therefore, where domestic ruminants are not released to New Zealand farms, the likelihood of exposure was assessed as negligible for animals outside captivity. Where domestic ruminants are released to New Zealand farms, the likelihood of exposure was assessed as non-negligible for animals outside captivity.

Indirect consequences:
The indirect consequences in terms of surveillance and control were difficult to quantify. The reason for this is that the type of control measures that may be advocated for a particular disease could not be predicted or assumed at this stage. Each probable outbreak of disease or identification of an unwanted agent in New Zealand requires an individual assessment. There are numerous factors that

come under consideration before a joint decision is made by the government as to the type of control measures that are to be implemented. The consequences for a disease/agent that merely requires passive surveillance would be much lower than one that requires active surveillance and eradication with accompanying compensation costs.

Volume of trade:
The volume of trade has been included in all parts of the assessment, as it relates to the entry, exposure and consequences of an agent entering and establishing in New Zealand.

There are relatively few numbers of zoological collections in New Zealand. Most collections (with exceptions e.g. Orana Wildlife Park) are usually in the middle of urban areas and have limited capacity to house animals. Enclosures within these collections will only hold the maximum number of animals that the carrying capacity will allow, such that animals are comfortable and have adequate shelter and living space, to prevent any potential stress or health conditions that could be caused by overcrowding.

Furthermore, these collections will only have a select number of females that would be part of a breeding programme and require insemination.

As a result, the volume of live captive wild Bovidae, Giraffidae, Tragulidae and their semen that could be imported into New Zealand is likely to be very limited and infrequent.

In the entry assessment in this risk analysis the likelihood of entry of a disease is assessed as very low where the volume of trade is likely to be very low.

Additionally, the very low numbers of probably infected live captive wild species are likely to lead to a very low number of initially exposed animals. This exposure is confined due to the separation of animals within their own enclosures. The very low number of imported semen samples may only expose a very limited number of captive wild ruminant females. The likelihood that the inseminated female becomes infected would be even lower, as there is uncertainty surrounding the transmission of most agents via semen of wildlife ruminants. For example, *Coxiella burnetii* is the only agent (of those assessed) with for which there is published evidence of presence in the semen of a species within the scope of this IRA.

Finally, the consequences of exposure of a limited number of animals is likely to be very low. However, other aspects of the consequence assessment would determine the final consequence risk indicator.

Risk mitigation measures:
In New Zealand, all hazards with a non-negligible risk estimate, require risk mitigation.

Formulating risk management measures for wildlife species can be challenging. Captive wild animals are not necessarily docile and can be quite flighty if they are not habituated to human presence. A large majority of the agents under assessment are OIE-listed with accompanying OIE Code recommendations to manage the risk. Most of these recommendations include diagnostic tests for use in the international trade of domestic ruminants (unless stated specifically for wildlife).

Diagnostic testing in domestic ruminants is considered to be practical, as these animals can be handled relatively easy when obtaining diagnostic samples. Domestic ruminants are more tolerant to repeated interventions compared to captive wild animals. For wildlife species, restraint or intervention events and taking multiple samples may require general anaesthesia or special restraint equipment. Furthermore, these animals may be kept for prolonged periods in isolation facilities This is stressful for wildlife, which can predispose them to various infectious and non-infectious conditions.

The animal welfare of these captive wild animals has been taken into consideration when recommending risk management measures. Safe alternatives had to be devised to limit the number of times an animal should be tested, while still managing the risk to an acceptable level.

The handling of captive wild animals may also pose a health and safety risk for humans.

In some cases, long-term ‘premises freedom’ of the exporting facilities may help to minimise entry requirements. Combination testing was recommended in other cases, as this may be more reliable for highly sensitive animals that require limited handling (Kottwitz & Ortiz, 2016).

Very few OIE Code-recommended tests are validated for wildlife species. The use of invalidated tests is a challenge and may not produce reliable or trustworthy results.

6 Hazard identification

The preliminary list of hazards of potential biosecurity concern was compiled from:

- OIE-listed diseases, infections and infestations
- Diseases identified in previous import risk analyses of germplasm, cattle, and zoo animals conducted by MPI
- Australian animal IRA: Importation of zoo bovids and their semen from approved countries
- Fowlers: Zoo and Wild Animal Medicine, Volume 8
- American Association of Zoo Veterinarians Infectious Disease Manual
- Internet literature searches, and
- Other diseases identified as occurring in zoo bovids including emerging diseases.

Table 2. Preliminary hazard list

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Viruses										
Akabane *(Akabane virus)*	Cattle, sheep, goats	No, antibodies only	No evidence	No	No	No	Yes	No	No known competent vector in NZ; found in cattle semen	(Davies & Jessett, 1985; Orynbayev et al., 2016)
Aujeszky's disease (*Aujeszky's disease virus*)	Pigs, other mammals recorded as dead-end hosts	No	No evidence	Yes	No	Yes	Yes	No	Spill over into other species is typically fatal (dead-end hosts) after a short incubation period (2-10 days); found in pig semen	(CFSPH, 2017; Hall, 1982; OIE Terrestrial Manual, 2019a)
Bluetongue (*Bluetongue virus*)	Artiodactyla	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle semen	(Hourrigan & Klingsporn, 1975; Spickler, 2015a)
Border disease (*Border disease virus*)	Sheep and cattle	No	No evidence	No	Yes	No	Yes	No	Found in cattle semen	(Braun et al., 2019; McFadden et al., 2012)
Borna disease (*Borna disease virus 1 and 2*)	Equids, mainly sheep; cattle, camelids, dogs, cats, ostriches	No	No evidence	No	No	No	Yes	No		
Bovine ephemeral fever (*Bovine ephemeral fever virus*)	Cattle, yaks and water buffalo (*Bubalus bubalis*)	No, antibodies only	No	No	No	No	Yes	No	No evidence of role in epidemiology	(Walker & Klement, 2015)

[3] Diseases/agents were checked against "OIE-Listed disease, infections and infestations in force in 2020", retrieved at https://www.oie.int/animal-health-in-the-world/oie-listed-diseases-2020/, on 22 January 2020.
[4] If diseases/agents were OIE-listed diseases, country presence or absence was confirmed via "World Animal Health Information Database (WAHIS) Interface", retrieved from https://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statuslist on 23 August 2019.
[5] Diseases/agents were checked against "Biosecurity (Notifiable Organisms) Order 2016", retrieved from https://www.mpi.govt.nz/dmsdocument/6403/send on 23 August 2019.

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Bovine herpes (*Bovine herpesvirus 2*)	Ruminants	No	No evidence	No	Yes	No	Yes	No	Found in cattle semen	(Vermunt & Parkinson, 2000)
Bovine herpes (*Bovine herpesvirus 4*)	Ruminants	No	No evidence	No	Yes	No	Yes	No	Found in cattle semen	(de Boer et al., 2014)
Bovine herpes (*Bovine herpesvirus 5*)	Cattle	No	No evidence	No	No	No	Yes	No	Found in cattle semen	
Bovine malignant catarrhal fever – sheep-associated (*Ovine gammaherpesvirus 2)*	Sheep; spillover into other artiodactyls	No	No evidence	No	Yes	No	Yes	No	Covered in malignant catarrhal fever; found in sheep semen	(Hüssy et al., 2002)
Bovine viral diarrhoea (*Bovine viral diarrhoea 1*)	Bovidae	Yes	No evidence	Yes	Yes	No	Yes	No	Found in cattle semen	(Casaubon et al., 2012)
Bovine viral diarrhoea (*Bovine viral diarrhoea 2*)	Bovidae	Yes	No evidence	Yes	No	No	Yes	Yes	Found in cattle semen	(Casaubon et al., 2012; Doyle & Heuschele, 1983a)
Bovine viral diarrhoea (*Bovine viral diarrhoea 3/Hobi-like virus*)	Bovidae, sheep	No	No evidence	Yes?	No	No	Yes	Yes	Found in cattle semen	(Bauermann et al., 2013)
Cache Valley virus infection (*Cache Valley virus*)	Sheep primarily; deer reservoir; antibodies in goats, Cervidae, horses	No	No evidence	No	No	No	Yes	No		(Uehlinger et al., 2018)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Caprine arthritis and encephalitis (*Caprine arthritis and encephalitis virus*)	Caprinae, sheep	No	No evidence	Yes	Yes	No	Yes	No	Found in Caprinae semen	(CFSPH, 2007; Santiago-Moreno et al., 2011)
Coronavirus infection (*Bovine coronavirus*)	Bovine, giraffes	Yes	No evidence	No	Yes	No	Yes	No	Found in semen of some species	(Al Mawly et al., 2015; Alekseev. et al., 2008; Hasoksuz et al., 2007)
Cowpox (*Cowpox virus*)	Okapis, giraffes, cattle, llamas, alpacas	Yes	No	No	No	No	Yes	Yes		(Bush, 2003; Wolters & van Bolhuis, 2008)
Crimean Congo haemorrhagic fever (*Crimean Congo haemorrhagic fever virus*)	Mammals including humans	Yes	No	Yes	No	Yes	Yes	Yes		(Shepherd et al., 1987; Spickler, 2019)
Eastern, Western and Venezuelan equine encephalomyelitis (*Eastern, Western and Venezuelan equine encephalomyelitis viruses*)	Equids, humans, occasionally birds and other mammals	No	No evidence	Yes	No	Yes	Yes	No		(CABI, 2019; Spickler, 2017b)
Equine herpes (*Equine herpesvirus 1*)	Equids, artiodactyls, camelids, rhinoceroses	Yes	No evidence	Yes	Yes (strains 1P, 2, 3, 4, 5)	No	Yes	No		(Donald, 1998)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Equine herpes (*Equine herpesvirus 9/Gazelle herpesvirus 1*)	Equids, giraffes, gazelles, artiodactyls, camelids, rhinoceroses	Yes	No evidence	No	Not reported	No	Yes	No		(Donald, 1998; Kasem et al., 2008)
Enzootic bovine leukosis (*Bovine leukaemia virus*)	Cattle primarily, sheep, water buffalo, capybaras, yaks	No	No	Yes	Yes	Yes	Yes	No	New Zealand dairy herd is free – lasted reported case in 2008; found in reproductive tract only, not semen	(More et al., 2017)
Epizootic haemorrhagic disease (*Epizootic haemorrhagic disease virus*)	Cervidae primarily, cattle, Caprinae, occasionally other species including rhinos, black bears, oryxes, etc.	Yes	No evidence	Yes	No	Yes	Yes	Yes		(Frölich et al., 2005; Spickler, 2006)
Flavivirus encephalitis (*Flaviviruses*)	Ruminants, goats, sheep, cows, dogs	Uncertain	No evidence	No	No	No	Yes	No	Mammals other than rodents are spillover dead-end hosts; large ruminants serve as feeding hosts for ticks only	(Thompson et al., 2012)
Foot-and-mouth disease (*Foot-and-mouth disease virus*)	Artiodactyls	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle and buffalo semen	(Bastos et al., 2000)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Getah virus infection (*Getah virus*)	Horses, pigs, other warm-blooded species, including humans occasionally	No	No evidence	No	No	No	Yes	No	Rare spillover events into multiple species types (including humans)	(Shi et al., 2019; Spickler, 2017c)
Infectious bovine rhinotracheitis (*Bovine herpesvirus 1*)	Cattle, sheep, goats, wild artiodactyls	Yes	No evidence	Yes	Yes, BHV-1.2b present	Yes	Yes	Yes	Subtypes 1.1 and 1.2a are absent; found in cattle semen	(Anderson & Rowe, 1998; Doyle & Heuschele, 1983b)
Jembrana disease (*Jembrana disease virus*)	Cattle (*Bos* spp.) and water buffalo	No	No evidence	No	No	No	No	No		(Desport & Lewis, 2010)
Louping ill (*Louping ill virus*)	Sheep, grouse, other warm-blooded species occasionally infected	No	No evidence	No	No	No	Yes	No	No competent vector in New Zealand	(Jeffries et al., 2014; Spickler, 2009a)
Lumpy skin disease (*Lumpy skin disease virus*)	Cattle, wild artiodactyla, occasionally sheep and goats	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle semen	(Davies, 2019; Young et al., 1970)
Maedi-visna (*Maedi-visna virus*)	Sheep and goats primarily, occasionally wild Caprinae species	No	No evidence	Yes	No	Yes	Yes	No	Found in sheep semen	(MacDiarmid, 1988; Ruiz-Fons et al., 2014; Sanjosé et al., 2016)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Malignant catarrhal fever – wildebeest-associated (*Alcelaphine gammaherpesvirus 1*)	Artiodactyla	Yes	No evidence	No	No	No	Yes	Yes		(Horner, 1996; Horner & Tham, 2003)
MERS-CoV infection (*Middle East respiratory syndrome-related coronavirus*)	Dromedary camels, humans	No	No evidence	No	No	No	Yes	No		(Omrani et al., 2015)
Nairobi sheep disease (*Nairobi sheep disease orthonairovirus*)	Sheep, goats primarily, duikers	Yes	No	Yes	No	Yes	Uncertain	Yes	Tick vector present in New Zealand	(CABI, 2017; Spickler, 2016a)
Ovine pulmonary adenomatosis (*Jaagsiekte sheep retrovirus*)	Sheep, rarely goats	No	No	No	No	Yes	Yes	No		(Spickler, 2009b)
Papillomatosis (*Bovine papillomavirus*)	Giraffe, sable antelope	Yes	No evidence	No	Yes	No	Yes	No	Found in cattle semen	(van Dyk et al., 2011)
Peste des petits ruminants (*Peste des petits ruminants virus*)	Goats, sheep, wild artiodactyla	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in sheep semen	(Kinne et al., 2010)
Pestivirus infection (*Pestivirus Giraffe 1*)	Giraffes	Yes	No evidence	No	No	No	No	No	Other pestiviruses are found in semen (e.g. BVDV)	(Harasawa et al., 2000)
Rabies (*Rabies lyssavirus*)	All mammals including humans	Yes	No evidence	Yes	No	Yes	Yes	Yes		(Hikufe et al., 2019)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Rift Valley fever (*Rift Valley fever virus*)	Artiodactyla, primates, rodents, humans	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in sheep semen	(Dondona et al., 2016; Rostal et al., 2017)
Rinderpest (*Rinderpest morbillivirus*)	Wide range of artiodactyla	Yes	Yes	Yes	No	Yes	No	No	Eradicated from the world	(OIE Technical Disease Cards, 2013d)
Rotavirus infections (*Bovine rotaviruses*)	Mammals	Yes	No	No	Yes	No	Yes	No		(Al Mawly et al., 2015; ESR, 2017)
Schmallenberg virus infection (*Schmallenberg virus*)	Ruminants, pigs, camelids, elephants, perissodactyls, deer, elks	No, antibodies only	No evidence	No	No	No	Yes	No	Emerging disease of concern; zoo bovid role in epidemiology uncertain; no known competent vectors in New Zealand; found in cattle semen	(EFSA, 2014; Larska et al., 2013; Mouchantat et al., 2015; MPI, 2013; Ponsart et al., 2014)
Sheep and goat pox (*Sheep pox virus* and *Goat pox virus*)	Caprine species, sheep	No	No evidence	Yes	No	Yes	No	No	Found in sheep semen	(EFSA AHAW Panel, 2014)
Lentivirus infection *(Small ruminant lentivirus)*	Sheep, goats, Caprinae subfamily	No	No evidence	No	No	No	Yes	No	Found in Caprinae semen	(Minardi da Cruz, J. C et al., 2013)
Transmissible gastroenteritis (*Transmissible gastroenteritis virus*)	Pigs	No	No	Yes	No	Yes	Yes	No		(Iowa State University College of Veterinary Medicine, 2019)
Vesicular exanthema (*Vesicular exanthema of swine virus*)	Pigs	No	No evidence	No	No	Yes	No	No		(Horak et al., 2016)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Vesicular stomatitis (*Vesicular stomatitis virus*)	Artiodactyla, horses, camelids, humans	No	No	No	No	Yes	Yes	No		(OIE, 2013; Spickler, 2016b)
Wesselsbron disease (*Wesselsbron virus*)	Sheep, goats, rodents, birds, pigs, humans, wild artiodactyla	No, antibodies only	No evidence	No	No	Yes	Yes	No, no evidence of role in epidemiology	Found in sheep semen	(Barnard, 1997; Spickler, 2017f)
West Nile virus infection (*West Nile virus*)	Horses, humans, birds, occasionally other mammals	No	No evidence	Yes	No	Yes	Yes	No	Most mammals are dead-end hosts that cannot transmit the virus to mosquitoes.	(Root & Bosco-Lauth, 2019; Spickler, 2013)
Bacteria and Mollicutes										
Anthrax (*Bacillus anthracis*)	Mammals and some birds	Yes	No	Yes	No	Yes	Yes	Yes		(Pienaar, 1961)
Bovine brucellosis (*Brucella abortus*)	Wide range of mammals	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle and bison semen	(Godfroid, 2002)
Bovine genital campylobacteriosis (*Campylobacter fetus* subsp. *venerealis*)	Cattle	No	No evidence	Yes	Unknown	Yes	Yes	No	Last reported in New Zealand 1992 (passive surveillance); *Cff* present in New Zealand; Found in cattle semen	(McFadden & Heuer, 2011)
Bovine tuberculosis *(Mycobacterium bovis)*	Wide range of mammals	Yes	No evidence	Yes	Yes	No	Yes	Yes	National control programme in New Zealand; found in cattle semen	(Cousins, 2001; Govender, 2013; Krajewska-Wędzina et al., 2018)
Caprine and ovine brucellosis (*Brucella melitensis*)	Wide range of mammals	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle semen	(Spickler, 2018b)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Clostridium perfringens infection (*Clostridium perfringens*)	Livestock, domestic animals	Yes	No evidence	No	Yes	No	Yes	No		(Milton et al., 2017)
Contagious agalactia (*Mycoplasma agalactiae*)	Sheep, goats, some wild Caprinae	No	No evidence	Yes	No	Yes	Yes	Yes	Reported only in ibex and chamois – Caprinae subfamily and semen of Caprinae	(Verbisck et al., 2010)
Contagious bovine pleuropneumonia (*Mycoplasma mycoides* subsp. *mycoides* SC)	Bovids from *Bos* and *Bubalus* genera	No	No evidence	Yes	No	Yes	Yes	Yes	Last detected in New Zealand in 1864; found in cattle semen	(Spickler, 2015b)
Contagious caprine pleuropneumonia (*Mycoplasma capricolum* subsp. *capripneumoniae*)	Goats and other non-domestic Bovidae, occasionally sheep	Yes	No evidence	Yes	No	Yes	Yes	Yes	Found in cattle semen	(Arif et al., 2007)
Dermatophilosis (*Dermatophilus congolensis*)	Cattle, sheep, goats, horses, less frequently pigs, dogs, cats	Yes	No	No	Yes	No	Yes	No		(CFSPH, 2006a; Pastoret et al., 1988; Pennsylvania Game Commission, 2019a; Smith et al., 1967)
Enzootic abortion of ewes (*Chlamydophila abortus)*	Goats, sheep, less commonly cattle, pigs, horses, deer	No	No evidence	Yes	No	Yes	Yes	No	Found in cattle, sheep and goat semen	(Spickler, 2017a; Teankum et al., 2007)
Haemorrhagic septicaemia (*Pasteurella multocida* serotypes B2 6:B and E2 6:E)	Ruminants, camels, deer, hares, horses, pigs, elephants, birds	Yes	No evidence	Yes	Yes (A, B, D)	Yes	Yes	Yes	Found in dog semen	(Bjurström & Linde-Forsberg, 1992; Jones, 2018)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Legionellosis (*Legionella* spp.)	Wide range of domestic and wild mammals, humans	Yes	No evidence	No	Yes	No	Yes	No		(Boldur et al., 1987; Massimo et al., 1998)
Leptospirosis (*Leptospira* spp.)	Wide range of mammals	Yes	Yes	No	Yes (serovars Hardjo, Pomona, Copenhageni, Ballum and Tarassovi)	No	Yes	No	Few wild animal reservoirs have been identified, and wild bovids have a low seroprevalence.	(Biosecurity Australia, 2019; Leptospirosis New Zealand, 2019)
Listeriosis (*Listeria monocytogenes*)	Wide range of domestic and wild mammals	Yes	No evidence	No	Yes	No	Yes	No		(CFSPH, 2006b; Pennsylvania Game Commission, 2019b)
Lyme disease (*Borrelia burgdorferi*)	Wild mammals, dogs, horses, humans, birds, rodents, lizards	No	No	No	No	No	Yes	No	No competent tick vectors known in New Zealand; ruminants rarely infected	(Parker & White, 1992; Spickler, 2011)
Mycoplasma bovis infection (*Mycoplasma bovis*)	Cattle, sheep and goats	No	No evidence	No	Yes (National eradication programme in place)	No	Yes	Yes	Due to national eradication programme *M. bovis* will be retained for assessment; evidence in cattle semen	M. bovis IRA
Ovine epididymitis (*Brucella ovis*)	Sheep	No	No evidence	Yes	Yes	No	Yes	No	Found in sheep semen	(Picard-Hagen et al., 2015; Reichel & West, 1997)
Paratuberculosis /Johne's disease (*Mycobacterium avium* subsp. *paratuberculosis*)	Ruminants	Yes	No evidence	Yes	Yes	No	Yes	No	Found in cattle semen	(Larsen et al., 1981; National Research Council, 2003)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Salmonellosis (*S. abortus-equi*)	Equids	No	No evidence	No	No	Yes (exotic strains)	Yes	No		
Salmonellosis (*S. abortus-ovis*)	Sheep primarily; occasionally goats and rabbits	No	No evidence	Yes	No	Yes (exotic strains)	Yes	No		
Tuberculosis *(Mycobacterium tuberculosis)*	Wide range of mammals, humans and non-human primates primarily	Yes	Yes	No	Yes	No	Yes	No	Disease present in New Zealand in humans; no control measures for *M. tb* in animals; Bovidae species are an unlikely source for *M. tb.*	(Govender, 2013; Ministry of Health, 2019)
Tularaemia (*Francisella tularensis)*	Wide range of mammals including humans	No	No evidence	Yes	No	Yes	Yes	No		(CFSPH, 2013)
Yersinia (*Yersinia pestis, Y. enterocolitica, Y. pseudotuberculosis*)	Many mammal species including humans; ungulates are highly resistant to infection	No	No evidence	No	Yes	No	Yes	No	*Y. pestis* absent from New Zealand (last reported 1900 and 1911)	(Gill, 1996)
Rickettsia										
Anaplasmosis (*Anaplasma bovis* formerly *Ehrlichia bovis*)	Ruminants	Gemsbok?	No	No	No	Yes	Yes	No	Gene sequence in gemsboks similar to *A. bovis*	(Harrison et al., 2013; Tonetti et al., 2009)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Bovine anaplasmosis *(Anaplasma marginale, A. centrale, A. caudatum)*	Ruminants including non-domestic Bovidae	Yes	No	Yes	No	Yes	Yes	Yes		(Brandt, 2009; Kuttler, 1984)
Heartwater (*Ehrlichia ruminantium*)	Many species, ruminants including wildlife	Yes	No	Yes	No	Yes	Yes	Yes		(Spickler, 2015c)
Q fever *(Coxiella burnetii)*	Wide range of mammals	Yes	Yes	Yes	No	Yes	Yes	Yes		(Clemente et al., 2008; Garcia-Seco et al., 2016)
Protozoa										
Besnoitia (*Besnoitia besnoiti*)	Cattle and other wild ruminants	Yes	No	No	No	No	Yes	Yes		(McCully et al., 1966)
Bovine babesiosis (*Babesia bovis*, *B. bigemina, B. divergens*)	Ruminants including non-domestic Bovidae	Yes	No	Yes	No	Yes	Yes	Yes		(Cardenas-Canales et al., 2011)
Chagas' disease *(Trypanosoma cruzi*)	Mammals, but dogs and humans most often; pigs, cats	No	No	No	No	Yes	Yes	No		(Jansen et al., 2018)
Coccidiosis (*Eimeria* spp.)	Wide range of mammals	Yes	No	No	Yes	No	Yes	No		(Erber et al., 1984)
Cryptosporidiosis (*Cryptosporidium* spp.)	Mammals	Yes	No	No	Yes	No	Yes	No		(Geurden et al., 2009)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Cytauxzoonosis (*Cytauxzoon* spp.)	Kudu, elands, giraffes	Yes	No	No	No	No	Yes	No	No competent vector in New Zealand	(Nentwig et al., 2018) Nentwig
Leishmaniasis (*Leishmania* spp.)	Humans and dogs primarily; occasional reports in other species	No	No evidence	Yes	No	Yes	Yes	No	No competent vector for leishmaniasis in New Zealand; infection detected only in imported animals; Found in dog semen	(Diniz et al., 2005; Spickler, 2017d)
Sarcocystis (*Sarcocystis* spp.)	Mammals, humans	Yes	No	No	Yes	No	Yes	No		(Böttner et al., 1987; Fayer et al., 1982)
Surra (*Trypanosoma evansi*)	Mainly equids, camels, occasionally Bovidae	No	No	Yes	No	Yes	Yes	Yes	Vectors include biting insects	(Spickler, 2015d)
Theileriosis *(Theileria annulata* and *T. parva*)	Ruminants including cattle, buffalo, other antelopes	Yes	No	Yes	No	Yes	Yes	Yes		(Dolan, 1989)
Toxoplasmosis (*Toxoplasma gondii*)	Mammals	No	No evidence	No	Yes	No	Yes	No	Found in dog, human, cattle semen	(Spickler, 2017e)
Trichomonosis (*Tritrichomonas foetus)*	Cattle, cats	No	No evidence	Yes	Yes	No	Yes	No	Found in cattle semen	(Armstrong, 2016; Kingsbury et al., 2010)
Tsetse fly associated trypanosomosis (*Trypanosoma brucei, T. vivax*, etc.)	Wide range of mammals including non-domestic Bovidae	Yes	No	Yes	No	Yes	Yes	Yes	No competent vectors (Glossina) in New Zealand; limited mechanical transmission	(Mulla & Rickman, 1988; Osório, Ana Luiza Alves Rosa et al., 2008; Spickler, 2018a)

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Endoparasites										
Cysticercus bovis (*Taenia saginata*)	Wide range of mammals	Yes	No	No	Yes	Yes	Yes	No	Zoo Bovidae are dead-end hosts; no access to definitive, carnivore hosts	(Graber, 1974; Spickler, 2005)
Echinococcus *(Echinococcus granulosus, E. multilocularis)*	Cattle and multiple other species including humans	Probably	No	Yes	No (declared freedom in 2002)	Yes	Yes	No	Zoo Bovidae are dead-end hosts; no access to definitive, carnivore hosts	(MPI, 2018; OIE, 2019)
Internal parasites	Wide range of mammals	Yes	No	No	Yes	Selected species	Yes	Yes		
Trichinellosis (*Trichinella spiralis*)	Canids, pigs, horses, other carnivorous mammals	No	No	Yes	Yes	Yes	Yes	No		(Mason, 1978)
Ectoparasites										
External parasites	Wide range of mammals	Yes	No	No	Yes	Specific species	Yes	Yes		
Giraffe skin disease (unknown)	Giraffe	Yes	No	No	No	No	No	No	Unknown aetiology	(Lee & Bond, 2016)
Screw-worm fly - New World (*Cochliomyia hominivorax*)	All mammals including humans	Yes	No	Yes	No	Yes	No (Japan and Europe uncertain	Yes	Agent of significant consequence; resistant to many common anti-parasitics	
Screw-worm fly - Old World (*Chrysomya bezziana*)	All mammals including humans	Yes	No	Yes	No	Yes	Yes	Yes	Agent of significant consequence; resistant to many common anti-parasitics.	

Disease (*disease agent*)	Susceptible species	Do captive wild Bovidae, Giraffidae and Tragulidae play a role in epidemiology?	Evidence in semen of captive wild Bovidae, Giraffidae and Tragulidae	Is it an OIE-listed disease?[3]	Is it present in New Zealand?[4]	Is it nationally notifiable in New Zealand?[5]	Is it present in approved countries?	Has it been retained for risk assessment?	Comments	References
Sheep scab (*Psoroptes ovis*)	Sheep primarily	Yes	No	No	No	Yes	Yes	No	Covered under external parasites	(MPI, 2009; Spickler, 2009c)
Warble-fly myiasis (*Hypoderma* spp.)	Wide range of mammals	Yes	No	No	No	No	Yes	No	Covered under external parasites	
Prion										
Transmissible spongiform encephalopathies (prions)	Wide range of mammals	Yes	No	Yes	No	Yes	Yes	Yes		(Kirkwood & Cunningham, 1994; Sigurdson & Miller, 2003)
Miscellaneous										
Seeds								Yes		

7 Bluetongue

7.1 Technical review

7.1.1 Aetiological agent

Family: *Reoviridae*
Genus: *Orbivirus*
Species: *Bluetongue virus* (BTV) (ICTV, 2019)

Bluetongue virus (BTV) is the causative agent of bluetongue (BT).

To date, there are 26 distinct serotypes of BTV recognised worldwide (Gaudreault et al., 2014).

7.1.2 OIE list

Bluetongue is an OIE-listed disease affecting multiple species (OIE, 2020d).

7.1.3 New Zealand status

New Zealand is free from all serotypes of BTV (WAHIS, 2019d), as reported to the OIE.

Bluetongue is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

7.1.4 Zoonotic potential

Bluetongue is not a zoonotic disease.

7.1.5 Epidemiology

Host range

Bluetongue is enzootic in wild ruminants in large parts of Africa and North America (Gerdes, 2004). Experimental infections of various African antelope (blesbok and mountain gazelle) have been demonstrated (Bengis & Erasmus, 1988; Hoff & Hoff, 1976; Shimshony et al., 1988). Clinical BT was recorded after experimental infections of American bison and Cape buffalo (Tessaro & Clavijo, 2001). Disease may also be present under natural conditions in wapiti, axis deer, fallow deer, sika deer, musk deer, roe deer (Cervidae), Spanish ibex and captive yak (Bovidae) (Fernández-Pacheco et al., 2008; Ruiz-Fons et al., 2008).

In 1997, a serosurvey of 24 species of wild African ruminants was conducted. Of those sampled, 10 tested positive for BTV antibodies. Species with the highest seroprevalences included the blue and black wildebeest and buffalo. Other species that tested positive included red hartebeest, impala, springbok, eland, blesbok, gazelle and giraffe (Barnard, 1997).

There are various studies to investigate the role of wildlife in the epidemiology of BT. Fernandez-Pacheco et al. (2008) demonstrated that wild sheep such as bighorn and mouflon (Caprinae) are susceptible to BTV infection and can develop fatal clinical disease (Fernández-Pacheco et al., 2008).

There is no published evidence of BT in species of the Tragulidae families.

Captive wild ruminants

During the 2007 and 2008 BT epizootics in Europe, numerous zoos were affected. In 2008 a survey of 313 European zoos was conducted. Of these, 49 zoos within a 20-kilometre radius, had confirmed BT cases. These zoos held over 1000 susceptible individuals of 53 different species and 7 ruminant families indigenous to Europe, North and South American, Africa and Asia. Bluetongue affected 62 individuals in the Bovidae, Cervidae and Camelidae families (Sanderson, 2019).

Geographical distribution

Bluetongue virus is enzootic in many tropical, subtropical and temperate regions of the world, including America, Australia, Africa and some regions of Asia, during times of the year that are optimal for vector activity (Mellor et al., 2000).

Historically, Europe has experienced only sporadic outbreaks of BT (Mellor & Boorman, 1995). However, since 1998, BTV has spread northwards, potentially due to climate change and vector expansion. Outbreaks have since been occurring in the Netherlands, Belgium, Germany, France, Luxembourg (Wilson & Mellor, 2009) and Italy.

There are areas in Europe that are seasonally vector free. The European commission have annual published dates for "bluetongue seasonally vector free periods"[6] for various European countries. The most up-to-date dates can be found on their website.

In North America, BT is seasonally absent in the central and northwestern states. The northern and northeastern regions are BTV-free (USDA, 2016a).

Subject to its continued adherence to the OIE Code, Canada is recognised by the European Commission as seasonally free for bluetongue between 1 November and 15 May (European Commission, 2019).

Pathogenesis

Bluetongue is a non-contagious, vector-borne, viral disease of domestic and wildlife ruminants.

Following the bite of a *Culicoides* spp. midge, BTV multiples in the regional lymph node prior to spreading to the rest of the body. Bluetongue virus appears in circulation 3 to 6 days after infection.

Viral replication occurs in endothelial cells and pericytes of capillaries and small blood vessels. Cells undergo degeneration and necrosis with accompanying hyperplasia of the endothelium causing vascular occlusion, stasis and exudation, finally resulting in hypoxia, oedema and haemorrhage and secondary lesions in the overlying epithelium.

Severity of secondary lesions is influenced by mechanical stress and abrasions. Severe lesions often occur in tissues exposed to the environment such as oral mucosa and skin of the coronary band of hooves (Verwoerd & Erasmus, 2004).

The virulence of BTV and severity of BT is not solely dependent on the BTV serotype, but rather a combination of the viral serotype, host, vector and environmental factors (Gaudreault et al., 2014).

The incubation period ranges from 4 to 10 days in domestic ruminants (OIE Technical Disease Cards, 2013a) and may be similar in wildlife ruminants. Studies have reported varying incubation periods in different species as well as in different breeds of the same species.

The duration of viraemia that resulted in *Culicoides* spp. being infected was recorded as a maximum of 21 days post infection in both cattle and sheep. Data from this study suggested that viraemia in domestic ruminants is transient (Bonneau et al., 2002).

In the study by Gard and Melville (1992), the longest duration of viraemia detected was 36 days, when BTV was isolated from a sentinel steer for 6 consecutive weeks (Gard & Melville, 1992).

In another study, viraemia of cattle was reported for up to 100 days (Sperlova & Zendulkova, 2011). In some instances, this may be due to latent BTV infections (Luedke et al., 1977).

In the study by Koumbati et al. (1999), European breeds of sheep and goats had detectable viraemias that lasted from 3 to 6 days until 27 to 54 days post infection (Koumbati et al., 1999).

Experimental infections in gazelles reported viraemia of up to 35 days post infection (Shimshony et al., 1988).

[6] Bluetongue Seasonally Vector free periods 2018 – 2019: https://ec.europa.eu/food/sites/food/files/animals/docs/ad_control-measures_bt_overview_seasonally_vfp_2018-2019.pdf

The OIE Code states that the infective period for BTV is 60 days in ruminants.

Clinical signs

Bluetongue in susceptible wildlife ruminants may lead to a wide variety of lesions and clinical manifestations, ranging from subclinical infections to sudden death (Howerth et al., 2001).

There is a large degree of variability relating to clinical disease in domestic ruminants (sheep).

The disease course can vary from peracute to chronic.

In peracute cases, animals may die within 7 to 9 days from lung oedema and asphyxiation, in the absence of many premonitory signs.

In chronic cases, death may result from secondary bacterial infection and exhaustion or protracted recovery may occur.

Many infections in sheep are clinically inapparent. Mild cases recover rapidly and completely.

Mortality rates in sheep may range from 2% to 30%.

Clinical signs in cattle are rare and may include a transient fever, increased respiration, lachrymation and salivation, stiffness and inflammatory changes of the skin (Verwoerd & Erasmus, 2004).

Experimental infections of various African antelope (blesbok and mountain gazelle) demonstrated that they do not develop clinical disease but rather experience subclinical infections (Bengis & Erasmus, 1988; Hoff & Hoff, 1976; Shimshony et al., 1988).

Bluetongue under natural settings in various African wildlife ruminants is subclinical, and the virus has been isolated from several species of antelope including addax, Nubian ibex, sable antelope and Cape buffalo (Verwoerd & Erasmus, 1994).

During the 2007 and 2008 BT epizootics in Europe where numerous zoos were affected, clinical disease was noted in 55 individuals of the Bovidae family (Sanderson, 2019).

Mortality and morbidity rates were reported on, from data collated from zoos during the 2007/2008 European epizootic. The Bovidae family were most susceptible to clinical disease, with 4 species showing morbidity rates of greater than 20% and mortality rates of greater than 10%. The average case fatality rate for the affected Bovidae species was 69%. Ruminant species affected were indigenous to Europe, Asia or South America (Sanderson, 2019). However, of over 200 African wildlife ruminants (20 different species) held by the zoos in at-risk areas, none showed clinical signs of disease.

Transmission

Bluetongue virus is transmitted by biting midges of the genus *Culicoides* (Mellor et al., 2000). Transmission of BTV in wild ruminants is almost exclusively dependent on midges in the environment.

Other suggested methods of transmission include transplacental, oral (including colostrum), semen and mechanical means (wounds) (Menzies et al., 2008; Santiago-Moreno et al., 2011).

Other arthropods such as sheep keds, cattle lice, ticks and mosquitoes may play a role in mechanical transmission, however this may be of minor significance. *Bluetongue virus* can be spread mechanically via surgical equipment and needles. Some field strains and attenuated vaccine strains have reported to infect the foetus *in utero* (Savini et al., 2014).

Experimental studies have demonstrated that ixodid and argasid ticks have potential vectorial capacity for BTV. After ticks ingested BTV, the virus was found in various tissues for up to 21 to 26 days after feeding. *Bluetongue virus* was also found in adult ticks after moulting and in eggs. This suggests that transstadial and transovarial passage in ticks is likely (Bouwknegt et al., 2010).

In Europe, BT prevalence and outbreaks usually follow vector seasonal patterns. Outbreaks are dependent on *Culicoides* vector abundance and vectors are dependent on climatic changes such humidity and temperature (Sleeman et al., 2009).

Interannual cycles of BT outbreaks occur in North America. In enzootic areas, animals are infected in 1 to 3 year cycles, whereas in epizootic zones, BT occurs in 8 to 10 year cycles. These cycles are a combination of the effects of herd immunity and vector abundance (Howerth et al., 2001).

In New Zealand, the arbovirus surveillance programme was put in place in 1991. The programme provides assurance of New Zealand's freedom from arboviruses. The surveillance strategy includes 3 components namely:

- an early warning system for reporting suspicious cases,
- herd testing, and
- vector surveillance (Peacock et al., 2019).

The 2018 statistics include 638 blood samples from cattle in areas that are most suitable for the survival and establishment of *Culicoides* spp. All samples were seronegative for BTV and other arboviruses (Peacock et al., 2019). In the vector surveillance, 285 464 insects were analysed and no *Culicoides* spp. were found (Peacock et al., 2018).

In the 2019 vector surveillance, 136 632 insects were analysed and no *Culicoides* spp. were found (Peacock et al., 2019).

In 2020, 640 blood samples from cattle in Northland, Auckland, Waikato and Bay of Plenty were seronegative for BTV and other arboviruses. In the vector surveillance, 505 876 insects were analysed and no *Culicoides* spp. were found (Peacock et al., 2020).

The results of the arbovirus surveillance demonstrate that New Zealand is free of BTV as well as the *Culicoides* spp. vector.

Diagnosis

The recommended methods for agent identification for clinical cases and individual animal freedom from infection prior to movement are RT-PCR and virus isolation.

C-ELISA and viral neutralisation is recommended for the detection of immunity for individual animal freedom from infection prior to movement.

Other diagnostic methods include agar gel immunodiffusion (AGID) and the complement fixation test (CFT) (OIE Terrestrial Manual, 2018c).

Treatment, control and prevention

There is no effective treatment for BT.

Vector control is instituted in BT-infected areas.

Vaccination is another means of protection for susceptible animals. Live attenuated and killed BTV vaccines are available for use in domestic animals, with attenuated vaccines being serotype-specific. Animals should be vaccinated with the same serotype as those causing infection in the area. Attenuated vaccine strains have the ability to re-assort with field strains when transmitted to unvaccinated animals, resulting in new viral strains (Batten et al., 2008). Recombinant BTV vaccines are under development.

In 2009, as a control strategy, over 2000 individuals of 57 species, in 47 zoos, in 9 European countries, were vaccinated for BTV 8 (Sanderson, 2019).

In disease-free areas, preventative measures include animal movement control, quarantine, serological surveys and vector control.

Semen

An experimental study in Australia of both young and old bulls revealed the presence of BTV (serotypes 1 and 23) in semen. The difference noted was the absence of BTV in the semen of young

bulls that were both naturally and experimentally infected. *Bluetongue virus* was detected in the semen of old experimentally infected bulls. Detection of BTV occurred during or immediately after the period of detectable viraemia (Kirkland, 2004).

Osburn (1994) reported the successful isolation of BTV from semen of 2 out of 18,000 bull semen samples after natural BTV infection, indicating that the presence of BTV field strains in semen is likely, but quite rare (Osburn, 1994).

Semen collected during the viraemic period from experimentally BTV-infected bulls was inoculated transcervically into 9 naïve heifers. Three heifers became viraemic and developed antibodies to BTV. One infected heifer became pregnant. Evidence of BTV fetal infection was absent (Bowen & Howard, 1984).

In the study by Leemans et al. (2012), BTV 8 was isolated from naturally infected rams. *Bluetongue virus* could be detected in the semen of some flocks at 25 to 57 days post-observation and other flocks for up to 116 days post-observation (Leemans et al., 2012).

There is no published evidence demonstrating the presence of BTV in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with BTV-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

7.1.6 Hazard identification conclusion

Bluetongue is an OIE-listed disease affecting multiple species. Bluetongue affects various species within the scope of this IRA.

Bluetongue virus has been isolated from the semen of domestic ruminants. In the absence of evidence in wildlife ruminants, extrapolation is made to species within the scope of this IRA.

Bluetongue virus is identified as a hazard in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

7.2 Risk assessment

7.2.1 Entry assessment

Bluetongue has been reported in all approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

Experimental infections of African antelope species covered in this IRA have shown that these animals become subclinically infected. Serological surveys have also demonstrated seropositivity for BTV in numerous other species covered in this IRA.

Zoos in Europe suffered clinical cases of BTV in domestic and captive wild Bovidae species during the BT epizootic.

The fact that some species within the scope of this IRA are likely to become subclinically infected suggests that they may go unnoticed and be passed as clinically sound for export or as donor males for semen collection if they are being exported from BT-affected countries or zones.

Bluetongue virus has been isolated from the semen of viraemic domestic Bovidae. Experimental studies have shown that BTV can be transmitted to naïve heifers via artificial insemination (AI) with BTV-contaminated semen. There are currently no literature reports of BTV in the semen of wildlife ruminants or transmission via AI. Thus, extrapolation is made to species within this IRA.

Bluetongue is an OIE-listed disease, and therefore, approved, licenced premises with valuable exotic species are likely to have biosecurity measures to protect their animals from BTV. Some European zoos were vaccinating susceptible species during the epizootic. As New Zealand is free from BT,

importing BTV-vaccinated animals is not recommended. However, importing BTV-vaccinated animals would not affect New Zealand's BT-free status.

Therefore, the likelihood of entry of BTV via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) and their semen from BTV-affected countries is assessed as moderate.

The likelihood of entry of BTV via semen of captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from BTV-affected countries is assessed as very low.

7.2.2 Exposure assessment

The most significant transmission route of BTV is via *Culicoides* midges. Other routes include transplacental, oral (including colostrum), semen and mechanical means (wound). Other arthropods such as sheep keds, cattle lice, ticks and mosquitoes may play a minor role in mechanical transmission. However, this route is likely to be insignificant. *Bluetongue virus* can be spread mechanically via surgical equipment and needles. Some field strains and attenuated vaccine strains have been reported to infect the fetus in utero.

If subclinically infected animals are imported into zoos they would serve as a source of infection for other susceptible animals. However, as New Zealand is free of *Culicoides*, which are the main BTV vector, transmission via this route would not be possible. *Bluetongue virus* is not contagious, and therefore, BTV-infected animals cannot directly infect in-contact animals. Transmission via mechanical means is likely to be negligible.

The European BT outbreaks between 2007 and 2008, in which numerous captive wild animals were affected, were caused by BTV transmitted by *Culicoides*. In the absence of this vector, the likelihood of spread outside of the New Zealand zoos to susceptible ruminants would be negligible.

In the absence of the primary vector, *Culicoides* spp., BTV would only affect the imported, already infected species. Other routes of mechanical transmission by other arthropods may play a very minor role of transmission to other susceptible species but are assessed as negligible. Therefore, BTV is unlikely to establish within or outside the zoo in domestic or wildlife ruminant populations.

If *Bluetongue virus*-contaminated semen is inadvertently imported, it may be inseminated into susceptible captive wild ruminants. Artificial insemination with contaminated semen has been proven to transmit BTV infections to naïve domestic ruminants. There is uncertainty as to whether BTV-infected wildlife ruminants are likely to produce BTV-contaminated semen. It is also not known whether naïve wildlife ruminants will become infected with BTV if BTV-contaminated semen is used for AI. However, by extrapolating the outcomes from domestic ruminants to wildlife ruminants, it is assumed that infection may occur.

The number of captive wild ruminants in New Zealand zoos is very low in comparison to the domestic and wild ruminant populations. Therefore, the number of captive wild animals that would be inseminated would be very low. If these animals become infected, they could either suffer clinical disease and die, have mild signs or be subclinically infected. In the outbreaks in European zoos, mortality in European, Asian and South American ruminants was only 10%, while African ruminants were subclinically infected.

However, irrespective of the number of animals inseminated and that may become infected, transmission of BTV from these inseminated animals to susceptible animals, establishment and further spread is still assessed as negligible, due to the absence of a competent vector in New Zealand.

Article 8.3.3 in the OIE Code chapter on bluetongue (Country or zone free from bluetongue) states: "*A country or zone free from bluetongue in which ongoing vector surveillance, performed in accordance with point 5) of Article 8.3.16., has found no Culicoides will not lose its free status through the introduction of vaccinated, seropositive or infective ruminants or camelids, or their semen or embryos from infected countries or zones*".

Results from the arbovirus surveillance programme over the previous 2 years demonstrated BTV and *Culicoides* spp. freedom in New Zealand.

The outcome of this exposure assessment is based on these results. Ongoing active surveillance would be necessary to provide assurance of New Zealand's BTV and *Culicoides* spp. freedom status. Should results of future surveillance activities indicate entry and establishment of BTV or *Culicoides* spp., a review of this assessment would be required.

Therefore, the likelihood of BTV exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as negligible, the likelihood of exposure and establishment outside the zoo is assessed as negligible, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae, Giraffidae and Tragulidae is assessed as negligible.

7.2.3 Risk estimation

Since the exposure is assessed as negligible, the risk estimate for BTV is negligible, and it is not a risk in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

Therefore, risk management measures are not warranted.

8 Bovine viral diarrhoea

8.1 Technical review

8.1.1 Aetiological agent

Family: *Flaviviridae*
Genus: *Pestivirus*
Species: *Bovine viral diarrhoea virus 1* (BVDV 1), *Bovine viral diarrhoea virus 2* (BVDV 2), (Booth et al., 1995) and *Bovine viral diarrhoea virus 3* (HoBi-like virus, BVDV 3 or atypical pestivirus) (Bauermann et al., 2013)

Bovine viral diarrhoea virus 1, BVDV 2 and BVDV 3 are the causative agents of bovine viral diarrhoea (BVD).

Cytopathic and non-cytopathic biotypes (strains) occur in all 3 species, all of which are capable of infecting domestic and wildlife ruminants (Kottwitz & Ortiz, 2016).

Bovine viral diarrhoea virus 3 is a new putative pestivirus species, tentatively called “HoBi-like”, “BVDV 3”, or “atypical pestiviruses”. *Bovine viral diarrhoea virus 3* is related to BVDV at the genetic and antigenic levels (Bauermann et al., 2013).

8.1.2 OIE list

Bovine viral diarrhoea is an OIE-listed disease of cattle.

8.1.3 New Zealand status

Bovine viral diarrhoea virus 1 is enzootic in New Zealand and will not be assessed further.

Reviewing the literature shows that BVDV 2 and BVDV 3 have not been reported in New Zealand.

Bovine viral diarrhoea is not a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

8.1.4 Zoonotic potential

Bovine viral diarrhoea is not a zoonotic disease.

8.1.5 Epidemiology

Host range

Cattle are the natural hosts for BVDV (Walz et al., 2010). *Bovine viral diarrhoea viruses* do not possess strict host specificity.

Infection between domestic species can occur following close contact with sheep, goats, pigs (OIE Terrestrial Manual, 2018h) and alpacas (Mattson et al., 2006).

Bovine viral diarrhoea virus infections have been reported in both new and old world camelids (OIE Terrestrial Manual, 2018h).

Bovine viral diarrhoea virus 3 infections have been reported in water buffalo and cattle in affected countries (Bauermann et al., 2013).

Bovine viral diarrhoea virus has been documented in a variety of wildlife ruminants, which includes deer, elk (Cervidae) (BVDV 1 and BVDV 2), mouse deer (Tragulidae), Canadian bison (BVDV 1), bongo, eland, wildebeest and nilgai (Bovidae) (BVDV 1) (Deregt et al., 2005; Doyle & Heuschele, 1983a; Kottwitz & Ortiz, 2016; Tessaro et al., 1999).

There are currently no known published cases of BVDV 3 infections in wildlife ruminants.

Serological evidence of BVDV infection has been demonstrated in over 50 species within 7 of the 10 families of the mammalian order Artiodactyla (Nettleton, 1990). However, serological evidence does not indicate that these species play a significant role in the epidemiology of the disease.

There is no published evidence of BVD (infection with BVDV 1, BVDV 2 or BVDV 3) in species of the Giraffidae family.

Captive wild ruminants

There is evidence of BVDV infections in wildlife ruminants in zoological collections. However, these are mostly BVDV 1 or unidentified strains.

In the serological survey of US zoos, various captive wild ruminants were identified as seropositive for BVD (strain not reported). *Bovine viral diarrhoea virus* isolates were detected in wildebeest and nilgai (Bovidae) from 2 zoos (Doyle & Heuschele, 1983a).

At Los Angeles Zoo, BVD (strain not reported) seropositive springbok, wildebeest and gaur (Bovidae) had no history of vaccination and were presumed to have been naturally infected with BVD (Doyle & Heuschele, 1983a).

An outbreak of BVD (strain not reported) in pygmy goats occurred at Memphis Zoo. Subsequently, severe diarrhoea developed in a black-backed duiker housed 15 metres away and a scimitar-horned oryx housed on the opposite side of the zoo. *Bovine viral diarrhoea virus* was not isolated from these animals (Doyle & Heuschele, 1983a).

San Antonia Zoo experienced an outbreak of BVD (strain not reported) in waterbuck. The disease spread to sable antelope, cape hartebeest, topi and gemsbok (Bovidae). These animals were then vaccinated as a means of protection (Doyle & Heuschele, 1983a).

Between 1973 and 1994, a serological survey was undertaken at Whipsnade Wild Animal Park (UK). Several semi-free-ranging and captive wild ruminants of the Bovidae and Cervidae families were tested for antibodies against 3 BVDV-like virus strains. Thirteen percent of the animals had antibodies to 1 or more of the 3 BVDV-like viruses (Frölich & Flach, 1998).

A population of lesser Malayan mouse deer was confirmed positive for BVDV 1 at a Copenhagen zoo. The initial positive case was identified as part of the import procedure from ARTIS, the Amsterdam royal zoo. Further investigations demonstrated that the mother and sibling of the initial positive case were persistently infected (PI) animals as well (Uttenthal et al., 2006).

There is no published evidence of BVDV 2 or BVDV 3 in captive wild ruminant species within the scope of this IRA.

There was only serological evidence of BVDV exposure in Giraffidae species.

Geographical distribution

Bovine viral diarrhoea is distributed in cattle populations throughout the world (Walz et al., 2010).

According to WAHIS, all approved countries except Singapore have reported cases of BVD (WAHIS, 2019d).

Evidence of BVDV 2 has been reported in the UK and various other European countries, North America including Canada (Wakeley et al., 2004) and RSA (Ularamu et al., 2013).

Bovine viral diarrhoea virus 2 has not been reported in Australia or Singapore. It is not known whether BVDV 2 is present in the UAE. Therefore, in the absence of this information, it is presumed that UAE is affected by BVDV 2.

Natural infection in cattle due to BVDV 3 has been reported in Asia, Europe and South America (Bauermann et al., 2013; Weber et al., 2014).

Pathogenesis
The pathogeneses of BVDV in domestic and wildlife ruminants appear to be similar (Kottwitz & Ortiz, 2016). However, there may be slight differences in individual species.

The incubation period is usually about 3 to 7 days (Brownlie, 2005) and animals may remain viraemic for 4 to 15 days after initial infection (Potgieter, 2004). Some authors state that viraemia seldom exceeds 10 to 14 days (Brownlie, 2005).
Antibodies develop 2 to 4 weeks after infection.

As mentioned, BVDV comprises cytopathic and non-cytopathic biotypes.
In domestic ruminants, if a naïve dam is infected with the non-cytopathic biotype between 45 and 125 days of gestation, the fetus may become persistently infected (Brock, 2003). Research has not detailed this period in wildlife ruminants. Therefore, a similar scenario is presumed in wildlife ruminants should they become infected within the first third of gestation.

Factors that affect the outcome of BVDV infections are host and viral factors.
Host-dependant factors include immune status, the species of host, pregnancy status and gestational age of the fetus, and the presence of concurrent infections with other pathogens. Viral factors include biotypic variation, genotypic variation and antigenic diversity (Walz et al., 2010).

Experimental infections of white-tailed deer (Cervidae) with BVDV 2 between days 45 and 52 of gestation have resulted in a persistently infected (PI) fawn (Passler et al., 2007).

This specific window period in both domestic and wildlife ruminants allows the virus to infect the fetus prior to maturation of the immune system. These fetuses may develop normally and be born healthy, but will remain infected for life (Brock, 2003; Passler et al., 2007). Persistently infected animals will shed virus for life and act as a significant reservoir of infection, especially in a confined environment (Brock, 2003).

Persistent infections have also been reported in mouse deer (Tragulidae) (BVDV 1), North American elk (Cervidae) (BVDV 1 and BVDV 2) and eland (Bovidae) (BVDV 1) (Tessaro & Clavijo, 2001; Uttenthal et al., 2005; Vilcek et al., 2000).

Animals that are infected with the non-cytopathic biotype are immunotolerant to that particular biotype and are therefore unable to clear the infection (Brock, 2003; Passler et al., 2007).

Cytopathic BVDV biotypes can infect PI and completely naïve animals. Infection with these biotypes are characterised by unrestricted viral replication with the production of large volumes of virus that contaminate the environment. This is, however, self-limiting, as infection usually results in death of the animal (Peterhans et al., 2010). These biotypes are unable to cause persistent infection and thus fail to establish in animal populations (Brock, 2003).

Superinfection of PI domestic ruminants with a cytopathic biotype results in mucosal disease (MD) (Brock, 2003). Mucosal disease has not been described in wildlife ruminants thus far.

Bovine viral diarrhoea virus can infect domestic animals such as sheep, goats, pigs and alpacas. These animals are sometimes kept at zoos and together with wildlife ruminants, may act as reservoirs for infection of other susceptible animals (Kottwitz & Ortiz, 2016).

Clinical signs
There is a wide range of clinical manifestations for BVDV infections, from subclinical infections to fatal disease.

Experimentally infected white-tailed deer showed no clinical signs of BVD or abortions. However, the fawn was determined to be PI with BVDV 2 (Passler et al., 2007).

Persistent infections with non-cytopathic biotypes are often associated with decreased fertility, immunosuppression, stunted growth and secondary infections (Brock, 2003; Potgieter, 2004).

Infection with cytopathic biotypes alone typically cause an acute-phase disease, with rapid onset of clinical signs, debilitation and death (Kottwitz & Ortiz, 2016).

In domestic cattle, MD results in low morbidity and high case fatality in the age group of 6 months to 2 years old. Gross lesions in domestic species include extensive mucosal ulceration primarily within the gastrointestinal tract, with resultant diarrhoea, weight loss and wasting (Brock, 2003). It is not known whether wildlife ruminants can develop MD with associated clinical signs as in domestic ruminants.

Clinical signs attributed to natural BVDV 3 infections in cattle included blind newborn calves, reproductive failure in herds, acute gastroenteric and respiratory disease. In some cases, serologically positive animals showed no clinical signs (Bauermann et al., 2013).

Transmission

Vertical (Meyling & Jensen, 1988) and horizontal transmission has been demonstrated. Horizontal transmission can occur directly and indirectly.

The most important source of BVDV in nature is the immunotolerant, PI animal. These animals secrete high viral loads in their nasal discharges, saliva, tears, semen, milk, urine and faeces. These PI animals are highly infectious to naïve in contact susceptible animals (Houe, 1995).

Another source of infection are those animals undergoing primary or postnatal infections. These animals are transiently infected and will eventually clear the infection. However, their secretions and excretions bear viral loads capable of infecting in contact animals, even though they are several times lower than viral loads found in the secretions of PI animals (Kirkland et al., 1997).

Indirect transmission has been reported via the airborne route and unhygienic vaccine practices. Airborne transmission was reported to occur at a distance of 1.5 to 10 metres from a PI animal (Niskanen & Lindberg, 2003).

Iatrogenic infections may occur via hypodermic needles, nose tongs, ear-tagging instruments, castration operations, oral infusions and repeated rectal examinations (Potgieter, 2004).

Interspecies transmission can occur following close contact with sheep, goats, pigs (OIE Terrestrial Manual, 2018h) and alpacas (Mattson et al., 2006).

Diagnosis

The variability of clinical signs for BVDV in wildlife ruminants complicates diagnosis and may result in infections being overlooked because of the lack of pathognomonic or clinical signs (Kottwitz & Ortiz, 2016).

The OIE recommends virus isolation and antigen detection by ELISA as a means of agent identification for identifying clinical cases and individual animal freedom from infection prior to movement. ELISA may also be used for eradication purposes and surveillance (OIE Terrestrial Manual, 2018h).

Nucleic acid detection by real time RT-PCR is the method of choice for all of the above purposes, including confirming that a population of animals is free from infection.

ELISA and virus neutralisation tests may be used to detect an immune response in various scenarios (OIE Terrestrial Manual, 2018h).

The above diagnostic tests are approved for domestic ruminants and have not been validated for wildlife species. The only reliable tests would be identifying the virus via virus isolation (Kottwitz & Ortiz, 2016) or PCR.

There are various studies in which serological surveys for BVDV have been conducted in wildlife ruminants both in the wild and in zoological collections, in order to screen for disease conditions. It was demonstrated that these serological tests are capable of identifying BVDV antibodies produced by wildlife ruminants (Kottwitz & Ortiz, 2016; Probst et al., 2010).

The use of a combination of antigen-capture ELISA (ACE) on haired skin, RT-PCR on whole blood (buffy coat, collected in EDTA) and antibody detection via serum neutralization has the greatest

likelihood of identifying PI animals and those that may be transiently infected (Brock, 2003; Walz et al., 2010).

Diagnostic tests that identify BVDV 1 and BVDV 2 are less efficacious for BVDV 3 (Bauermann et al., 2013).

“To date there is no test fully validated to detect all bovine pestivirus species or to specifically detect HoBi-like viruses” (Bauermann et al., 2013). This highlights the need for more than just a single test to obtain a reliable result.

Treatment, control and prevention

In the zoological garden setting, animals experiencing clinical signs of BVD such as diarrhoea and weight loss may be held off display, quarantined, and examined or tested to determine the cause of illness. By the time clinical signs are noticed, the virus would have contaminated the environment. In contact susceptible animals are likely to have become infected at this stage.
Appropriate isolation of animals and testing for BVDV prior to introduction into collections would help prevent introduction and spread of the disease.

If a zoo has had a history of BVDV cases; identifying subclinical PI animals in populations is vital in preventing further spread and establishment. Depending on the BVDV strain, their importance in a zoological collection and the country’s disease sanitary status, culling may be considered. However, for highly valuable species in countries that already have BVDV, management may be the route followed (Kottwitz & Ortiz, 2016).

Vaccination has been practised in domestic ruminants to prevent fetal spread. However, this is not well documented in wildlife (Kottwitz & Ortiz, 2016). Zoos in the USA have a history of BVD vaccination (Doyle & Heuschele, 1983a).

Semen

In the case study by Rikula et al. (2008), it was demonstrated that cattle semen contaminated with BVDV was the only plausible explanation for the source of infection of dairy herds. The semen, the herds that received the semen, PI calves from that herd, and contact herds all tested positive for the identical strain of BVDV (Rikula et al., 2008).

In another case, 12 heifers were experimentally inseminated with semen from a bull that was persistently infected with BVDV. All heifers became infected within 2 weeks of insemination. Out of the 12 calves that were born, one was diagnosed as PI with BVDV. This study demonstrates that BVDV contaminated semen can result in infection of naïve dams and in the birth of PI calves (Meyling & Jensen, 1988).

There is no published evidence demonstrating the presence of BVDV in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with BVDV-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

8.1.6 Hazard identification conclusion

Bovine viral diarrhoea is an OIE-listed disease of cattle. Bovine viral diarrhoea can affect multiple domestic and wildlife ruminants.

There was only serological evidence of BVDV exposure in the Giraffidae family, so Giraffidae are not assessed to play a significant role in BVD epidemiology.

Bovine viral diarrhoea virus 2 and *Bovine viral diarrhoea virus 3* are not identified as hazards in captive wild Giraffidae and their semen.

Bovine viral diarrhoea virus has been isolated from the semen of domestic ruminants. In the absence of evidence in wildlife ruminants, extrapolation is made to species within the scope of this IRA.

Bovine viral diarrhoea virus 2 and *Bovine viral diarrhoea virus 3* are identified as hazards in captive wild Bovidae, Tragulidae and their semen.

8.2 Risk assessment

8.2.1 Entry assessment

Bovine viral diarrhoea virus 2 and BVDV 3 have not been reported in Australia or Singapore. Bovine viral diarrhoea is present in all other approved countries.

Evidence of BVDV has been documented in a variety of wildlife ruminants which include deer, elk (Cervidae) (BVDV 1 and BVDV 2), mouse deer (Tragulidae), Canadian bison (BVDV 1), bongo, eland, wildebeest, nilgai (Bovidae). *Bovine viral diarrhoea virus 1* infections have been reported in wildlife ruminants in zoological collections.

There is a lack of evidence of BVDV 2 and BVDV 3 infections in captive wild ruminants. However, the fact that BVDV 1 has the ability to infect these species suggests that infection with other strains (BVDV 2 and BVDV 3) is likely.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Wildlife ruminants infected with BVDV do not show clinical signs of disease while being persistently infected and shedding the virus. These animals are likely to be passed as clinically sound for export or as donor males for semen collection.

In cattle, transiently infected or PI bulls produce large volumes of BVDV in their semen. In the absence of evidence of BVDV in the semen of wildlife ruminants, extrapolation is made from cattle.

Therefore, the likelihood of entry of BVDV 2 and BVDV 3 via captive wild Bovidae, Tragulidae (within the scope of this IRA) and their semen from BVD-affected countries is assessed as very low.

8.2.2 Exposure assessment

Vertical and horizontal transmission has been described for BVDV infections.

Direct transmission includes close contact of susceptible animals with contaminated secretions and excretions of PI and transiently infected animals. Various routes of indirect transmission have also been described, such as aerosol transmission, veterinary equipment and procedures.

The small number and occasional importation of probably infected live captive wild ruminants, implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If BVDV-infected animals are imported and are subclinically infected, the likelihood of transmission to in-contact susceptible species is high, especially if these animals are PI. Since aerosol transmission and infection via fomites is probable, separation may not entirely prevent spread to susceptible animals that are not in direct contact.

If animals display clinical signs of BVDV infection like diarrhoea, they will often be isolated from healthy herd members. Diagnostic testing is likely to be performed by veterinary staff. Assuming that isolation is prompt, this may reduce risk of exposure of other susceptible animals.

Most domestic and wildlife ruminants may be reservoir hosts if infected with the non-cytopathic biotypes of BVDV. They are likely to become immunotolerant to that biotype and be unable to clear the infection. These reservoir animals, together with PI animals will continuously shed virus, thus contaminating the environment and being a source of infection to susceptible animals within the zoo. BVDV is therefore likely to establish and spread within the zoo if not identified and contained promptly.

There is evidence of airborne transmission of BVDV of up to 10 metres.

If domestic ruminants are kept at the zoo, they could become infected with BVDV strains not present in New Zealand. Should these domestic ruminants be released onto New Zealand farms, they could transmit BVDV to other domestic ruminants. *Bovine viral diarrhoea virus 1* has established in

New Zealand, and therefore, other strains of BVDV could spread and establish in the domestic ruminant population.

Artificial insemination with BVDV-contaminated semen has resulted in the infection of naïve domestic ruminants and birth of PI calves. There is uncertainty as to whether BVDV-infected wildlife ruminants are likely to produce BVDV-contaminated semen. It is also not known whether naïve wildlife ruminants will become infected with BVDV if BVDV-contaminated semen is used for AI. However, by extrapolating the outcomes from domestic ruminants to wildlife ruminants, it is assumed that infection may occur.

There are limited numbers of captive wild female ruminants in New Zealand zoos. Thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of BVDV 2 and BVDV 3 exposure and establishment within the zoo via infected captive wild Bovidae and Tragulidae is assessed as high, the likelihood of exposure and establishment outside the zoo is assessed as very low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae and Tragulidae is assessed as low.

8.2.3 Consequence assessment

Bovine viral diarrhoea is an OIE-listed disease of cattle.

Most cattle-producing countries, including New Zealand, are affected by BVDV 1. *Bovine viral diarrhoea virus 2* and BVDV 3 have been reported in some of the approved countries and could therefore enter New Zealand either via imported captive wild ruminants or their semen.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of BVDV would depend on the strain of BVDV that imported animals may be infected with.

Non-cytopathic strains would result in subclinical infections of captive wild ruminants that could go unnoticed. These animals would then become a source of contamination of the environment and transmission to other susceptible domestic and captive wild ruminants. Persistently infected animals are likely to be born under these conditions. *Bovine viral diarrhoea virus* could thus spread and establish in zoos as a result. There may be low mortality but higher morbidity such as reproductive failure and losses, and unthrifty animals. This would affect the zoos' breeding programmes.

Cytopathic strains would result in clinical disease and higher mortalities. These infected animals are likely to be isolated promptly with further disease investigation. This course of action may limit the spread and establishment of the disease.

The likelihood of exposure and establishment of BVDV to domestic and wildlife ruminants outside the zoo is assessed as very low and is only likely to happen if domestic ruminants are released from the zoo to New Zealand farms. *Bovine viral diarrhoea virus* could be transmitted to cattle, sheep, goats, deer and pigs. Severity of disease could range from subclinical infections to mortality, depending on host and viral factors.

Bovine viral diarrhoea is not a zoonotic disease, and therefore, the consequences for human health are negligible.

Indirect consequences would entail the costs for control and surveillance within the affected zoos. Should the disease spread further than expected, loss of valuable species within zoos may also occur. This would negatively impact the Australasian Species Management Program (ASMP).

There would be additional costs to New Zealand for BVD control in the event of an incursion in domestic ruminants.
Bovine viral diarrhoea is an OIE-listed disease. *Bovine viral diarrhoea 1* is present in New Zealand. Therefore, negative trade impacts are likely to be minimal and may be restricted only to countries that are free of the particular BVDV strain.

Therefore, the overall consequences as a result of a BVDV 2 or BVDV 3 incursion are assessed as low.

8.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for BVDV 2 or BVDV 3 is non-negligible, and they are assessed to be risks in captive wild Bovidae, Tragulidae and their semen.

Therefore, risk management measures can be justified.

8.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Bovine viral diarrhoea is an OIE-listed disease of cattle.
- *Bovine viral diarrhoea virus 2* and BVDV 3 have not been reported in New Zealand.
- Bovine viral diarrhoea is not a notifiable disease in New Zealand.
- Bovine viral diarrhoea is not a zoonotic disease.
- Bovine viral diarrhoea affects multiple species.
- *Bovine viral diarrhoea virus 2* and BVDV 3 has not been reported in Australia or Singapore.
- Natural infection in cattle due to BVDV 3 has been reported in Asia, Europe and South America.
- The incubation period is usually about 3 to 7 days.
- Animals may remain viraemic for 4 to 15 days after initial infection.
- *Bovine viral diarrhoea virus* comprises cytopathic and non-cytopathic biotypes.
- Persistently infected animals will shed the virus for life and act as a significant reservoir of infection.
- Vertical and horizontal transmission has been demonstrated. Horizontal transmission can occur directly and indirectly.
- Diagnostic tests include ELISA, real time RT-PCR and virus neutralisation. These tests are not validated for wildlife species.
- Combination testing may be more reliable for animals that require limited handling.
- Appropriate isolation of animals and testing for BVDV prior to introduction into collections would help prevent the introduction and spread of the disease.
- *Bovine viral diarrhoea virus* has been detected in domestic ruminant semen.
- *Bovine viral diarrhoea virus*-contaminated semen has resulted in the infection of naïve dams and the birth of PI calves.

8.3.1 Options

One or a combination of the following options may be used:

Bovine viral diarrhoea virus 2 and BVDV 3 are not identified as hazards in species within the Giraffidae family and their semen, and therefore, risk management measures are not warranted for these species.

Option 1

1. Country freedom for BVDV 2 and BVDV 3; AND
2. the animal(s)/donor male(s) were resident in BVDV 2- and BVDV 3-free countries since birth; AND
3. the animal(s)/donor male(s) showed no clinical signs on the day of export or semen collection.

Option 2

Animals:

During pre-export isolation:

1. A whole blood sample from the animal(s) was tested by RT-PCR. The test result was negative for BVDV; AND
2. a haired skin sampled (ear notch or caudal tail fold) from the animal(s) was tested by an antigen-capture ELISA test. The test result was negative; AND
3. a serum sample from the animal was tested by virus neutralisation. The test result was negative.

Semen:

1. The semen was tested by a virus isolation or real-time RT-PCR for BVDV, with negative results; OR
2. donor male(s) were tested by the above animal protocol during collection. Semen may only be exported if all test results are negative.

Due to the unreliability of test results and lack of validation of tests for wildlife species, a combination of various tests is recommended. A single test may not identify PI animals. Samples for all 3 tests can be obtained at one time.

Option 3

1. For 180 days immediately prior to export or collection of semen, the animal(s)/donor male(s) were part of a captive wild animal collection, where no clinical, epidemiological or other evidence of BVDV (strains not present in New Zealand) has occurred during the previous 12 months; AND
2. For 180 days immediately prior to export or collection of semen, the animal(s)/donor male(s) were part of a captive wild animal collection subject to a documented BVDV screening program. The screening program must include:
    a. Diagnostic testing of all captive wild bovids in the collection, performed prior to the animal(s) entering the collection. The diagnostic tests must be of a type approved by MPI. The testing regime must be able to identify infected or PI animals; AND
    b. The collection must have been a 'closed herd' during that time.

This option allows animals to circumvent pre-export isolation testing requirements if the exporting facility can prove BVDV freedom in the captive wild animal collection.

9 Cowpox

9.1 Technical review

9.1.1 Aetiological agent

Family: *Poxviridae*
Genus: *Orthopoxvirus*
Species: *Cowpox virus* (ICTV, 2019)

Cowpox virus (CPXV) is the causative agent of cowpox.

9.1.2 OIE list

Cowpox is not an OIE-listed disease.

9.1.3 New Zealand status

Cowpox is absent from New Zealand (Vermunt & Parkinson, 2000) and is not a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

9.1.4 Zoonotic potential

Cowpox is a zoonotic disease (Eder et al., 2017). Humans usually become infected after contact with cats or pet rats (Campe et al., 2009).

In patients without underlying conditions, symptoms manifest as a mild skin disease. Single pock-like lesions heal in 3 to 6 weeks.

In humans that are immunocompromised, the disease may have a protracted clinical presentation, taking several months to subside. Severe cases could lead to generalised disease and result in fatality (Eis-Hübinger et al., 1990).

9.1.5 Epidemiology

Host range

Wild rodents are the primary hosts and reservoirs of CPXV. Cats become infected due to their hunting behaviour.

Infection with CPXV has been observed in a wide range of animals including cows, cats, dogs, primates, elephants and various zoo animals (Bush, 2003; Nitsche & Pauli, 2007; Wolters & van Bolhuis, 2008; Zwart et al., 1971). These animals, along with humans, are incidental hosts.

Infection may also occur in okapis and giraffes (Giraffidae) (Bush, 2003; Wolters & van Bolhuis, 2008; Zwart et al., 1971).

Outbreaks of cowpox in other captive wild animal species include but are not limited to banded mongooses, primates, carnivores and elephants (Kurth et al., 2009; Marennikova et al., 1977; Wisser et al., 2001).

There is no published evidence of cowpox in wildlife species of the Giraffidae or Tragulidae families.

Captive wild ruminants

There have been limited published cases of cowpox in captive wild ruminants. Only okapis have shown infection with CPXV and giraffes have reported to be susceptible.

Five okapis at a Rotterdam zoo had confirmed CPXV infections. The source of infection was unknown. The disease did not spread to any other animals in the zoo or to the giraffes that were in close contact with the okapis even though they were handled by the same zoo personnel until the outbreak (Zwart et al., 1971).

Geographical distribution

Cowpox is prevalent in Europe (Campe et al., 2009; Nitsche & Pauli, 2007) and adjacent areas of North and Central Asia (Vorou et al., 2008).

Pathogenesis

The incubation period is 3 to 7 days (Gibbs, 2013; Mayr & Czernt, 1990).

The pathogenesis of CPXV is similar to that of vaccinia virus infections.

Once the virus enters abrasions or skin lesions, they form pustules in the skin. In some animals, the virus may enter the circulation and cause a severe systemic generalised disease.

Following a viraemia, the systemic form may affect the skin, mucous membranes, internal organs and central nervous system (Mayr & Czernt, 1990).

Younger animals are more severely affected than older animals (Mayr & Czernt, 1990).

Latent infections are likely to occur. This theory has not yet been proven (Mayr & Czernt, 1990) but is suggested based on observations in rats.

Subclinical infections have been described in cats with cat-to-cat transmission (Shimsony, 2009) and in reservoir animals such as rodents (Feore et al., 1997).

Clinical signs

Cows become mildly febrile during incubation of the disease. Papules appear on the udder, teats and scrotum. Vesicles are formed but may not be evident, as they easily rupture, leaving raw, ulcerated areas that form scabs (Gibbs, 2013). Pustules reach maturity after 8 to 11 days, then scab over (Mayr & Czernt, 1990). Pustules may become infected with bacteria, causing abscesses or ulcers. Lesions often heal within 1 month (Gibbs, 2013).

In animals that suffer systemic infections, disseminated pin-sized, whitish foci may develop on internal organs. In severe cases, haemorrhagic diathesis occurs. Exudation of blood is often seen in the pleural cavity and pericardial sac (Mayr & Czernt, 1990).

Abortions may be observed in pregnant animals.

The prognosis is favourable if lesions are localised. However, systemic disease with haemorrhagic signs could lead to 50% mortality (Mayr & Czernt, 1990).

Lesions observed in affected okapis included papules, vesicles and pustules on the skin and mucosa of the mouth and tongue. The calf (2 months old) was severely affected with generalised lesions and died 18 days after the onset of clinical signs. The other 4 adults had varying degrees of pox lesions. The adults recovered over 4 to 6 weeks (Bush, 2003; Zwart et al., 1971).

Transmission

Transmission occurs by direct contact between infected and susceptible animals (Gibbs, 2013).

An oronasal route of transmission has been suggested in cat infections (Shimsony, 2009).

There is potential for transmission via ingestion in carnivores. Cats often become infected after hunting infected rats (Nitsche & Pauli, 2007). In cases of outbreaks in captive wild carnivores, sources of infection were suggested to be infected rats that were fed to carnivores (Kurth et al., 2009; Marennikova et al., 1977). This route of transmission has not been described in ruminants.

Diagnosis

A tentative diagnosis of cowpox can be made on clinical lesions; however, a laboratory confirmation is required.

Blood or skin samples can be used for diagnosis.

Orthopoxviruses can be detected by electron microscopy or serology. However, differentiation needs to be completed. This can be achieved by DNA sequencing or real-time PCR (Nitsche & Pauli, 2007).

Virus isolation can also be used.

Treatment, control and prevention

There is currently no treatment for cowpox virus (Nitsche & Pauli, 2007). Antibiotics may be used to prevent secondary bacterial infections. Skin and mucous membrane inflammation may be treated symptomatically.

In severe human cases, lesions may respond to anti-vaccinia immunoglobulin. However, this course of treatment is used as a last resort due to the adverse side effects (Nitsche & Pauli, 2007).

Veterinary personnel or animal handlers coming into contact with infected animals must take appropriate safety precautions. Proper hygiene and sanitary measures must be followed, and personal protective equipment (PPE) should be worn.

In an outbreak situation, susceptible animals may be protected using vaccines (Mayr & Czernt, 1990).

Infected animals must be separated from susceptible animals.

Semen

There is no published evidence of cowpox virus in the semen of ruminants.

Due to the absence of any evidence demonstrating the presence in or transmission of *cowpox virus* via semen, we conclude that it is absent in semen and cannot be transmitted by semen.

9.1.6 Hazard identification conclusion

Cowpox is not an OIE-listed or nationally notifiable disease and is absent from New Zealand.

It can cause disease in 2 species within the scope of this IRA.

There is no published evidence of cowpox in wildlife species of the Giraffidae or Tragulidae families.

Cowpox virus is not identified as a hazard in captive wild Bovidae and Tragulidae and will not be assessed further.

Cowpox virus is identified as a hazard in captive wild Giraffidae.

There is no evidence demonstrating the presence of CPXV in the semen of domestic or wildlife ruminants.

Cowpox virus is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

9.2 Risk assessment

9.2.1 Entry assessment

Cowpox is known to occur in Europe and parts of Asia.

Since cowpox is not an OIE-listed disease, it is unlikely to be nationally notifiable in approved countries. As a result, it may be difficult to establish country freedom.

There is likely to be a very small number of live captive wild giraffes and okapis imported into New Zealand, and these imports are also likely to be infrequent. Therefore, the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

Cowpox virus has been detected in okapis, and giraffes are reported to be susceptible to infection. However, these cases were quite rare.

Latent and subclinical infections have only been described in reservoir animals and carnivores. The disease transmission and progression is different in carnivores and reservoir animals than in incidental ruminants.

It is unlikely that ruminants would experience subclinical infections or latency, due to the route of transmission, which is likely to be direct contact with infected rodents or cats.

The last reported case of cowpox in okapis was in 1971 at a European zoo. Infected animals suffered clinical cutaneous lesions. In this case, no other animals in the zoo were affected. Giraffes that were in close contact and handled by the same keeper did not become infected.

According to the literature, there has only been 1 clinical case of cowpox in okapis in the past 51 years. Therefore, the disease is extremely rare.

Therefore, the likelihood of entry of CPXV via captive wild Giraffidae (within the scope of this IRA) from cowpox-affected countries is assessed as negligible.

9.2.2 Risk estimation

Since the entry is assessed as negligible, the risk estimate for cowpox is negligible, and it is not a risk in captive wild Giraffidae.

Therefore, risk management measures are not warranted.

10 Crimean Congo haemorrhagic fever

10.1 Technical review

10.1.1 Aetiological agent

Family: *Nairoviridae*
Genus: *Orthonairovirus*
Species: *Crimean Congo haemorrhagic fever orthonairovirus* (CCHFV) (ICTV, 2019)

Crimean Congo haemorrhagic fever orthonairovirus is the causative agent of Crimean Congo haemorrhagic fever (CCHF).

10.1.2 OIE list

Crimean Congo haemorrhagic fever is an OIE-listed disease affecting multiple species.

10.1.3 New Zealand status

New Zealand is free from CCHF (WAHIS, 2019d), as reported to the OIE.

Crimean Congo haemorrhagic fever is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

10.1.4 Zoonotic potential

Crimean Congo haemorrhagic fever is a zoonotic disease (Shayan et al., 2015). In humans, clinical signs include high fever, malaise, severe headache, gastrointestinal symptoms, haemorrhage and a fatality rate of 5 to 50% (Tuncer et al., 2014).

10.1.5 Epidemiology

Host range

Numerous wild and domestic ruminants have been exposed to CCHFV, as indicated by serological surveys (Nalca & Whitehouse, 2007). These animals may serve as hosts for viral replication.

Various studies have detected antibodies to CCHFV and viraemias in sheep, cattle and goats in non-enzootic areas (Tuncer et al., 2014).

Camels were also found to be seropositive (Nalca & Whitehouse, 2007).

Antibodies against CCHFV have been detected in giraffes (Giraffidae), elands, buffaloes and kudus (Bovidae) (Shepherd et al., 1987).

Reptiles and birds (except ostriches) appear to be refractory to CCHFV infection (Shayan et al., 2015).

Due to the wide host range of CCHFV, it is assumed that species of the Tragulidae family may be susceptible to infection.

Captive wild ruminants

There is no published evidence of CCHF in captive wild animals. However, there is evidence of exposure of wild ruminants to CCHFV.

Retrospective serological studies were conducted on archived wild ruminant sera from South Africa and Zimbabwe between 1964 and 1985. Antibodies against CCHFV were detected in various wild ruminants (Shepherd et al., 1987).

Another study of 29 wild vertebrate species in the Kruger National Park (South Africa) between 1974 and 1992 demonstrated antibodies against CCHFV in buffaloes and giraffes (Burt et al., 1993).

Geographical distribution

The geographical distribution of CCHF closely corresponds with the principal tick vector, reported to be ticks of the genus *Hyalomma.*

Crimean Congo haemorrhagic fever was initially discovered in the Crimea and Democratic Republic of Congo. It has since been reported in many regions of Africa, the Middle East and Asia (Shayan et al., 2015).

Between 2000 and 2013, outbreaks of CCHF in humans were reported in parts of Europe, India, Africa and Asia (Shayan et al., 2015).

Seropositive domestic animals and camels have been reported in the UAE (Nalca & Whitehouse, 2007).

Antibodies against CCHFV have been detected in domestic animals over large areas of South Africa and Zimbabwe (Swanepoel et al., 1987).

Many factors, including climate, population growth, mobility, agriculture, ecological changes and movement of livestock, may provide opportunities for CCHFV to spread to previously unaffected countries (Shayan et al., 2015).

According to the WAHIS interface, CCHF has never occurred in animals in Australia, Singapore, the UAE, the USA, Canada or the UK (WAHIS, 2019d).

Regarding UAE freedom, there is evidence of seropositive animals (Nalca & Whitehouse, 2007) in the UAE with reported human cases (Mohamed Al Dabal et al., 2016). The human cases have been linked to the slaughtering of animals carrying infected ticks that have been imported from CCHF-affected countries during the Hajj season.

Pathogenesis

Crimean Congo haemorrhagic fever is a tick-borne disease affecting humans.

The pathogenesis of CCHF is not completely understood. It has been suggested that the virus first replicates at the site of inoculation, with haematogenous and lymphogenous spread of the virus to organs such as the liver, which become major sites of replication. The widespread infection of the endothelium with degenerative change could lead to capillary dysfunction and result in haemorrhagic diathesis and other haemorrhagic lesions (Swanepoel & Burt, 2004).

Crimean Congo haemorrhagic fever orthonairovirus generally circulates in nature unnoticed in an enzootic tick–vertebrate–tick cycle (Nalca & Whitehouse, 2007; Tuncer et al., 2014).

Crimean Congo haemorrhagic fever orthonairovirus is apathogenic in its natural hosts, but highly pathogenic in humans.

Domestic ruminants (cattle, sheep and goats) are regarded as the main hosts of CCHFV. Animals are able to produce a viraemia capable of infecting ticks (Shayan et al., 2015); however, only viraemia over a certain threshold level will be sufficient to infect feeding ticks (Nalca & Whitehouse, 2007).

A comparison of experimental studies on calves by Causey et al. (1970) and Zarubinsky et al. (1976) showed that CCHFV could be recovered from the blood of a 2-month-old calf on days 3 and 7 post infection, but not from 6-month-old calves, as they did not develop a sufficient viraemia. Both calves in the later study did, however, produce high antibody titres against CCHFV (Causey et al., 1970; Zarubinsky et al., 1976).

In the study by Gonzalez et al. (1998), virus was isolated from the blood of experimentally infected sheep from day 3 to day 9 post infection (Gonzalez et al., 1998).

These studies could suggest a viraemic period of up to 2 weeks in domestic and wildlife ruminants (Gunes et al., 2011).

Carrier status has not been reported in animals.

Clinical signs

Experimental studies have attempted to establish infection in animal models. However, the only animal to manifest disease was a newborn mouse (Nalca & Whitehouse, 2007).

Crimean Congo haemorrhagic fever orthonairovirus infections are generally subclinical in domestic and wildlife animal hosts.

Experimental infections of 2 calves produced mild illness characterised by dullness, lassitude and decreased appetite (Causey et al., 1970).

Experimental infections in lambs produced a moderate, but constant fever which correlated with viraemia, hepatic dysfunction and abnormal blood cell counts. No other overt clinical signs were observed (Gonzalez et al., 1998).

Transmission

Animals are infected after being bitten by ticks carrying CCHFV.

The principal vectors are ticks of the genus *Hyalomma* (Hoogstraal, 1979). Natural hosts of these ticks are small- to medium-sized mammals such as hares, hedgehogs and large ruminants.

Crimean Congo haemorrhagic fever orthonairovirus has been isolated from over 30 tick species including 28 *Ixodidae* and 2 *Argasidae* species. Isolation of the virus in these ticks does not imply that they are competent vectors (Bell-Sakyi et al., 2012).

Transmissibility of CCHFV has, however, been demonstrated in 5 other genera: *Rhipicephalus, Dermacentor*, *Amblyomma (A. variegatum), Haemaphysalis* (*H. punctate)* and *Ixodes* (*I. ricinus)* (Heath et al., 2016; Shepherd et al., 1989; Swanepoel & Burt, 2004).

Studies that have demonstrated isolation of CCHFV in tick species other than *Hyalomma* have been experimental or lack virus transmission data. There is limited evidence indicating that CCHFV can be maintained or transmitted naturally in the environment by species of ticks other than *Hyalomma* spp. (Papa et al., 2015). Studies to prove natural infection of ticks and transmission by individual tick species would be required to confirm vector competency (Shepherd et al., 1989).

New Zealand has a number of Ixodid ticks; however, there is uncertainty as to the vector competency of these ticks or the role they could play in transmission of CCHFV. Currently, none of the recognised competent tick species are present in New Zealand.

Ticks may also become infected from co-feeding with infected ticks on uninfected hosts (Gordon et al., 1993). Transovarial, transstadial and vertical transmission of CCHFV in ticks is likely (Whitehouse, 2004). Therefore, ticks are not only vectors but reservoirs of CCHFV as well.

Transmission to humans occurs through tick bites, crushing engorged ticks or contact with infected animal or human blood (Whitehouse, 2004). Humans become infected during high-risk behaviours such as slaughtering, handling or examining livestock without PPE, and unauthorised slaughtering of animals (Shayan et al., 2015). Healthcare workers are at risk of becoming infected through the blood and bodily fluid of patients (Celikbas et al., 2014).

Diagnosis

The OIE recommends real-time RT-PCR (Tuncer et al., 2014) for individual animal freedom from infection prior to movement. In order to detect an immune response, immunoglobulin G ELISA and competitive ELISA can be used to determine animal population freedom and for surveillance purposes (Gonzalez et al., 1998; OIE Terrestrial Manual, 2018n).

Due to the short viraemic period, recent infections in animals are rarely diagnosed.

Treatment, control and prevention

In countries that are affected by CCHF, limiting exposure to infected ticks and the blood or fluids of infected animals or humans would aid in the prevention of CCHF in both animals and humans.

Slaughterhouse workers, veterinarians, stock workers and others involved with livestock should take the proper precautions and use appropriate PPE. They should avoid handling or being bitten by ticks (OIE Terrestrial Manual, 2018n). Education and awareness about the disease may also help those who are unaware of the severity of infection.

The use of acaricides on animals could reduce the tick density among animals and thus reduce the risk of tick bites in animal handlers (Mertens et al., 2013). However, this may not be practical in extensive farming practices.

Countries in which CCHF is not enzootic or countries that are free from the disease can prevent the entry of CCHF by limiting the importation of animals infected with CCHFV and animals that may be carrying infected ticks. Eliminating the illegal transportation of animals between countries may also aid in prevention.

Semen

There is no published evidence demonstrating the presence of CCHFV in semen of wildlife ruminants.

10.1.6 Hazard identification conclusion

Crimean Congo haemorrhagic fever is an OIE-listed disease affecting multiple species. Wild ruminants have shown serological evidence of exposure to CCHFV but are usually subclinically infected.

Crimean Congo haemorrhagic fever orthonairovirus is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

There is no evidence demonstrating the presence of CCHFV in semen of wildlife ruminants.

Crimean Congo haemorrhagic fever orthonairovirus is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

10.2 Risk assessment

10.2.1 Entry assessment

Crimean Congo haemorrhagic fever has never occurred in Australia, Singapore, the USA, Canada or the UK.

Crimean Congo haemorrhagic fever does not cause clinical disease in animals. Mild fever and blood chemistry abnormalities have been reported in experimentally infected domestic ruminants. These studies suggest that domestic and wildlife ruminants may remain viraemic for up to 2 weeks post infection. *Crimean Congo haemorrhagic fever orthonairovirus* seropositivity has been reported in wild ruminants including giraffes (Giraffidae), elands, buffaloes and kudus (Bovidae) in South Africa. There are no reports of CCHF in animals from zoological collections.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Should animals be imported from CCHF-affected countries (Europe, South Africa and UAE), there is a likelihood that they could be viraemic, subclinical and be passed as clinically sound for export. During this viraemic period, they could infect ticks that may be present on these animals.

These viraemic animals along with infected ticks could be imported into New Zealand if they are not treated with acaricides or examined for external parasites.

Therefore, the likelihood of entry of CCHFV via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from CCHF-affected countries is assessed as very low.

10.2.2 Exposure assessment

The main route of transmission of CCHFV to other animals is via Ixodid ticks. The main tick vectors are those of the genus *Hyalomma*, but other tick species may play a minor role in the transmission of CCHFV.

Currently, all reported competent tick vectors of CCHFV are absent from New Zealand. It is uncertain whether ticks present in New Zealand could play a role in transmission of CCHFV.

Crimean Congo haemorrhagic fever is a zoonotic disease. Transmission to humans occurs through tick bites, crushing engorged ticks or contact with infected animal or human blood or bodily fluids.

Animals imported will become part of a zoological collection and will not be slaughtered. There may be minimal exposure of veterinary staff at zoos to infected animal blood during veterinary procedures. Therefore, the likelihood of human exposure to infected animal blood is very low.

Transmission via direct contact or airborne transmission has not been reported in animals. The virus has also not been detected in secretions or excretions of animals.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If animals imported into New Zealand are viraemic, the likelihood of these animals directly infecting other animals or humans is negligible.

However, if the ticks they carry are infected, there is a likelihood that ticks could drop off and bite other in-contact animals, and there is a low likelihood that ticks could bite human handlers. The only humans that are likely to be exposed to infected ticks are zoo and veterinary staff. There would be a negligible likelihood of exposure to the public.

Domestic and wildlife ruminants outside the zoo would only become infected if bitten by infected ticks. If infected ticks attach to domestic ruminants that are kept at the zoo and domestic ruminants are released to New Zealand farms, they could in turn infect other domestic ruminants. However, this likelihood is assessed as very low. The likelihood of establishment is assessed as extremely low, because ticks would require a constant source of infection in the form of infected animals. A few infected domestic ruminants are not likely to provide this source of infection.

Competent tick vectors are necessary to establish CCHFV infection in an animal population. In the absence of these vectors, CCHF is not likely to spread and establish. There is uncertainty surrounding the vector competence of New Zealand ticks and the ability of new tick species to establish in New Zealand.

Therefore, the likelihood of CCHFV exposure and establishment within the zoo via infected ticks is assessed as very low, and the likelihood of exposure and establishment outside the zoo is assessed as very low.

10.2.3 Consequence assessment

Crimean Congo haemorrhagic fever is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of CCHF would be in the form of animal disease and production losses and public health concerns.

Crimean Congo haemorrhagic fever does not cause clinical disease in animals. Therefore, the consequences in terms of the adverse effect on animals would be negligible.

Crimean Congo haemorrhagic fever is, however, a serious zoonotic disease with a case fatality rate of 5% to 50%. The people that may be exposed to infection would be limited to zoo and veterinary staff. However, if domestic ruminants are released to farms carrying infected ticks, farm staff could be exposed to CCHFV if bitten by infected ticks.

The likelihood of transmission of CCHFV to domestic and wildlife ruminants outside of the zoo is assessed as very low.

Indirect consequences would entail the costs for control and surveillance in the unlikely event of CCHF spreading to the domestic ruminant population.

There may be additional costs to New Zealand for control in the event that CCHF is detected.

As CCHF is an OIE-listed disease, there are likely to be negative trade impacts if New Zealand has an incursion.

Therefore, the overall consequences as a result of a CCHF incursion are assessed as moderate.

10.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for CCHFV is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae, Tragulidae.

Therefore, risk management measures can be justified.

10.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Crimean Congo haemorrhagic fever is an OIE-listed disease affecting multiple species.
- *Crimean Congo haemorrhagic fever virus* has not been reported in New Zealand and is a notifiable disease.
- Crimean Congo haemorrhagic fever is a zoonotic disease.
- Crimean Congo haemorrhagic fever has never occurred in animals in Australia, Singapore, the USA, Canada and the UK.
- Domestic ruminants are regarded as the main hosts of CCHFV.
- Studies suggest a viraemic period of up to 2 weeks in domestic and wildlife ruminants.
- *Crimean Congo haemorrhagic fever virus* infections are generally subclinical in animal hosts.
- Transmission is via the principal tick vector (*Hyalomma* spp.) as well as exposure to infected blood and bodily fluids (in humans).
- Diagnostic methods include real-time RT-PCR and ELISA tests.
- There is no evidence to indicate that CCHFV is found in animal semen or can be transmitted via AI.

10.3.1 Options

One or a combination of the following options may be used:

Crimean Congo haemorrhagic fever orthonairovirus is not identified as a hazard in semen. Therefore, risk management measures for semen are not warranted.

Option 1

1. Country freedom for CCHF; AND
2. Animal(s) were resident in CCHF-free countries since birth.

Option 2

1. There have been no reported cases of CCHF in animals or personnel at the exporting facility during the 6 months prior to export; AND
2. Animals were in pre-export isolation for at least 28 days; AND
3. Animals were treated with an acaricide for external parasites prior to export (at the start of the pre-export isolation period); AND
4. Animals were examined for external parasites (at the end of the pre-export isolation period) and deemed free of parasites; AND/OR
5. Animals are treated with an acaricide on arrival at their destination.

The 28 day isolation period is twice the suggested viraemic period (14 days).

11 Epizootic haemorrhagic disease

11.1 Technical review

11.1.1 Aetiological agent

Family: *Reoviridae*
Genus: *Orbivirus*
Species: *Epizootic haemorrhagic disease virus* (EHDV) (ICTV, 2019)

Ten EHDV serotypes (EHDV 1 through EHDV 8, EHDV 318, and Ibaraki virus) have been described.

Ibaraki disease is caused by the Ibaraki strain of EHDV 2 (formerly Ibaraki virus).

Epizootic haemorrhagic disease virus is the causative agent of epizootic haemorrhagic disease (EHD).

11.1.2 OIE list

Epizootic haemorrhagic disease is an OIE-listed disease.

11.1.3 New Zealand status

New Zealand is free from EHDV (WAHIS, 2019d), as reported to the OIE.

The arbovirus surveillance programme has also demonstrated freedom from EHDV (Peacock et al., 2019).

Epizootic haemorrhagic disease is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

11.1.4 Zoonotic potential

Epizootic haemorrhagic disease is not a zoonotic disease.

11.1.5 Epidemiology

Host range

Epizootic haemorrhagic disease is a naturally occurring disease of cervids.

Various cervid species can be infected with EHDV (Dubay et al., 2004). White-tailed deer are highly susceptible (Brodie et al., 1998).

Clinical cases of EHD have been reported in yaks, American bison and bighorn sheep (Bovidae) in the USA, but rarely in sheep and alpacas (Brodie et al., 1998; Stevens et al., 2015).

Outbreaks and sporadic cases caused by various serotypes, including EHDV 1,
EHDV 2, EHDV 6 and EHDV 7, have been reported in cattle (Maclachlan & Osburn, 2004a).
Antibodies against EHDV have been found in goats, although the virus could not be recovered from experimentally infected animals (Nol et al., 2010).

Captive and free-ranging Arabian oryxes were identified as seropositive for EHDV (Frölich et al., 2005).

There is no published evidence of EHD in the Giraffidae and Tragulidae families.

Captive wild ruminants

There are no reports of EHD outbreaks in zoos.

There is, however, one case of an EHD outbreak in a captive ungulate research institute in the USA. Mortalities were initially experienced in white-tailed deer, which required further investigation. *Epizootic haemorrhagic disease virus* was detected in the deer via RT-PCR. Virus neutralisation tests

indicated that elks (Cervidae), bison, cattle and goats (Bovidae) were exposed to the virus. None of these animals showed clinical disease (Nol et al., 2010).

Captive and free-ranging Arabian oryxes were serologically surveyed for EHDV. Only animals in the captive herds were reported as seropositive (Frölich et al., 2005).

Geographical distribution

Epizootic haemorrhagic disease virus has been reported in North America (Pybus et al., 2014), South America, the Caribbean, Australia, Asia, Africa and the Middle East (WAHIS, 2019d).

Europe is currently regarded as free from EHDV, despite the virus being present on the southern and eastern rims of the Mediterranean Basin (Savini et al., 2011).

Singapore, the UK, UAE and South Africa have never reported cases of EHD (WAHIS, 2019d).

Ibaraki disease occurs in parts of Asia. Ibaraki disease was initially reported in Japan in 1959, and since then, there have been epizootics in East Asia (Bak et al., 1983), Korea and Taiwan.

Epizootic haemorrhagic disease viruses are found in temperate and tropical climates that support vector populations (Ruder et al., 2015).

Pathogenesis

Epizootic haemorrhagic disease is a non-contagious vector-borne viral disease.

The pathogenesis has only been described in experimentally infected cattle and naturally infected deer.

After infection has occurred by feeding of infected *Culicoides*, the virus replicates in the lymph nodes draining the sites of inoculation. The virus is then disseminated to secondary sites of replication such as the lung and spleen. Clinical signs are associated with widespread vascular injury and disseminated intravascular coagulation that leads to haemorrhage, oedema and tissue necrosis (Maclachlan & Osburn, 2004b).

Epizootic haemorrhagic disease virus could be isolated from experimentally infected cattle for a mean of 1 to 4 weeks, with a maximum of 2 to 8 weeks depending on the serotype. The Australian EHDV serotypes 2, 5, 6, 7 and 8 caused no clinical disease in cattle (Uren, 1986; Weir & Agnihotri, 2014). The duration of acquired immunity in cattle is still unknown, but evidence from natural infections suggests it may last for life (OIE Terrestrial Manual, 2018o).

In deer, the incubation period for EHD is estimated to be 5 to 10 days (Ruder et al., 2012).

Deer have reported to be viraemic up to day 21 post infection (Stallknecht et al., 1997).

In a study by Ruder et al. (2012), some white-tailed deer were viraemic for up to 2 months in laboratory experiments, although most deer seemed to clear the virus by 3 weeks (Ruder et al., 2012).

Viraemia in EHDV-infected ruminants may be prolonged due to their association with the animals' erythrocytes (Stallknecht et al., 1997).

Clinical signs

The range of clinical signs vary from subclinical infections to severe disease depending on serotype, breed of ruminant and geographical location (Weir & Agnihotri, 2014).

Cattle

Natural EHDV infection in domestic ruminants causes subclinical or mild to moderate transient febrile disease (Weir & Agnihotri, 2014). However, there has been an apparent increase in clinical disease in cattle associated with EHDV infection in numerous parts of the world, including Reunion Island (Bréard et al., 2004). Morbidity from the Reunion Island outbreaks ranged from 0.8% to 7%, with case fatality rates of 11% (Bréard et al., 2004; Sailleau et al., 2012).

Ibaraki disease, which occurs among cattle in parts of Asia, can result in mortality rates of up to 10% (Inaba, 1975; Weir, 2003). Common clinical signs of Ibaraki disease include fever, anorexia, conjunctival injection with lachrymation, nasal discharge and foamy salivation. Infected animals may develop oedema, haemorrhages, erosions or ulcerations in the mouth, on the lips and around the coronets. They can also be stiff and lame, and the skin may be thickened and oedematous (Inaba,

1975). Swallowing disorders, which are the pathognomonic sign of Ibaraki disease, occur in 20 to 30% of affected animals.

Deer
Clinical cases in white-tailed deer range from peracute illness, with death often occurring within 36 hours, to a more chronic course with animals remaining ill for several weeks.

In clinical cases, there may be fever, anorexia, lethargy, weakness, stiffness/lameness, respiratory distress, and severe and rapid oedema of the head and neck. Swelling of the mucous membranes of the oral cavity and swelling and hyperaemia of the conjunctiva are common. Ulcers and erosions in the oral cavity can result in excessive salivation and nasal discharge, which may both be blood-tinged (Weir & Agnihotri, 2014).

In parts of the USA, EHDV 1 and EHDV 2 are enzootic in white-tailed deer where they cause minimal or no clinical disease in these animals.

This difference in viral pathogenesis is attributed to differences in virulence of viral serotypes, increased host susceptibility in non-enzootic areas, and geographic variance in vector competency (Brodie et al., 1998).

Clinical disease has not been described in species of captive wild Bovidae, Giraffidae or Tragulidae within the scope of this IRA.

Transmission
Epizootic haemorrhagic disease virus is transmitted by biting midges of the genus *Culicoides*, which act as biological vectors. Female *Culicoides* become persistently infected and can transmit the virus after approximately 10 to 14 days.

Mosquitoes or other blood-sucking insects might theoretically be able to transmit this virus mechanically. There is one report of virus isolation (EHDV 4) from 2 *Anopheles* mosquitoes in Asia, but no transmission studies have been conducted (Brown et al., 1992). These mosquitoes were collected in 1980 and 1981. These insects are thought to have little or no role in the epidemiology of the disease.

Experimentally infected deer (Cervidae) can also shed EHDV in oral secretions and faeces; however, this is not thought to be significant in transmission, except possibly where animal densities are high in captive populations (Gaydos et al., 2002). This has not been described in Bovidae species.

Iatrogenic transmission has not been reported.

Diagnosis
The virus can be identified with techniques such as immunofluorescence, serogroup-specific sandwich ELISAs or RT-PCR assays. Methods to identify the viral serotype include virus neutralization or plaque inhibition tests with reference antisera, or serotype-specific RT-PCR assays (OIE Terrestrial Manual, 2018o).

Currently available serological tests include ELISAs, virus neutralization and agar gel immunodiffusion (AGID).

OIE recommends a monoclonal antibody-based competitive ELISA (C-ELISA). Agar gel immunodiffusion and some ELISAs cannot distinguish EHDV from bluetongue or other orbiviruses.

Antibodies to EHDV can usually be found 10 to 14 days after the animal was exposed, and neutralizing antibodies and viruses may be found concurrently in infected animals. Many deer and cattle have pre-existing antibodies to EHDV, and a rising titre should be diagnosed with paired serum samples (OIE Terrestrial Manual, 2018o).

Treatment, control and prevention
Prophylactic or therapeutic strategies may be used in enzootic areas.

Treatment of EHDV-infected wildlife ruminants is impractical. Livestock rarely suffer clinical disease and do not require treatment. An exception to this is infection with Ibaraki disease in cattle. Clinical signs in these animals may be prevented or treated to limit mortalities (Maclachlan et al., 2015).

Prevention in these animals could include protection from vectors and vaccination against EHDV.

The practicalities of vector protection are quite difficult in both free-ranging and pastoral domestic ruminants (Maclachlan et al., 2015). However, in zoological settings, housing valuable susceptible species in fully vector-protected enclosures may be achievable during outbreaks.

In countries such as the USA and Australia, the disease is notifiable, and domestic and wildlife ruminants are regularly surveyed. Additionally, Australia has border precautions and further monitoring of domestic and wildlife ruminants (WAHIS, 2019b).

In countries that are free from EHD, preventing the introduction of infected animal hosts and vectors would be an appropriate control measure.

Semen

There is no published evidence demonstrating the presence of EHDV in semen of wildlife ruminants.

The OIE have included risk mitigation measures for semen of bovids and cervids by making inferences from BTV, which is an *Orbivirus* similar to EHDV, and the severe economic impact the disease could have if introduced into a country.

Bluetongue virus has been detected in the semen of cattle. However, there is currently no known published evidence of EHDV in the semen of Cervidae or other wild or domestic ruminants (EFSA Panel on Animal Health and Welfare (AHAW), 2009).

11.1.6 Hazard identification conclusion

Epizootic haemorrhagic disease is an OIE-listed disease with the ability to affect multiple species. Natural hosts are cervids, with the disease also occurring in cattle.

There is only a single report of serological evidence of EHDV exposure in a captive wild Arabian oryx. There was no evidence of clinical disease in the oryx or transmission of EHDV to other animals.

There is also no evidence of EHDV in species of the Giraffidae and Tragulidae families.

A review of the literature found insufficient evidence to suggest that captive wild Bovidae, Giraffidae or Tragulidae play a significant epidemiological role in EHDV.

There is no evidence demonstrating the presence of EHDV in semen of wildlife ruminants.

Epizootic haemorrhagic disease virus is not identified as a hazard in captive wild Bovidae, Giraffidae, Tragulidae and their semen and will not be assessed further.

12 Foot and mouth disease

12.1 Technical review

12.1.1 Aetiological agent

Family: *Picornaviridae*
Genus: *Aphthovirus*
Species: *Foot-and-mouth disease virus* (ICTV, 2019)

Foot-and-mouth disease virus (FMDV) is the causative agent of foot and mouth disease (FMD).

The virus exists as 7 serotypes (O, A, C, Asia1 and Southern African Territories (SAT) 1–3, with multiple subtypes in each serotype) (Domingo et al., 2003).

12.1.2 OIE list

Foot and mouth disease is an OIE-listed disease affecting multiple species (OIE, 2020d).

12.1.3 New Zealand status

New Zealand has never experienced an FMD outbreak and is officially free from FMD (WAHIS, 2019d).

According to the OIE's "List of FMD free Members", New Zealand is regarded as FMD-free where vaccination is not practised (OIE, 2020b).

Foot and mouth disease is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

12.1.4 Zoonotic potential

Foot and mouth disease is a zoonotic disease. Isolation of the virus has been documented in more than 40 human cases. The occurrence in humans is rare (Bauer, 1997). Clinical manifestation of infections are mild and self-limiting and include fever, sore throat and blisters on the feet and in the mouth (Prempeh et al., 2001).

12.1.5 Epidemiology

Host range

Foot and mouth disease is primarily a disease of artiodactyls (even-toed ungulates). It affects all cloven-hoofed animals and camelids.

Foot and mouth disease has been reported in Cape buffalo and various antelope species (Bovidae) (Karesh, 2012; Shimshony et al., 1986), giraffes (Giraffidae) (Thomson et al., 2003) and chevrotains (Tragulidae) (OIE/FAO Reference Laboratory Network for Foot-and-Mouth Disease, 2018).

Captive wild ruminants

There have been numerous reports of FMD infections in animals in zoological collections.
In 1927, 2 bison and 3 buffalo (Bovidae) were infected with FMDV in a Copenhagen zoo (Denmark).

Reindeer (Cervidae) at a zoological farm of the Siberian Veterinary Institute contracted FMD from in contact cattle in 1928 (Boïko & Shuliak, 1974).

Foot and mouth disease outbreaks in captive wild animals were reported at the Paris Zoo in 1937. Of the 250 susceptible animals, 32 showed clinical signs and 4 died (Schaftenaar, 2002).

In 1951, an outbreak was reported in Rotterdam Zoo (Netherlands). Captive yak (Bovidae) were the only animals reported to have shown clinical signs of FMD.

In 2014, an FMD outbreak was reported in a wildlife park in Murree (Pakistan). Of the various susceptible animals present at the park, yak again were the only animals to experience clinical FMD (Abubakar et al., 2015).

It should be noted that the above species are not included within the scope of this IRA.

An FMD outbreak at the Mahendra Chaudhary Zoological Park in Punjab's Chhatbir led to the death of numerous captive wild animals including gaur, blackbuck, chowsingha and mouse deer (Bovidae and Tragulidae) (OIE/FAO Reference Laboratory Network for Foot-and-Mouth Disease, 2018).

The above non-exhaustive list demonstrates that captive wild ruminant species are highly susceptible to FMD and that the disease is likely to enter zoos in the absence of appropriate biosecurity measures.

In the mid to late 1900s, European zoos carried out regular vaccination programmes for selected susceptible animals in their collections. Zoos in the European Economic Community ceased preventive FMD vaccination programmes in 1991 as a result of the 1985 Council Directive 85/511/EEC, which banned preventive vaccination against FMD. As this directive remains in force under the European Union, EU member states do not vaccinate for FMD. In 2002, some zoos outside the EU were still vaccinating selected susceptible animals sporadically (mostly domestic stock in zoos for children) (Schaftenaar, 2002).

Geographical distribution

Evidence of FMD in wildlife has been reported globally, including in Asia, Middle East, Europe, North America and Africa (Karesh, 2012).

In terms of the approved countries, Australia, Singapore, the USA, Canada and the UK are FMD-free where vaccination is not practised (OIE, 2020b).

Foot and mouth disease is restricted to certain zones/regions in the RSA.

The disease was last reported in the UAE in domestic animals and wildlife in 2018.

Various countries within Europe remain affected by FMD (WAHIS, 2019d).

Pathogenesis

The most common route of infection in ruminants is inhalation. The initial site of replication is the respiratory bronchioles of the lungs. Other studies have suggested that viral replication takes place in the mucosa and lymphoid tissue of the pharynx (tonsillar region of the soft palate). The virus is then released into the efferent lymphatic system, resulting in the initial viraemia and spread to multiple organs and tissues. Development of the characteristic vesicular lesions is due to infection of the squamous epithelium and persistent local irritation. Lysis of these cells by ballooning degeneration results in release of intracellular fluid or focal intercellular oedema (Thomson & Bastos, 2004).

The incubation period is reported as 2 to 14 days (OIE Technical Disease Cards, 2013b).

A viraemia of approximately 6 days was demonstrated in domestic ruminants (cattle) (Windsor et al., 2011).

Carrier status in wildlife has been reported for various antelope species: up to 28 days in sable antelope (Ferris et al., 1989), 32 days in elands, 45 days in wildebeest and up to 160 days in greater kudus (Hedger et al., 1972; Karesh, 2012).

The Cape buffalo is the only known wildlife reservoir of FMDV (Thomson & Bastos, 2004).

Clinical signs

Different species and breeds of animals develop variable clinical signs of FMD. This variation depends upon the susceptibility to infection, viral strain differences and the routes and rates of viral excretion by different species after infection (Thomson et al., 2003).

In cloven-hoofed domestic animals, clinical manifestation is exhibited by anorexia, pyrexia, lameness and the development of vesicles and erosions in the mucosa of the mouth and skin of interdigital spaces and coronary bands. The disease is usually characterised by high morbidity and low mortality (Thomson & Bastos, 2004).

Clinical signs in wildlife ruminants vary and are similar to those described in domestic ruminants. However, mortality rates tend to be higher.

Infections in wildlife ruminants range from inapparent in Cape buffalo to high mortality in mountain gazelle (Karesh, 2012).

In a 10-year study of impala in Kruger National Park (KNP), clinical signs of disease were only reported once in impala, while serological evidence of FMDV was regularly detected, implying that subclinical infections were present (Vosloo et al., 2009).

Other clinical signs observed in antelope species include neurological signs: impaired coordination of movements and rigidity of the limbs (Boĭko & Shuliak, 1974).

Animals that do not succumb to infection generally recover within 1 to 2 weeks. However, a carrier state may persist in some species (Weaver et al., 2013).

Transmission

The excretion and transmission of FMDV in wildlife has not been researched in great detail.

Transmission pathways are therefore an extrapolation from domestic animal scenarios. Pathways include:

- direct contact between infected and susceptible animals
- direct contact of susceptible animals with contaminated inanimate objects (hands, footwear, clothing, vehicles, etc.)
- consumption (primarily by pigs) of untreated contaminated meat products (swill feeding), ingestion of contaminated milk (by calves)
- artificial insemination with contaminated semen
- inhalation of infectious aerosols
- airborne, especially in temperate zones (up to 60 km overland and 300 km by sea) (OIE Technical Disease Cards, 2013b; OIE Terrestrial Manual, 2018p; Thomson et al., 2003).

With regards to aerosol transmission over long distances, a large concentration of infected animals is required to generate a plume of virus-containing aerosols derived from expired air to cause infection. This scenario is sometimes seen in severe outbreaks of large pig populations (Thomson et al., 2003).

High viral loads occur in oronasal secretions for 1 to 3 days prior to and for 7 to 14 days after the development of lesions. The virus has also been found in urine, faeces and meat (Thomson & Bastos, 2004).

Transmission by persistently infected domestic or wildlife animals to susceptible individuals has been demonstrated in the Cape buffalo and impalas. Transmission to other buffalo and cattle has been observed (Weaver et al., 2013).

Impalas in the KNP have been implicated in FMDV transmission to cattle. The study provided evidence of subclinical infections in impala and confirmed the potential role of impalas for propagating FMDV in southern Africa at the wildlife–livestock interface (Vosloo et al., 2009).

A study by Hargreaves et al. (2004) to investigate the spread of FMD to domestic ruminants outside a wildlife conservancy in Zimbabwe showed that antelope (impalas or kudus) were responsible for the transmission of FMDV to cattle.

Buffalo infected with SAT serotypes were introduced into the conservancy and were contained by a 1.8 metre high double fence system. Investigations revealed that antelope became infected after contact with buffalo and thereafter infected cattle outside the perimeter by jumping over the fences (Hargreaves et al., 2004).

Diagnosis
Diagnosis may be based on clinical signs and confirmatory laboratory diagnosis.

Diagnostic methods recommended by the OIE Terrestrial Manual (2018p) to confirm clinical cases and for eradication policies include agent identification via virus isolation, antigen-detection ELISA, lateral flow device and RT-PCR. Recommended methods for the detection of an immune response include ELISA for antibodies against structural and non-structural proteins and virus neutralisation tests (OIE Terrestrial Code, 2019b).

Treatment, control and prevention
If treatment is allowed by the national authority, hyper-immune serum can be administered parenterally (Schaftenaar, 2002). Mild cases can be treated symptomatically.

In countries that are affected by FMD, control is difficult and expensive. It includes animal and people movement control, vaccination, and culling of infected and in-contact animals. However, culling of wildlife has not been demonstrated as an effective means of control due to the difficulty in eliminating all infected individuals (Karesh, 2012).

Prevention rather than control is the ultimate goal when dealing with FMD. This is due to the extensive negative economic and ecological impacts of an FMD outbreak in a country that has been free from FMD.

The status of a country or zone will not be affected by applying official emergency vaccination to FMD-susceptible animals in zoological collections in the face of a FMD threat identified by the Veterinary Authorities, provided that certain OIE Code conditions are met (OIE Terrestrial Code, 2019b).

Semen
In an experimental study by Cottral et al. (1968), 16 bulls were infected with FMDV. It was demonstrated that FMDV could be isolated from bull semen as early as 12 hours after inoculation and up to 10 days. Five heifers artificially inseminated with semen from infected bulls and 5 heifers inseminated with FMDV in various diluents developed FMD (Cottral et al., 1968).

Foot-and-mouth disease virus has been detected in bull semen for up to 4 days before clinical signs of disease. Five days after lesions appeared, the virus was detected from the semen and trace amounts of the virus were still detected for an additional 37 days (Sellers et al., 1968).

In the FMD enzootic area in South Africa, FMDV was isolated from the semen and sheath wash of a wild seropositive Cape buffalo showing no clinical signs of FMD (Bastos et al., 1999).

Due to a lack of information demonstrating the presence of FMDV in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with FMDV-contaminated semen, extrapolation is made from domestic to wildlife ruminants.

12.1.6 Hazard identification conclusion
Foot and mouth disease is an OIE-listed disease affecting multiple species. The disease has been identified in wild and captive wild ruminants.

Foot-and-mouth disease virus has been isolated from the semen of domestic and wild ruminants (although not covered within the scope of this IRA), and thus, extrapolation is made to species within this IRA.

Foot-and-mouth disease virus is identified as a hazard in Bovidae, Giraffidae, Tragulidae and their semen.

12.2 Risk assessment

12.2.1 Entry assessment
Australia, Singapore, the USA, Canada and the UK are FMD-free. Foot and mouth disease is restricted to certain zones/regions in the RSA. The UAE and various countries within Europe remain affected by FMD.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

There is evidence of FMDV infection of captive wild ruminants covered in this IRA from approved countries. Clinical disease has been reported in animals such as bison, reindeer, giraffes, chevrotains and yaks in zoological collections. Subclinical and carrier states have also been demonstrated in various antelope species. It is plausible that animals with extended carrier states and subclinical infections, not exhibiting signs of FMD, could be passed as clinically sound for export or as donor males for semen collection.

Foot-and-mouth disease virus has been isolated in the semen of domestic and wild ruminants (although not in ruminants covered in the scope of this IRA), and thus, extrapolation is made to species within this IRA.

Foot and mouth disease is an OIE-listed disease with major trade impacts for affected countries. Approved, licenced premises with valuable exotic species are likely to have high biosecurity measures to protect their animals from FMD. Some non-EU European zoos may still be vaccinating animals within their collection.

Therefore, the likelihood of entry of FMDV via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) and their semen from FMD-affected countries is assessed as low.

12.2.2 Exposure assessment

Transmission routes include direct contact, contact with contaminated fomites, artificial insemination and inhalation of aerosols.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

Susceptible animals within the receiving institutes have a high likelihood of being exposed to FMDV from infected animals if they are in direct contact with these animals, in contact with contaminated material or within a 60-kilometre radius for aerosol transmission.

Numerous studies have demonstrated the transmission of FMDV from captive wild animals to livestock surrounding zoos, as well as vice versa. Susceptible livestock within a 60- to 300-kilometre (over sea) radius of zoos have the potential of being exposed to an airborne virus. However, the likelihood of this scenario is very low, as a large concentration of infected animals is required to generate a large enough plume of virus-containing aerosols derived from expired air to cause infection.

If domestic animals are kept at the zoo, they could become infected with FMDV. Should these domestic animals be released onto New Zealand farms, they could transmit FMDV to other domestic animals.

Susceptible species within zoological gardens have higher mortality rates than domestic ruminants. While a larger number of species may die, others may recover and become a source of infection for susceptible animals.

Domestic ruminants and pigs are shown to have higher morbidity and lower mortality rates. Foot and mouth disease is therefore likely to establish in these animal populations and become a source of infection for susceptible animals outside the zoo.

Contaminated semen will be inseminated into susceptible animals. Artificial insemination with contaminated semen has been proven to transmit FMDV infections to naïve domestic ruminants. There is evidence of FMDV in the semen of one wild ruminant species (Cape buffalo) with potential transmission to domestic ruminants.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of FMDV exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as high, the likelihood of exposure and establishment outside the zoo is assessed as low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae, Giraffidae and Tragulidae is assessed as low.

12.2.3 Consequence assessment

Foot and mouth disease is a highly contagious disease of cloven-hoofed animals.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

Infected animals are likely to infect susceptible species they come into contact with. If an outbreak of FMD in a zoological collection is not contained, the consequences of spreading to other susceptible species within the zoo and neighbouring livestock species will be very high.

Foot-and-mouth disease is an OIE-listed disease and has the potential to cause major trade and severe economic impacts for affected countries.

The impacts experienced by New Zealand in an FMD outbreak would have direct and indirect components.

Direct losses would include reduced production and changes in herd structure of livestock/domestic animals, mortality of valuable species within zoological collections and zoonotic risk.

Foot-and-mouth disease virus infections in humans are rare, and therefore, the likelihood of probable consequences for human health is low.

Indirect losses would be due to the costs of FMD control, poor access to markets (negative trade impacts) and limited use of improved production technologies (Knight-Jones & Rushton, 2013).

Foot and mouth disease affects multiple species, and therefore, New Zealand's primary industries, which include dairy, beef, sheep and goat, pork and deer, would be at risk. New Zealand exports over 90% of its beef and lamb – this amounts to 3.2% (NZ$7 billion) of New Zealand's gross domestic production.

It is estimated that an outbreak of FMD could cost New Zealand NZ$16 billion over 4–5 years (MPI Biosecurity New Zealand, 2017).

Therefore, the overall consequences as a result of an FMD incursion are assessed as high.

12.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for FMD is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

Therefore, risk management measures can be justified.

12.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Foot and mouth disease is an OIE-listed disease affecting multiple species.
- New Zealand is officially free from FMD.
- Foot and mouth disease is zoonotic, although occurrence is extremely rare.
- Foot and mouth disease affects all cloven-hoofed animals and camelids.
- Australia, Singapore, the USA, Canada and the UK are FMD-free where vaccination is not practised.
- South Africa, the UAE and various countries in Europe are affected by FMD.

- The incubation period is reported as 2–14 days.
- Mortality rates are higher in wildlife ruminants.
- Carrier status and subclinical infections have been reported for various Bovidae species.
- Transmission routes include direct contact, contact with contaminated fomites, artificial insemination and inhalation of aerosols.
- Diagnostic methods include virus isolation, antigen-detection ELISA, lateral flow device, RT-PCR, ELISA for antibodies against structural and non-structural proteins and virus neutralisation tests.
- *Foot-and-mouth disease virus* has been detected in cattle and Cape buffalo semen, and therefore, the assumption is made that FMDV would be present in semen of species covered in this IRA.
- Artificial insemination with FMDV-contaminated semen has resulted in the infection of naïve cattle.

12.3.1 Options

One or a combination of the following options may be used:

Option 1

1. Country/zone freedom for FMD; AND
2. Animal(s)/donor male(s) were resident in FMD-free countries/zones since birth.

Wildlife ruminants are known to have extended carrier states of up to 160 days, and thus, residency since birth in an FMD-free country/zone will ensure the animal is truly free.

Option 2

1. Country/zone freedom for FMD; AND
2. The following apply for animals and/or semen:

Animals:

a. The animals showed no clinical signs of FMD on day of shipment; AND
b. were resident since birth or for at least 6 months in a FMD free country/zone where vaccination is not practised; AND
c. if transiting an infected zone, were not exposed to any source of FMDV during transportation to the place of shipment.

This option is in accordance with the OIE Code chapter 8.8 and recommendations for the importation of FMD-susceptible animals from FMD-free countries or zones where vaccination is not practised or FMD-free compartments. The longer timeframe for residency has been included due to extended carrier states in wildlife ruminants.

Semen:

a. The semen's donor male(s) showed no clinical signs of FMD on the day of semen collection and for the following 30 days; AND
b. the donor male(s) were resident for at least 6 months prior to collection in an FMD-free country or zone where vaccination is not practised.

This option is in accordance with the OIE Code chapter 8.8 and recommendations for the importation of domestic ruminant and pig semen from FMD-free countries or zones where vaccination is not practised or FMD-free compartments. The longer timeframe for residency has been included due to extended carrier states in wildlife ruminants.

Option 3

Semen:

1. The semen's donor male(s) showed no clinical signs of FMD on the day of semen collection and for the following 30 days; AND
2. the donor male(s) were resident since birth or for at least 6 months at a facility where no animal had been added in the 30 days before collection and where no FMD had occurred within a 10-kilometre radius of the facility for the 30 days before and after collection; AND
3. a straw from each semen sample was subjected to an MPI-approved test for FMDV with negative results; AND

4. semen was stored in the country of origin for a period of at least one month following collection, and during this period, no animal at the facility where the donor males were kept showed any sign of FMD.

This option is similar to the recommendations of the OIE Code for the importation of frozen semen from domestic ruminants and pigs from FMD-infected countries and zones.

13 Infectious bovine rhinotracheitis

13.1 Technical review

13.1.1 Aetiological agent

Family: *Herpesviridae*
Genus: *Varicellovirus*
Species: *Bovine alphaherpesvirus 1* (BHV 1) (ICTV, 2019)

Bovine alphaherpesvirus 1 is the causative agent of infectious bovine rhinotracheitis/infectious pustular vulvovaginitis (IBR/IPV).

There are 3 subtypes of BHV 1, namely BHV 1.1, BHV 1.2a and BHV 1.2b. The BHV 1.1 subtype is more virulent than the BHV 1.2 subtypes (Edwards et al., 1990).

13.1.2 OIE list

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis (IBR/IPV) is an OIE-listed disease of cattle (OIE, 2020d).

13.1.3 New Zealand status

In New Zealand, IBR/IPV causing respiratory disease and genital lesions is prevalent in the cattle population. The virulent strains causing abortion and encephalitis have not been reported (Durham, 1974; Fastier, 1967; Horner, 1990).

In a study of 28 isolates collected over 28 years, BHV 1.2b was the most prominent subtype identified in New Zealand. BHV 1.2a and the more virulent BHV 1.1 subtypes have not been isolated in New Zealand (Wang et al., 2006).

Bovine alphaherpesvirus 1 (abortifacient strain) causing IBR abortion is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

13.1.4 Zoonotic potential

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is not a zoonotic disease (EFSA AHAW Panel, 2017).

13.1.5 Epidemiology

Host range

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is a disease primarily affecting domestic and wild cattle (OIE Terrestrial Manual, 2018s).

Other domestic ruminants susceptible to infection include goats, sheep and camelids (Babiuk et al., 2008; OIE Terrestrial Manual, 2018s).

Cervids, water buffalo, Cape buffalo and wildebeest (Mushi & Karstad, 1979; Mushi et al., 1979) (Bovidae) are susceptible to infection (Babiuk et al., 2008).

Serum surveys of wild ruminants in Africa, Great Britain and North America, demonstrated BHV 1 antibodies in 21 species (Doyle & Heuschele, 1983b). Animals that showed seropositivity to BHV 1 included buffalo, eland, impala, kob, Thomson's gazelle, topi (Bovidae) (Rampton & Jessett, 1976) and giraffe (Giraffidae) (Anderson & Rowe, 1998).

There is no published evidence of IBR/IPV in species of the Tragulidae family.

Captive wild ruminants

A serosurvey of various US zoos was conducted to determine the BHV 1 seroprevalence in captive wild ruminants. Captive wild ruminants of the Bovidae and Giraffidae families showed antibodies to BHV 1. Of the 1,146 sera that were tested, only 34 were positive (Doyle & Heuschele, 1983b).

Experimental infections of wild wildebeest that were brought into captivity resulted in clinical IPV/infectious pustular balanoposthitis (IPB) of these animals when immunocompromised (Mushi & Karstad, 1979; Mushi et al., 1979).

Geographical distribution

Bovine alphaherpesvirus 1 has a worldwide distribution.

Bovine alphaherpesvirus 1.2b is present in Australia and causes disease in domestic ruminants. BHV 1.1 and BHV 1.2a have never occurred in Australia (Australian Government, 2005; WAHIS, 2019d).

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is present in domestic ruminants in the USA and is notifiable (WAHIS, 2019d). All subtypes of BHV 1 are recognised in the USA (d'Offay et al., 1995).

Canada and Japan have demonstrated clinical IBR/IPV in domestic ruminants (WAHIS, 2019d).

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is also present in 23 EU member states (EFSA AHAW Panel, 2017), South Africa and the UAE (WAHIS, 2019d) and is the cause of significant disease in domestic ruminants in the UK (Graham, 2013).

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis has never occurred in Singapore (WAHIS, 2019d).

Pathogenesis

Bovine alphaherpesvirus 1 is the cause of various disease syndromes namely infectious bovine rhinotracheitis, IPV/IPB, conjunctivitis, abortion and meningoencephalitis depending on the clinical presentation.

Factors such as immune status of the animal, dose of the virus and route of exposure may influence the severity and type of clinical manifestation (Babiuk et al., 2008).

In the respiratory form of the disease, a viral dose of as little as 10^3 to 10^4 is sufficient to cause infection. This explains why IBR is highly infectious between animals in close proximity. Once the virus enters the upper respiratory tract, it multiplies in epithelial cells, then moves to the lower respiratory tract. As it multiplies in epithelial cells, it causes destruction of the cells and alterations of the mucociliary clearance mechanisms, resulting in respiratory disease (Babiuk et al., 2008).

The genital form of the disease occurs in both sexes, IPV in females and IPB in males. In domestic ruminants, IPV occurs 1 to 3 days after mating with an infected bull. Some may become infected and shed virus without showing genital lesions (Babiuk et al., 2008).

In captive wild ruminants, pathogenesis has only been studied in detail in wildebeest.

Bovine alphaherpesvirus 1 seropositive wildebeest that were captured from the Kenyan plain and relocated to a paddock to graze freely were studied for IBR/IPV (caused by BHV 1) (Mushi & Karstad, 1979). The animals were injected on 7 consecutive days with a corticosteroid. Signs of IPV developed 5 days after the start of corticosteroid injections (Mushi et al., 1979). In male wildebeest that were experimentally infected with BHV 1, IPB lesions developed 3 days post infection (Mushi & Karstad, 1979). This could imply that incubation period is approximately 3 to 5 days in wildebeest.

The virus in these animals could not be isolated prior to corticosteroid injections. The virus could, however, be isolated repeatedly from vaginal swabs from day 5 until the lesions started to heal. The virus could not be isolated from blood, nasal or ocular secretions (Mushi et al., 1979). In the experimentally infected male and female wildebeest, the virus was isolated from preputial swabs from day 1 to day 14 post infection and from vaginal swabs from day 2 to 11 post infection, respectively (Mushi & Karstad, 1979). This could suggest a viraemic period of approximately 1 to 14 days.

Wildebeest that developed clinical signs of IPV were serologically positive to BHV 1 antibodies before and after corticosteroid injections (Mushi et al., 1979). This would imply that wildebeest were healthy carriers of BHV 1 and due to latency, they began experiencing clinical disease after stressful interactions and the immunosuppression of corticosteroids.

It is presumed that all BHV 1-infected animals are likely to progress into latency or chronic infections, which is a characteristic of alphaherpesviruses.

A carrier status similar to the carrier status of cattle has been demonstrated in wildebeest (Mushi & Karstad, 1979).

Cervids, water buffalo, Cape buffalo and wildebeest may act as potential reservoirs of the virus (Babiuk et al., 2008). Of these species, only wildebeest fall within the scope of this IRA.

Clinical signs

In most susceptible ruminants, the virus causes localised lesions in the mucous membranes at the site of infection. These are characterised by vesicles that progress to pustules and then ulcerations or erosions that heal within 12 to 14 days (Babiuk et al., 2008).

Initial clinical signs in domestic ruminants include frequent urination, elevation of the tail and mild vaginal discharge. Genital infections may result in the development of vesicles, pustules, erosions or ulcers in the mucosa of the vulvar and the vagina (Babiuk et al., 2008).

In New Zealand, BHV 1 infections are subclinical or result in disease with mild clinical signs (Lawrence, 2012; Vermunt & Parkinson, 2000a).

Infectious pustular vulvovaginitis in wildebeest were characterised by yellowish raised plaques and hyperaemia of the vulva and vagina. There was a mucoid to mucopurulent discharge from the vagina. The plaques progressed to pustules and healed in 10 to 14 days without scarring. Abortion was also observed in some animals. Respiratory disease was absent. Although abortions were observed in these study animals, further observations of these captive wildebeest have demonstrated that being carriers of BHV 1 had no apparent negative effect on reproduction (Mushi et al., 1979).

In experimentally infected male wildebeest that had BHV 1 instilled into the prepuce, signs of IBP developed. Preputial discharges ranged from serous to mucoid and copious to serous again. Pustules were observed on the preputial orifice edges (Mushi & Karstad, 1979).

Transmission

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is a contagious disease. Horizontal transmission via direct and indirect contact has been described.

Transmission of BHV 1 occurs mainly through the airborne (close contact between animals) and venereal routes. The airborne route is limited to a few metres (EFSA AHAW Panel, 2017). The study by Mushi et al. (1979) suggests that IBR/IPV in wildebeest is a venereal disease that is similar to the IBR/IPV venereal disease found in cattle.

The main source of the virus is secretions such as nasal exudates, respiratory droplets, genital secretions, semen, fetal fluids and tissues (Nandi et al., 2009).

Indirect transmission through contaminated feed, water and fomites may occur, as the virus is quite stable in the environment (Babiuk et al., 2008).

Transmission can also occur via AI with BHV 1-contaminated semen (van Engelenburg et al., 1995) from subclinically infected bulls (Babiuk et al., 2008).

The wildebeest study tested the theory of transmission of BHV 1 from wildebeest to cattle kept in close contact. No clinical signs or antibodies to BHV 1 developed in cattle. However, naïve heifers developed clinical signs of IPV when the wildebeest BHV 1 isolate was instilled intravaginally (Mushi et al., 1979).

A second study also showed that in-contact male wildebeest did not develop clinical signs or antibodies to BHV 1 after mingling with known BHV 1 female carriers for 3 months. However, after experimental preputial instillation of the virus, these wildebeest developed clinical signs of IPB. Intravaginal inoculation of BHV 1 in a wildebeest produced only mild vulvovaginitis. Experimental intranasal exposure of virus did not result in transmission of BHV 1 to wildebeest (Mushi & Karstad, 1979).

Diagnosis

Clinical diagnosis of BHV 1 infection can be done based on clinical, pathological and epidemiological findings. However, a definitive diagnosis can be achieved only by laboratory confirmatory tests (OIE Terrestrial Manual, 2018s).

Real time RT-PCR is the recommended test for agent identification to confirm clinical cases. Virus isolation may also be used, although there are some limitations to this method (OIE Terrestrial Manual, 2018s).

Serological tests for BHV 1 include ELISAs and virus neutralisation (VN). ELISA is the OIE-recommended test for identifying population freedom from infection, identifying individual animal freedom from infection prior to movement and determining immune status post vaccination. It is recommended as a component of eradication and surveillance policies (OIE Terrestrial Manual, 2018s).

Treatment, control and prevention

There is no specific treatment for IBR/IPV, except supportive care to treat the clinical signs.

The test and slaughter strategy has been effectively used in Europe (EFSA AHAW Panel, 2017).

Vaccination in domestic ruminants may reduce transmission within the herd, prevent the development of clinical signs and reduce shedding of the virus after infection. However, it does not completely prevent infection (OIE Terrestrial Manual, 2018s).

Modified live vaccines, inactivated vaccines and marker vaccines are available (Maresca et al., 2018). Vaccination with modified live vaccines can produce latent infections in cattle and intranasal vaccination with modified live vaccines can cause shedding of virus for 7 to 14 days (Biswas et al., 2013).

Quarantine, appropriate serological testing and knowledge of the status of introduced animals and their source herds could reduce the risk associated with trade (EFSA AHAW Panel, 2017).

Semen

Bovine alphaherpesvirus 1 has been isolated in the semen of experimentally infected bulls (van Engelenburg et al., 1995) and naturally infected bulls (Parsonson & Snowdon, 1975).

In van Engeleburg et al.'s study, the virus could still be isolated in the semen of experimentally infected bulls 120 weeks after the initial infection. During the testing period, there were a number of months during which no virus could be isolated. This again demonstrates the latent and intermittent shedding nature of the BHV 1 (Bitsch, 1973).

The virus can be transmitted by natural mating and AI. In the study by Parsonson and Snowdon (1975), naïve heifers were inoculated (into the uterus) with semen and BHV 1. All heifers developed IPV. In a second group, natural mating was allowed between 4 BHV 1-infected bulls and 9 naïve cows and heifers. Mating resulted in IPV lesions in the cows but did not affect pregnancy rates (Parsonson & Snowdon, 1975).

In wildebeest, BHV 1 has been isolated from preputial swabs. However, there are no studies to confirm its presence in semen of these animals. There is also no evidence of presence in semen of other species within the scope of this IRA. In the absence of such data, extrapolation is made from domestic ruminants.

13.1.6 Hazard identification conclusion

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is an OIE-listed disease of cattle.

Bovine alphaherpesvirus 1 has been detected in domestic ruminant semen and has the ability to cause infection in naïve cows after AI and natural mating. There is evidence that IBR/IPV in wildebeest is a venereal disease. However, BHV 1-contaminated semen in wildebeest has not been reported. In the absence of evidence in wildlife ruminants, extrapolation is made from domestic ruminants.

There is no evidence to suggest that IBR/IPV can infect or be carried by species within the Tragulidae family.

Bovine alphaherpesvirus 1 is not identified as a hazard in captive wild Tragulidae species and their semen and will not be assessed further.

Bovine alphaherpesvirus 1 (strains not present in New Zealand) is identified as a hazard in captive wild Bovidae and Giraffidae and their semen.

13.2 Risk assessment

13.2.1 Entry assessment

Bovine alphaherpesvirus 1 has not been reported in Singapore. Infectious bovine rhinotracheitis is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

There is serological evidence of BHV 1 in wild and captive wild ruminants from the approved countries. The antibody titres and seroprevalence were, however, higher in wild ruminants. Some wildlife ruminants are suggested to be reservoirs for BHV 1. Studies in wildebeest demonstrate that captive wild ruminants can be healthy carriers of BHV 1 that can develop clinical IPV/IPB and shed the virus during periods of immunosuppression.

Captive wild ruminants that are healthy carriers or subclinically infected with BHV 1 are likely to be passed as clinically sound for export or as donors for semen collection.

Bovine alphaherpesvirus 1 has been detected in domestic ruminant semen and can cause infection in naïve cows after AI and natural mating. There is evidence that IBR/IPV in wildebeest is a venereal disease. However, BHV 1-contaminated semen in wildebeest has not been reported. In the absence of such data, extrapolation is made from domestic ruminants.

Therefore, the likelihood of entry of BHV 1 via captive wild Bovidae, Giraffidae (within the scope of this IRA) and their semen from IBR/IPV-affected countries is assessed as very low.

13.2.2 Exposure assessment

Horizontal transmission has been described for BHV 1 infections.

Transmission of BHV 1 occurs mainly through the airborne (close contact between animals) and venereal routes. The main source of virus is secretions such as nasal exudates, respiratory droplets, genital secretions, semen, fetal fluids and tissues. Indirect transmission through contaminated feed, water and fomites may occur, as the virus is stable in the environment.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If BHV 1-infected animals are imported, they may shed virus if immunocompromised. Handling and relocation is a stressful period for captive wild animals.
Infected domestic ruminants can rapidly spread virus via secretions to in contact animals over short distances. However, in studies of BHV 1-infected wildebeest, the virus could not be isolated from blood, nasal or ocular secretions. Infection was also not transmitted to in-contact cattle. Therefore, the infectivity of the virus in captive wild ruminants is unknown.

Bovine alphaherpesvirus 1-infected captive wild ruminants may be a source of infection to other susceptible animals within the zoo, but the likelihood is assessed as low.

Some species of domestic and wildlife ruminants are reservoirs of BHV 1 and have latent infections. *Bovine alphaherpesvirus 1* could therefore establish in domestic and captive wild ruminants within the zoo.

Bovine alphaherpesvirus 1 can be transmitted via the airborne route over a few metres, but it is unlikely to survive long enough and at an infectious dose large enough to result in IBR/IPV of susceptible animals outside the zoo.

However, if domestic ruminants are kept at the zoo, they could become infected with BHV 1 strains not present in New Zealand. Should these domestic ruminants be released onto New Zealand farms, they could transmit BHV 1 to other domestic ruminants. *Bovine alphaherpesvirus 1.2b* has established in New Zealand, and therefore, other strains of BHV 1 could spread and establish in the domestic ruminant population.

There is uncertainty as to whether BHV 1 infected wildlife ruminants are likely to produce BHV 1-contaminated semen. It is also not known whether naïve wildlife ruminants will become infected with BHV 1 if BHV 1-contaminated semen is used for AI. However, by extrapolating the outcomes from domestic ruminants to wildlife ruminants, it is assumed that infection may occur. Captive wild ruminants (wildebeest) did become infected after intravaginal instillation of BHV 1.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of BHV 1 exposure and establishment within the zoo via infected captive wild Bovidae and Giraffidae is assessed as low, the likelihood of exposure and establishment outside the zoo is assessed as very low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae and Giraffidae is assessed as low.

13.2.3 Consequence assessment

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is an OIE-listed disease.

Bovine alphaherpesvirus 1.2b is widespread in the New Zealand beef and dairy herds. The disease conditions are currently being managed by vaccination, on-farm biosecurity and herd health management. Other strains have not been detected. There are more virulent strains such as BHV 1.1 in some approved countries. These strains are responsible for causing severe respiratory disease and negative impacts on reproduction and fertility.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

The more virulent strains of BHV 1 could enter via these animals and establish in domestic and captive wild ruminants within zoos. The direct consequences of this establishment would depend on the effect of these strains on susceptible animals. Captive wild ruminants are not significantly affected by BHV 1 infections. Mild lesions may appear and shedding of the virus may occur during periods of immunosuppression.

If domestic ruminants are kept in the same enclosure as imported captive wild ruminants, these animals may contract the virus and succumb to more severe disease and possibly higher mortality rates. If they are released into New Zealand farms, they could transmit BHV 1 to other cattle, sheep, goats and deer. More virulent strains could result in abortions and encephalitis of these animals.

Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is not a zoonotic disease, and therefore, the consequences for human health are negligible.

Indirect consequences would include the costs for testing and surveillance of susceptible captive wild ruminants. *Bovine alphaherpesvirus 1* has led to abortions in captive wild ruminants, but this does not have a long-term effect and would therefore not affect the breeding programmes of the zoos.

There would be additional costs to New Zealand for BHV 1 (strains not present) control in the event of an incursion in domestic ruminants.

Therefore, the overall consequences as a result of a BHV 1 (strains not present in New Zealand) incursion are assessed as low.

13.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for BHV 1 (strains not present in New Zealand) is non-negligible, and it is assessed to be a risk in captive Bovidae, Giraffidae and their semen.

Therefore, risk management measures can be justified.

13.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is an OIE-listed disease.
- There are 3 subtypes of BHV 1, namely BHV 1.1, BHV 1.2a and BHV 1.2b.
- Subtype BHV 1.2b is present in New Zealand.
- Infectious bovine rhinotracheitis/infectious pustular vulvovaginitis is not a zoonotic disease.
- *Bovine alphaherpesvirus 1* can infect multiple domestic and wildlife ruminants.
- There is no evidence of IBR/IPV in species of the Tragulidae family.
- *Bovine alphaherpesvirus 1* is present in all approved countries except Singapore.
- Transmission can occur via direct (airborne over short distances and venereal) and indirect contact (secretions and excretions, and contaminated feed, water and fomites).
- Diagnosis includes the use of real-time RT-PCR, ELISAs and VN tests.
- *Bovine alphaherpesvirus 1* has been detected in the semen of domestic ruminants, and therefore, extrapolation is made to captive wild ruminants. Infection has been transferred to captive wild ruminants via the installation of BHV 1 into the vagina.

13.3.1 Options

One or a combination of the following options may be used:

Bovine alphaherpesvirus 1 is not identified as a hazard in species within the Tragulidae family and their semen, and therefore, risk management measures are not warranted for these species.

Option 1

1. Country freedom for BHV 1 (strains not present in New Zealand); AND
2. the animal(s)/donor male(s) were resident in BHV 1 (strains not present in New Zealand) free countries since birth; AND
3. the animal(s)/donor male(s) showed no clinical signs of disease on the day of export or semen collection.

This option allows animals to circumvent testing requirements. Residency since birth is recommended due to unknown periods of latency and carrier states.

Option 2

Animals:

1. For 180 days immediately before export, the animal(s) were continuously resident at the premises where no clinical, epidemiological or other evidence of BHV 1 (strains not present in New Zealand) occurred during the previous 12 months; AND
2. in the 30 days immediately before export, blood samples from the animal(s) were tested (using an MPI-approved test) for BHV 1 (strains not present in New Zealand) twice at an interval of no less than 21 days. The test results were negative.

Semen:

1. The semen was tested by a virus isolation or real-time RT-PCR for BHV 1 (strains not present in New Zealand), with negative results; OR
2. donor male(s) were held in isolation during the period of collection and for the 30 days following collection and were subjected to an MPI-approved test for BHV 1 (strains not present in New Zealand) on a blood sample taken at least 21 days after collection of the semen, with negative results.

This option is similar to the recommendations of the OIE Code for the importation of cattle destined for herds free from IBR/IPV and the importation of frozen semen.

Option 3

1. For 180 days immediately prior to export or collection of semen, the animal(s)/donor male(s) was part of a captive wild animal collection where no clinical, epidemiological or other evidence of BHV 1 (strains not present in New Zealand) occurred during the previous 12 months; AND

2. For 180 days immediately prior to export or collection of semen, the animal(s)/donor male(s) were part of a captive wild animal collection subject to a documented BHV 1 screening program. The screening program must include:

    a. Diagnostic testing of all captive wild bovids in the collection, performed prior to the animal(s) entering the collection. The diagnostic tests must be of a type approved by MPI. The testing regime must be able to identify infected or healthy carrier animals; AND
    b. the collection must have been a 'closed herd' during that time.

This option allows animals to circumvent pre-entry isolation testing requirements if the exporting facility can prove BHV 1 freedom in the captive wild animal collection.

14 Lumpy skin disease

14.1 Technical review

14.1.1 Aetiological agent

Family: *Poxviridae*
Genus: *Capripoxvirus*
Species: *Lumpy skin disease virus* (LSDV) (ICTV, 2019)

Lumpy skin disease virus is the causative agent of lumpy skin disease (LSD).

14.1.2 OIE list

Lumpy skin disease is an OIE-listed disease of cattle (OIE, 2020d).

14.1.3 New Zealand status

New Zealand is free from LSD (WAHIS, 2019d), as reported to the OIE.

Lumpy skin disease is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

14.1.4 Zoonotic potential

Lumpy skin disease is not a zoonotic disease (OIE Technical Disease Cards, 2017).

14.1.5 Epidemiology

Host range

Lumpy skin disease virus is highly host-specific. It usually causes disease in cattle and water buffalo (Bovidae) (OIE Technical Disease Cards, 2017). These species are not included within the scope of this IRA.

A serological survey of 3,445 sera (44 wild ruminants) in Africa demonstrated neutralising antibodies to LSDV in 6 species: kudu, waterbuck, reedbuck, impala, springbok (Bovidae) and giraffe (Giraffidae). The low seroprevalence of positive animals suggested that wild ruminants in Africa do not play a significant role as wildlife reservoirs (Hedger & Hamblin, 1983).

Experimental infections of calves of giraffes (Giraffidae), impalas and buffaloes, and adult wildebeest (Bovidae) led to the death of the giraffe and impala calves after succumbing to typical signs and lesions of LSD. The buffalo calf and wildebeest were not clinically or serologically affected (Young et al., 1970).

Lumpy skin disease has also been observed in a captive-bred female Arabian oryx (Greth & Schwede, 2007) in Saudi Arabia and has been suspected in springbok in Namibia and oryx in the RSA (Coetzer, 2004). A confirmed case of LSD was made in a wild springbok in the RSA in 2017 when skin lesions of the clinically infected animal were tested histopathologically (Last, 2017).

There is no published evidence of LSD in species of the Tragulidae family.

Captive wild ruminants

The case of LSD in an Arabian oryx in Saudi Arabia was a single case in the captive collection of 90 oryxes. The collection was created 3 years prior to the occurrence of this case. There were no introductions into the herd since its inception, and the oryx had no direct contact with other ungulates. After the first case of LSD, a serological survey was conducted, and 2 out of 90 oryxes were positive – the individual that suffered clinical LSD and another female kept in the same area.

There are no other reported cases of LSD in captive wild ruminant collections.

Geographical distribution
Of the approved countries, Australia, Singapore, the USA, Canada, Japan and the UK have never reported LSD (WAHIS, 2019d).

In 2012, outbreaks of LSD had spread from the Middle East to southeast Europe, affecting Greece, Bulgaria and countries in the Balkans (EFSA (European Food Safety Authority), 2019).

Lumpy skin disease is enzootic in most African countries (OIE Technical Disease Cards, 2017) including the RSA.

Cases of LSD have been reported in the UAE (Greth & Schwede, 2007; WAHIS, 2019d).

Pathogenesis
In experimentally infected domestic ruminants, local skin lesions appear 4 to 7 days post infection, while in more severe cases, generalised lesions appear 7 to 19 days after infection (Coetzer, 2004), implying an incubation period of 4 to 19 days.

Viraemia occurs intermittently from 7 to 21 days post infection (OIE Technical Disease Cards, 2017).

Viral replication in various cell types including cells of blood and lymph vessel walls cause vasculitis and lymphangitis in the affected areas (Coetzer, 2004).

Immunity in cattle after natural infection is lifelong (Coetzer, 2004).

Lumpy skin disease does not exhibit latency, and reactivation of the virus does not occur (OIE Technical Disease Cards, 2017).

In an experimentally infected giraffe calf, an incubation period of 6 days was recorded, compared to 25 days in an impala calf. The pathogenesis of the disease in these animals closely resembled that of the disease in cattle with the exception of the protracted incubation in impalas. The duration of disease in cattle is normally 14 days, compared to 6 and 15 days in impalas and giraffes, respectively (Young et al., 1970).

Serum samples from the impala showed no antibodies against LSDV on days 5 and 13 post infection.

Clinical signs
In experimentally infected domestic ruminants, 40% to 50% of animals will develop skin lesions only at the site of inoculation or no clinical signs at all. The rest will develop localised lesions, enlargement of regional lymph nodes and generalised eruption of skin lesions. Animals inoculated intravenously tend to become more severely affected (Coetzer, 2004).

Morbidity in cattle ranged from 1% to 20%, with a few cases reaching 50%. Mortality rates were usually less than 10%. Abortion occurred in 1% to 7% of cases. Severe economic losses in dairy farms were a result of mastitis (Coetzer, 2004).

In the experimentally infected giraffe, swellings developed at the sites of inoculation of LSDV. Swellings were painful to the touch, hard and firmly attached to the skin. The swellings gradually increased in size over 9 days with blackened skin over the swellings. By day 13 after appearing, the nodules became necrotic, oozing serosanguineous fluid. Several nodules could be palpated in the muscle tissue around the inoculation site. Fever developed 7 days after the appearance of the swellings and remained till shortly before death. Signs of systemic illness were observed 14 days after the development of swellings. The oral cavity revealed numerous nodules, erosions and ulcerations of varying size (Young et al., 1970).

The swellings observed in the impala were similar to those of the giraffe. However, the dissemination was diffuse over most of the body. Some nodules became detached, leaving depilated areas of skin. Numerous lesions in the mouth appeared 30 days after inoculation (Young et al., 1970).

Transmission
The modes of transmission from animal to animal and herd to herd are not fully understood.

Currently, it is proposed that the principal mode of transmission is mechanically by arthropod vectors. Mosquitoes (*Culex* and *Ades*), biting flies (*Stomoxys* and *Biomyia*) and ticks (*Riphicephalus* and *Amblyomma*) have been implicated in transmission (OIE Technical Disease Cards, 2017). New evidence suggests that the house fly (*Musca domestica*) may also play a role in transmission (Last, 2017; Sprygin et al., 2019).

In the captive Arabian oryx case, insect transmission from infected domestic sheep that graze along the perimeter fence was suggested as the potential explanation for the case (Greth & Schwede, 2007).

Direct contact plays a minor role, if any (OIE Technical Disease Cards, 2017).

Saliva of cattle has shown to be infectious in acute stages of LSD (OIE Technical Disease Cards, 2017).The virus could be detected for 11 days in saliva, 22 days in semen and 33 days in skin nodules (Coetzer, 2004), as well as in blood, ocular and nasal secretions (OIE Technical Disease Cards, 2017).

The persistence of skin nodules and scab material in the field may act as a source of virus for biting arthropods, thereby leading to further dissemination of LSD (Diallo & Viljoen, 2007).

Indirect transmission via fomites such as contaminated feed or water is unknown.

Diagnosis

The OIE recommends virus isolation for the confirmation of clinical cases and PCR for clinical cases and determining individual animal freedom from infection prior to movement (OIE Terrestrial Manual, 2013). Samples suitable for these tests are scabs, saliva, nasal secretions and blood (OIE Technical Disease Cards, 2017). From the first appearance of skin lesions, the virus can be isolated from skin nodules for 3 to 4 weeks and viral nucleic acid can be detected for up to 3 months (Tuppurainen et al., 2005).

Virus neutralisation is a suitable method for various purposes including population and individual animal freedom, clinical cases, surveillance, eradication and determining the immune status post vaccination (OIE Terrestrial Manual, 2013). The downfall is that serological tests cannot differentiate between *Sheeppox virus*, *Goatpox virus* and LSDV (OIE Technical Disease Cards, 2017) or between animals that are naturally infected and those that are vaccinated (Beard, 2016).

Treatment, control and prevention

Treatment may be symptomatic in valuable animals, as in the case of the oryx that was treated with antibiotics, anti-inflammatories and fluids until it recovered.

In countries that are affected with LSD, control measures could include movement restrictions, removal of clinically affected animals and vaccination (Beard, 2016). Appropriate disposal, such as incineration of dead animals, is recommended.

Control and eradication rely on early detection of the index case and rapid, widespread vaccination campaigns (OIE Technical Disease Cards, 2017).

Prevention for areas and countries bordering LSD-affected regions include surveillance over a distance of at least 20 kilometres from the affected area and vaccination to protect susceptible animals.

Trade restrictions on cattle, potential LSDV reservoir animals and their products from LSD-affected countries would aid in the prevention strategy of LSD-free countries.

Semen

Lumpy skin disease virus has been detected in the semen of domestic ruminants for extended periods (Irons et al., 2005; Weiss, 1968).

In a study to establish incidence and duration of LSDV excretion in semen, naïve bulls were experimentally infected with LSDV, observed and tested. Some bulls began excreting virus in their semen as early as 6 days post infection up until 159 days. The study demonstrated that shedding of

the virus in semen persisted well after the viraemic phase, seroconversion and clinical recovery. The infectivity of semen could not be determined in this study (Irons et al., 2005).

The study by Annandale et al. (2014) demonstrated that AI with LSDV-spiked semen was able to transmit infection to naïve heifers. However, it should be noted that the high virus titre used for insemination is unlikely to resemble the virus titre found in the semen of naturally infected bulls. The viral loads in semen of naturally infected bulls has not been established (Annandale et al., 2014; Irons et al., 2005).

There is no published evidence demonstrating the presence of LSDV in the semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with LSDV-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

14.1.6 Hazard identification conclusion

Lumpy skin disease is an OIE-listed disease of cattle. Lumpy skin disease has been reported in naturally infected captive wild Bovidae and experimentally infected wild Bovidae and Giraffidae.

Lumpy skin disease virus has been detected in domestic ruminant semen and has the ability to cause infection in naïve cows after AI. There is no literature evidence of LSDV in semen of species covered under this IRA or transmission of LSDV via contaminated semen of these species, and therefore, extrapolation is made from evidence found in domestic ruminants.

There is no evidence to suggest that LSDV can infect or be carried by species within the Tragulidae family.

Lumpy skin disease virus is not identified as a hazard in captive wild Tragulidae and their semen and will not be assessed further.

Lumpy skin disease virus is identified as a hazard in captive wild Bovidae, Giraffidae and their semen.

14.2 Risk assessment

14.2.1 Entry assessment

Lumpy skin disease virus has not been reported in Australia, Singapore, the USA, Canada, Japan and the UK. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and the imports are also likely to be infrequent. Therefore, the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

Serological surveys of African wild ruminants have shown low LSDV seroprevalence of various species covered within the scope of this IRA. Studies suggest that low seroprevalence meant that these species were not likely to be reservoir hosts of LSD. However, there have been suspected clinical cases of LSD in antelope species in Africa and the UAE. There has also been a confirmed clinical case of LSD in a wild springbok in the RSA. The scenario in the wild makes it difficult to determine the role that some wild animals play in the epidemiology of the disease, especially if the disease occurs at low prevalence rates. Sick or debilitated animals in the wild are likely to be at risk of predation or dying with their carcasses scavenged before being identified or a diagnosis is made. Therefore, these diseases are more likely to be identified in captive collections. Furthermore, experimental infections of giraffe and impala calves resulted in clinical LSD and the death of these animals. The incubation period in these wildlife ruminants ranged from 6 to 26 days.

If animals are imported while they are incubating LSDV and not showing any signs, they may go unnoticed and be passed as clinically sound for export or as donors for semen collection.

There has only been a single reported case of LSD in a captive collection in the UAE. Therefore, the occurrence of LSD in captive collections in affected countries is presumed to be very low.

Lumpy skin disease virus has been detected in domestic ruminant semen and has the ability to cause infection in naïve cows after AI. There is no literature on evidence of LSDV in semen of species

covered under this IRA or transmission of LSDV via contaminated semen of these species, and therefore, extrapolation is made from evidence found in domestic ruminants.

The likelihood of entry of LSDV via captive wild Bovidae and Giraffidae (within the scope of this IRA) and their semen from LSD-affected countries is assessed as very low.

14.2.2 Exposure assessment

The modes of transmission of LSDV are not fully understood. It is proposed that the principal mode of transmission is mechanically by arthropod vectors such as mosquitoes, biting and non-biting flies, and ticks. Transmission via direct contact is assessed to be insignificant, and indirect transmission via fomites is unknown.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If captive wild ruminants are subclinically infected or incubating LSD, they could be a source of virus for transmission by arthropod vectors. It is uncertain as to whether the viraemia achieved in these captive wild animals is able to infect arthropods, but we assume this could occur. New Zealand has various *Aedes* and *Culex* mosquitoes, *Stomoxys* and *Musca* flies and other arthropods that could act as mechanical vectors of LSDV. Vector competence has not been established in these species of arthropods in New Zealand. Due to the lack of understanding of the transmission pathways, it can be assumed that LSDV could be mechanically transmitted by the arthropods present in New Zealand.

As a result, captive wild ruminants within the zoo could be exposed to LSDV by vectors. It is uncertain whether these animals will be infected and exhibit clinical LSD. From experimental LSDV infections, species such as buffalo and wildebeest are resistant to infection, while impala and giraffe succumb to clinical disease and mortality. However, clinical, virological and serological studies suggest the likelihood that the disease would establish in these species is extremely low.

Arthropod vectors have the ability to travel over long distances, and therefore, there is a likelihood that they could reach domestic ruminants outside the zoo. Lumpy skin disease is host-specific and usually affects only cattle and water buffalo. Therefore, cattle would be the only susceptible species outside the zoo. The virus could establish in the naïve New Zealand cattle population. Infected cattle could then become a source for further spread of the disease. Spread of the disease to neighbouring regions and countries has been demonstrated in the Middle East.

Studies have demonstrated that AI with LSDV-spiked semen can transmit infection to naïve heifers. Infection as a result of naturally contaminated semen has not been demonstrated. The infectivity of semen is unknown. Infection has thus only been demonstrated experimentally in domestic ruminants but not wildlife ruminants. There is uncertainty as to whether LSDV-infected wildlife ruminants are likely to produce LSDV-contaminated semen. It is also not known whether naïve wildlife ruminants will become infected with LSDV if LSDV-contaminated semen is used for AI. However, by extrapolating the outcomes from domestic ruminants to wildlife ruminants, it is assumed that infection could occur.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of LSDV exposure and establishment within the zoo via infected captive wild Bovidae and Giraffidae is assessed as low, the likelihood of exposure and establishment outside the zoo is assessed as very low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae and Giraffidae is assessed as very low.

14.2.3 Consequence assessment

Lumpy skin disease is an OIE-listed disease of cattle.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

There may be direct consequences for imported captive wild ruminants that may have been incubating the disease. As reported, the clinical manifestations in wildlife ruminants are rare and varied. Some

may be seropositive and subclinically infected, and some may experience clinical disease and survive, while mortality may result in others.

Clinical signs of LSD are straightforward to identify. Animals with LSD in zoos would be rapidly identified, and a diagnosis is likely to be obtained swiftly. This would enable quick containment.

However, arthropod vectors cannot be contained. These vectors could spread the virus to other susceptible species within the zoo as well as susceptible cattle outside the zoo.

The direct consequences for the New Zealand cattle population would be as a result of morbidity and mortality rates, which have been reported to be 1% to 20% and under 10%, respectively. There could be severe negative impacts for dairy herds due to mastitis.

Lumpy skin disease is not a zoonotic disease, and therefore, the consequences for human health are negligible.

Indirect consequences would entail the costs for control and surveillance within the affected zoos. Should the disease spread further than expected, loss of valuable species within zoos may also occur. This would negatively impact their ASMP and conservation efforts.

If LSD spreads to the cattle population, establishment is likely to occur. Considerable costs could be incurred in order to contain and control the disease. If disease transmission is poorly understood, containment and control in the absence of vaccination would be difficult. If disease transmission is attributed to arthropods present in New Zealand, eradication may be difficult to achieve unless vaccination is implemented.

Lumpy skin disease is an OIE-listed disease, and therefore, negative trade impacts are likely.

Therefore, the overall consequences as a result of an LSD incursion are assessed as low.

14.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for LSD is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and their semen.

Therefore, risk management measures can be justified.

14.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Lumpy skin disease is an OIE-listed disease of cattle.
- New Zealand is free from LSD.
- Lumpy skin disease is not a zoonotic disease.
- *Lumpy skin disease virus* is highly host-specific. It usually causes disease in cattle and water buffalo.
- Serological evidence of LSD and clinical LSD have been described in captive wild Giraffidae and Bovidae species.
- There is no literature evidence of LSD in species of the Tragulidae family.
- Of the approved countries, Australia, Singapore, the USA, Canada and the UK have never reported LSD.
- Incubation period may be up to 25 days in wildlife ruminants.
- Viraemia occurs intermittently from 7 to 21 days post infection.
- The principal mode of transmission is proposed to be arthropod vectors.
- The virus could be detected for 11 days in saliva, 22 days in semen and 33 days in skin nodules as well as in blood, ocular and nasal secretions.
- Diagnosis includes VI, PCR and VN tests.
- *Lumpy skin disease virus* has been detected in the semen of infected domestic ruminants.
- *Lumpy skin disease virus*-spiked semen has resulted in the infection of naïve heifers.

14.3.1 Options

One or a combination of the following options may be used:

Lumpy skin disease virus is not identified as a hazard in species within the Tragulidae families and their semen, and therefore, risk management measures are not warranted for these species.

Option 1

Animals:

1. Country freedom for LSD; AND
2. the animal(s) were resident in LSD free countries since birth or at least 3 months; AND
3. the animals showed no clinical signs of disease on the day of export.

Semen

1. Country freedom for LSD; AND
2. the donor male(s) showed no clinical signs of disease on the day of collection; AND
3. the donor male(s) were resident in LSD-free countries since birth or at least 28 days prior to collection of semen.

This option is in line with the recommendations of the OIE Code for importation of bovine and water buffalo and their semen from countries or zones free from LSD.

Option 2

Animals:

1. For 180 days immediately before export, the animal was continuously resident in a country where no clinical, epidemiological or other evidence of LSD occurred during the previous 3 years, where the disease is compulsorily notifiable and where vaccination against LSDV has not occurred in the previous 3 years; AND
2. the animal showed no clinical signs of LSD during pre-export isolation; AND
3. the animal has not been vaccinated against *Capripoxviruses* in the previous 3 years (LSDV, sheeppox or goatpox strain vaccines).

Semen:

1. For 180 days immediately before semen collection, the donor male(s) were continuously resident in a country where no clinical, epidemiological or other evidence of LSDV occurred during the previous 3 years, where the disease is compulsorily notifiable and where vaccination against LSDV has not occurred in the previous 3 years.

Option 3

Animals:

1. The animals showed no clinical signs of LSD on the day of shipment; AND
2. the animals were resident since birth, or for the past 180 days prior to shipment, in an epidemiological unit where no case of LSD occurred during that period; AND
3. the animals were kept in pre-export isolation for the 28 days prior to shipment, during which time they were subjected to an agent identification test with negative results.

These recommendations are similar to those of the OIE Code for importation of bovine and water buffalo from countries or zones not free from LSD.

Semen:

1. The donor male(s) showed no clinical sign of LSD on the day of collection and the following 28 days; AND
2. the donor male(s) were kept for the 60 days prior to collection in a facility where no case of LSD occurred during that period; AND
3. the donor male(s) were subjected to a serological test to detect antibodies specific to LSDV, with negative results, at least every 28 days throughout the collection period and one test 21 days after the final collection for this consignment; AND
4. the donor male(s) were subjected to agent detection by PCR conducted on blood samples collected at commencement and conclusion of, and at least every 28 days during, semen collection for this consignment, with negative results; AND
5. A straw from each semen sample to be exported was subjected to LSDV detection by PCR.

These recommendations are similar to the recommendations of the OIE Code for importation of bovine and water buffalo semen from countries or zones not free from LSD.

15 Malignant catarrhal fever – wildebeest associated

15.1 Technical review

15.1.1 Aetiological agent

Family: *Herpesviridae*
Genus: *Macavirus*
Species: *Alcelaphine gammaherpesvirus 1* (AlHV-1) (ICTV, 2019)

The malignant catarrhal fever (MCF) subgroup of viruses, called malignant catarrhal fever viruses (MCFVs), contains at least 10 members, 6 of which are currently known to cause disease (OIE Terrestrial Manual, 2018t). *Alcelaphine gammaherpesvirus 1*, which falls within the MCFV, will be discussed under this chapter.

Alcelaphine gammaherpesvirus 1 (AlHV-1) is the causative agent of wildebeest-associated MCF (WA-MCF). It is enzootic in wildebeest populations worldwide.

15.1.2 OIE list

Malignant catarrhal fever and WA-MCF are not OIE-listed diseases. However, WA-MCF is part of the "Non OIE-listed diseases affecting wild animals". These are diseases that have not met the OIE's criteria to be listed. However, OIE experts of the working group on wildlife diseases have selected them to be monitored, both because of their importance for wild animals and for early warning purposes, in order to protect human and livestock health.

15.1.3 New Zealand status

Following a review of the literature, WA-MCF caused by AlHV-1 has not been reported in New Zealand.

Wildebeest-associated MCF is not a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

15.1.4 Zoonotic potential

Wildebeest-associated MCF is not a zoonotic disease.

15.1.5 Epidemiology

Host range

The natural hosts of AlHV-1 are wildebeest, but AlHV-1 can cause disease in cattle and other captive wild ruminants in zoological collections (Horner & Tham, 2003).

Herpesviruses antigenically similar to AlHV-1 have been isolated from hartebeest and topi (Hamblin & Hedger, 1984).

A total of 2,722 sera collected between 1963 and 1983, from 43 different species of wildlife ruminants in 11 African countries were examined for neutralising antibodies against WA-MCF. The results suggested that the high neutralising antibody titres from waterbuck and reedbuck were indicative of WA-MCF infection (Hamblin & Hedger, 1984).

Malignant catarrhal fever has been reported in giraffes mostly caused by *Ovine gammaherpesvirus 2* (OvHV-2) and not AlHV-1 (Horner & Tham, 2003).

There is no published evidence of WA-MCF in species of the Tragulidae or Giraffidae families. In the serosurvey by Hamblin and Hedger (1984), 41 giraffes were tested. None were positive.

Captive wild ruminants

There are various reports of WA-MCF in zoological collections.

In 1964, an outbreak of MCF occurred at Munich Zoo and killed all gaur and banteng (Bovidae). In the years following this event, sporadic outbreaks occurred in, elk, red deer, Père David's deer (Cervidae), European and American bison and again in gaur and banteng (Bovidae). It was suggested that WA-MCF was the cause of the outbreaks that occurred until 1979, with subspecies Caprinae and Alcelaphinae being the potential source of AlHV-1 (Hänichen et al., 1998).

In 1984, a suspected MCF outbreak occurred in captive wild ruminants at the Los Angeles zoo. Wildebeest were held at the zoo during this period. Within 2 months, there were deaths of Chinese water deer (Cervidae), a gerenuk and a red-flanked duiker (Bovidae). The clinical, histological and serologic data supported a diagnosis of MCF due to AlHV-1.

In 2004, 3 Ankole cattle died of WA-MCF at a zoological park in the UK. The cattle were grazed with a herd of wildebeest for part of the year and separated during winter. This outbreak only affected the Ankole cattle, although other susceptible ruminants (Cape buffalo, Père David's deer and others) were held in close proximity to the wildebeest. The wildebeest were known to be positive for AlHV-1; however, no previous outbreaks had occurred in the years prior to this, even though susceptible species were grazed with or in close proximity to the wildebeest (Whitaker et al., 2007).

Geographical distribution

The WA-MCF does not occur outside Africa except where wildebeest are held in zoos or facilities with other susceptible Bovidae (Horner, 1996).

It occurs in the cattle-raising regions of eastern and southern Africa where wildebeest and cattle are grazed together (OIE Terrestrial Manual, 2018t).

Spread of the disease is primarily dependent on the presence of the wildlife reservoir, i.e. wildebeest, hence, the distribution of WA-MCF is largely restricted to Kenya, Tanzania and the RSA, where wildebeest are present (Wambua et al., 2016).

Pathogenesis

Malignant catarrhal fever, also referred to as African malignant catarrhal fever, bovine malignant catarrhal fever, or *snotsiekte,* is a collective term for the clinicopathological signs manifested by cattle and other susceptible ungulates when infected with viruses of the genus *Macavirus* of the subfamily *Gammaherpesvirinae* (Wambua et al., 2016).

The pathogenesis of MCF remains to be fully elucidated. Authors have suggested various immunopathological mechanisms to explain the clinical manifestation. The pathological findings could consist of the following components: T-lymphocyte hyperplasia in lymphoid organs and accumulation of these cells in non-lymphoid tissue, epithelial degeneration, necrosis and hyperkeratosis, and vasculitis (Reid & Van Vuuren, 2004).

The incubation period is reported as ranging from 11 to 34 days but also up to 9 months (Horner & Tham, 2003; OIE Technical Disease Cards, 2013c; Whitaker et al., 2007).

Wildebeest calves are infected within the first few months of birth (Barnard et al., 1989). Wildebeest calves shed virus in nasal and ocular secretions mainly in the cell-free form (Mushi et al., 1980), implying that the virus is quite stable and has a longer survival rate in the environment (Whitaker et al., 2007).

The virus is excreted only by the natural hosts such as wildebeest, and therefore, other animals with clinical disease are not a source of infection (Horner & Tham, 2003) and may be regarded as dead-end hosts.

Infected wildebeest remain carriers for life (OIE Technical Disease Cards, 2013c; OIE Terrestrial Manual, 2018t).

Similar to other herpesviruses, latency is a feature of AlHV-1. The virus may become reactivated in adult wildebeest during periods of immunosuppression such as pregnancy or by stressful conditions such as captivity or starvation. They are then likely to become infectious to other susceptible species (Barnard et al., 1989; Wambua et al., 2016).

Varying disease latency periods have also been described in domestic ruminants (OIE Technical Disease Cards, 2013c).

Clinical signs

Alcelaphine gammaherpesvirus 1 causes inapparent infections in wildebeest (Horner & Tham, 2003).

The clinical signs of MCF in ruminants other than natural hosts range from peracute to chronic. In the peracute form, either no signs are observed, or depression, diarrhoea and dysentery occur within 12 to 24 hours before death.

Other signs include fever, erosions of the tongue, hard palate and gums, increased serous lachrymation and nasal exudate, which progresses to profuse mucopurulent discharges. Characteristic lesions include progressive bilateral corneal opacity, starting at the limbus (Horner & Tham, 2003; OIE Technical Disease Cards, 2013c).

Deaths due to WA-MCF have been reported in various captive wild ruminants from outbreaks in zoos. These include Chinese water deer, a gerenuk, a red-flanked duiker, European and American bison, elk, red deer, Père David's deer, Indian gaur and Javan banteng (Hänichen et al., 1998; Meteyer et al., 1989). However, during some of these outbreaks, other captive wild ruminants tested serologically positive for AlHV-1 in the absence of clinical disease (Meteyer et al., 1989).

Infection appears to persist in most groups of wildebeest held in zoological collections. Infection may be absent in animals that have been isolated during calf-hood or that live in small groups. However, this is difficult to verify due to the unreliability of the diagnostic tests (OIE Terrestrial Manual, 2018t).

Transmission

Transmission within free-ranging wildebeest populations occurs perinatally. The viral DNA of AlHV-1 has been detected in wildebeest placenta. In a study by Lankester et al. (2015), over 50% of 94 samples tested positive for AlHV-1 viral DNA (Lankester et al., 2015).

Wildebeest calves are infected within the first few months of birth, by in utero, direct contact or aerosol routes. Fomites may also contribute to transmission. Transmission by wildebeest calves occurs at 1 to 2 months of age. Transmission after 6 months of age is rare (Barnard et al., 1989).

The distance of WA-MCF aerosol transmission has not been studied in great detail. There is, however, a study on the distance of OvHV-2 transmission. *Ovine gammaherpes virus 2* is closely related to AlHV-1. In this particular case study, lambs from a feedlot infected with OvHV-2 were the source of transmission of virus to bison herds at a distance of up to 5 kilometres. The transmission over this distance was related to the amount of virus in the air. This was a function of the number of lambs in the feedlot and the age of the lambs. Lambs are highly infectious and shed large volumes of virus in comparison to adult sheep. This is similar to the shedding characteristics of wildebeest calves (Li et al., 2008).

The WA-MCF is transmitted to cattle or other susceptible ruminants by wildebeest and possibly hartebeest (Horner, 1996; Ramsay et al., 1982).

There is no unequivocal evidence that AlHV-1 can be horizontally transmitted by other species. Susceptible species are most commonly infected after exposure to parturient wildebeest, young calves or pasture contaminated by them (OIE Technical Disease Cards, 2013c).

Close contact is the most common route of transmission. Other routes of transmission include water sources, feeders, caretakers and birds (Okeson et al., 2007).

In newborn cattle, disease may occur after congenital/vertical transmission (OIE Technical Disease Cards, 2013c; Plowright et al., 1972). There is no documented evidence of horizontal transmission between cattle, and thus, cattle are regarded as terminal/dead-end hosts.

In the outbreak at the Los Angeles zoo, all animals were housed separately. However, the wildebeest were housed at the top of the hill and the other species further down the hill along a road. The drainage system ran from the wildebeest pens downhill, passing the affected species. Suspected

routes of transmission were drainage, common feeding, service shed and road access (Meteyer et al., 1989).

This scenario and others mentioned earlier imply that segregation may not necessarily prevent the spread of WA-MCF in a zoological setting.

Diagnosis

Diagnostic tests that may be used as an AlHV-1 agent identification are virus isolation, and virus neutralisation to detect an immune response. It must be noted that these tests may be used in some situations. However, there are factors such as cost and reliability that severely limit their applications (OIE Terrestrial Manual, 2018t).

Infected wildebeest consistently develop antibodies to AlHV-1, which can be detected by various assays including virus neutralisation, immunoblotting, ELISA, immunofluorescence and immunocytochemistry.

In latently infected adult wildebeest, the very low circulating viral load may reduce the reliability of PCR tests (OIE Terrestrial Manual, 2018t).

The antibody response of clinically affected animals (other than wildebeest) is limited, as no neutralising antibodies develop. Detection therefore relies on the use of immunofluorescence or immunoblotting (Horner & Tham, 2003).

The case study on the MCF outbreak at the Los Angeles zoo discussed further evidence of the testing and necropsy procedures conducted at the zoo prior to importing wildebeest and after deaths, respectively. There were no cases of MCF for 5 years after the initial introduction of wildebeest from Africa. However, the MCF outbreak occurred almost a year after the second introduction of a male wildebeest from a privately owned wild animal park. All wildebeest tested negative for MCF prior to introduction. This demonstrates that diagnostic testing of wildebeest prior to introduction may not be reliable. As a result, the Los Angeles zoo removed all wildebeest and experienced no further MCF cases (Meteyer et al., 1989).

Treatment, control and prevention

Control relies on the segregation of natural hosts (wildebeest and potentially hartebeest) from susceptible species and the elimination of indirect transmission pathways.

Alcelaphine gammaherpesvirus 1 infected animals other than natural hosts rarely transmit infection, implying that natural hosts are the primary source of infection. Wildebeest appear to transmit infection efficiently to most other ruminant species. Their segregation in zoos is therefore of paramount importance (Horner & Tham, 2003).

There is no licensed vaccine for MCF.

Semen

There is no published evidence demonstrating the presence of AlHV-1 in semen of domestic or wildlife ruminants.

There is, however, a study showing evidence of OvHV-2 DNA in ram semen as well as various tissues of the male urogenital tract (Hüssy et al., 2002). *Ovine gammaherpes virus 2* is closely related to AlHV-1. There is uncertainty surrounding the role contaminated semen may play in transmission; this is suggested by the authors to be insignificant. The study does not indicate whether OvHV-2 was viable and present in semen at a dose capable of infecting naïve recipient ewes. Domestic ruminants or Caprinae species are not covered in the scope of this IRA.

There is, therefore, no circumstantial evidence to conclude that AlHV-1 could contaminate the semen of species covered in this IRA.

15.1.6 Hazard identification conclusion

Wildebeest-associated MCF and other viruses within the MCF group are not OIE-listed diseases. However, WA-MCF is part of the “Non OIE-listed diseases affecting wild animals”. The WA-MCF

causes inapparent infections in wildebeest but can cause disease in cattle and other captive wild ruminants in zoological collections.

There is no evidence demonstrating the presence of AlHV-1 in semen of wildlife ruminants.

Alcelaphine gammaherpesvirus 1 is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

There is no published evidence of WA-MCF in species of the Tragulidae or Giraffidae families.

Alcelaphine gammaherpesvirus 1 is not identified as a hazard in captive wild Giraffidae and Tragulidae and will not be assessed further.

Alcelaphine gammaherpesvirus 1 is identified as a hazard in captive wild Bovidae.

15.2 Risk assessment

15.2.1 Entry assessment

Wildebeest-associated MCF does not occur outside Africa except where wildebeest are held in zoological collections with other susceptible species.

There is likely to be a very small number of live captive wild Bovidae imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported bovid will be infected is assessed as very low.

Cases of WA-MCF in susceptible captive wild ruminants have been reported in European and American zoos. The source of these outbreaks were captive wildebeest in these facilities.

Infection appears to persist in most groups of wildebeest held in zoological collections. Infection may be absent in animals that have been isolated during calf-hood or that live in small groups. However, this is difficult to verify due to the unreliability of diagnostic tests.

Wildebeest calves are infected within the first few months of birth. Infected wildebeest remain carriers for life. Latency has been described for WA-MCF. The virus may become reactivated in adult wildebeest during periods of immunosuppression. They are then likely to become infectious to other susceptible species.

Incubation periods can be as long as 9 months.

Other species that could be infected with WA-MCF were waterbuck and reedbuck as they showed high neutralising antibody titres in a serosurvey. However, these animals did not show clinical signs of disease.

Natural hosts (wildebeest) are the only animals that have demonstrated the ability to transmit AlHV-1 to susceptible species. Therefore, all animals except the natural hosts are dead-end hosts.

It is plausible that wildebeest with extended carrier states and inapparent infections, not exhibiting signs of WA-MCF, could be passed as clinically sound for export.

As a result, the likelihood of entry of AlHV-1 in animals of the genus *Connochaetes* spp. is assessed as high.

The likelihood of entry of AlHV-1 from species other than the genus *Connochaetes* spp. of the Bovidae family is assessed as very low.

15.2.2 Exposure assessment

Horizontal transmission has only been described in wildebeest. There is no evidence of transmission from other susceptible wildlife ruminants.

Captive wild ruminants in zoological collections that have been exposed to AlHV-1, became infected with resultant mortality. A handful of captive wild ruminants have shown seropositivity (in the absence of clinical signs) to WA-MCF after reported outbreaks of disease in other ruminants at the zoo. Transmission from these species were not reported.

Routes of transmission from infected wildebeest to other wildebeest and susceptible ruminants include in utero, direct contact, aerosol routes, water sources, feeders, caretakers and birds.

If infected wildebeest are imported into zoological collections, breeding is likely to occur. Most outbreaks within zoological collections occur within a few months of wildebeest calving. This is due to calves shedding high viral loads in nasal and ocular secretions and the presence of virus in parturient material and pastures contaminated with it.

Segregation of animals is likely to prevent direct transmission to susceptible species; however, various indirect routes and transmission pathways may still result in exposure of susceptible species within zoos.

Once infected, wildebeest remain carriers for life and will be a source of WA-MCF to susceptible species for as long as they are present in the zoo. Captive wild and domestic ruminants in zoos are dead-end hosts, and infection is likely to result in death after exposure. Wildebeest-associated MCF cannot establish in animal populations other than the natural hosts (wildebeest).

Aerosol transmission has been reported. Details as to the distance of WA-MCF aerosol transmission is scant. An extrapolation could be made from scenarios of a related herpesvirus (OvHV-2) in sheep. However, in the case described there were large numbers of lambs shedding virus.

Therefore, the likelihood of WA-MCF being transmitted via aerosols to susceptible animals such as cattle and deer outside the zoo is very low.

If transmission to susceptible ruminants outside the zoo does occur, infection is likely to result in the deaths of a few animals and is not likely to lead to further establishment or spread of the disease.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

Therefore, the likelihood of AlHV-1 exposure and establishment within the zoo is assessed as low, the likelihood of exposure and establishment outside the zoo is assessed as negligible.

15.2.3 Consequence assessment

Wildebeest associated malignant catarrhal fever is not an OIE-listed disease. However, WA-MCF is part of the “Non OIE-listed diseases affecting wild animals”. This is a disease that has not met the OIE’s criteria to be listed. However, OIE experts of the working group on wildlife diseases have selected it for monitoring, both because of its importance for wild animals and also for early warning purposes, in order to protect human and livestock health.

As mentioned, there is likely to be a very small number of live wildebeest imported. The direct consequences would be due to the importation of AlHV-1 infected wildebeest. These animals will be a source of infection for other susceptible captive wild and domestic ruminants at the zoo. Wildebeest experience inapparent infections and are not adversely affected. However, other susceptible species may experience clinical disease, which, in the majority of cases, would result in mortality. There is much variability regarding which ruminant species could be affected. From the case studies described, some captive wild ruminants experienced clinical disease with resultant mortality, while others merely showed serological evidence of exposure.

The likelihood of WA-MCF being transmitted via aerosols to susceptible animals such as cattle and deer outside the zoo is very low. If this does occur, these animals are likely to experience clinical disease with resultant mortality, and this is likely to not lead to further spread and establishment of the disease, as the animals are regarded as dead-end hosts. The WA-MCF is therefore unlikely to establish in domestic or wildlife populations other than the natural hosts (wildebeest).

Wildebeest-associated MCF is not a zoonotic disease, and therefore, the consequences for human health are negligible.

Indirect consequences would entail costs for control and surveillance within the affected zoos. Should the disease spread further than expected, loss of valuable species within zoos may also occur. This would negatively impact their ASMP.

If WA-MCF spreads outside the zoo to cattle and deer, there would be financial loss related to mortality of these animals only, as there is unlikely to be further establishment and spread.

An outbreak of WA-MCF would not affect trade.

Therefore, the overall consequences as a result of a WA-MCF incursion are assessed as very low.

15.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible for species of the Bovidae family covered in this IRA, the risk estimate for AlHV-1 is non-negligible, and it is assessed to be a risk in the captive wild Bovidae.

Therefore, risk management measures can be justified.

15.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Malignant catarrhal fever is not an OIE-listed disease. However, WA-MCF is part of the “Non OIE-listed diseases affecting wild animals”.
- Wildebeest-associated MCF caused by AlHV-1 has not been reported in New Zealand and is not a notifiable disease.
- Wildebeest-associated MCF is not a zoonotic disease.
- Natural hosts of WA-MCF are wildebeest.
- Other domestic and wildlife ruminants that can be infected include cattle and various antelope species.
- There are no reports of WA-MCF in Tragulidae or Giraffidae.
- Wildebeest-associated MCF does not occur outside Africa except where wildebeest are held in zoos with other susceptible Bovidae.
- The incubation period is variable ranging from 11 to 34 days or can be up to 9 months.
- Infected wildebeest remain carriers for life.
- Latency and persistent infections have been reported in wildebeest and cattle.
- Wildebeest infected with AlHV-1 show inapparent infections.
- Transmission routes include in utero, direct contact or aerosol routes, water sources, feeders, caretakers and birds.
- Diagnostic tests include virus isolation, virus neutralisation, serology and PCR. Cost and reliability are limiting factors of these tests.
- There is no literature evidence of AlHV-1 in semen of domestic or wildlife ruminants.

15.3.1 Options

One or a combination of the following options may be used:

Alcelaphine gammaherpesvirus 1 is not identified as a hazard in semen, species within the Giraffidae and Tragulidae families, and species within the Bovidae family (with the exception of the genus *Connochaetes),* and therefore, risk management measures are not warranted for these species.

Option 1

Animals:

1. Animals of the genus *Connochaetes* spp. may not be imported.

Animals of the genus *Connochaetes* spp. are at high risk of being carriers of AlHV-1, and testing is unreliable.

Option 2

Animals:

1. There have been no reported cases of confirmed or suspect WA-MCF at the facility of origin in the last 5 years; AND
2. the animal(s) were resident at the facility of origin for at least 12 months; AND
3. there have been no new introductions of *Connochaetes* spp. in the previous 12 months.

Incubation periods can be up to 9 months, and wildebeest are subclinically infected. A 5 year period is sufficient to allow for new introductions of wildebeest to mate and give birth and potentially transmit disease to susceptible captive wild ruminants.

16 Nairobi sheep disease

16.1 Technical review

16.1.1 Aetiological agent

Family: *Nairoviridae*
Genus: *Orthonairovirus*
Species: *Nairobi sheep disease orthonairovirus* (NSDV) (ICTV, 2019)

Nairobi sheep disease (NSDV) is the causative agent of Nairobi sheep disease (NSD).

The Asian variant, antigenically related to NSDV, is called *Ganjam virus* and has been identified in India and Sri Lanka (Lasecka & Baron, 2014).

16.1.2 OIE list

Nairobi sheep disease is an OIE-listed disease of sheep and goats.

16.1.3 New Zealand status

New Zealand is free from NSD (WAHIS, 2019d), as reported to the OIE.

Nairobi sheep disease is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

16.1.4 Zoonotic potential

The *Ganjam virus* variant has been isolated in humans in South Asia (Gong et al., 2015). Infections in humans can cause mild febrile illness, headache, nausea and vomiting (Yadav et al., 2011).

Laboratory infections from needle stick have been associated with fever and joint pain (Yadav et al., 2011; Zeller & Bouloy, 2000)

16.1.5 Epidemiology

Host range

Sheep and goats are the primary hosts affected by NSD. Goats are less susceptible to NSDV than sheep (CABI, 2017).

Cattle, other domestic animals and wildlife ruminants are refractory to infection with NSDV (Zeller & Bouloy, 2000).

However, there is a single report of NSD in 2 blue duikers at a Ugandan zoo in 1957 (CABI, 2017).

In a serological survey in Kenya, Davies (1978b) demonstrated low-level antibodies of NSDV in wild wildebeest, hartebeest, gazelle, impala and giraffe. However, it was suggested that these findings may likely be due to cross reactions to related viruses.

In a further study by Davies (1978a), no NSDV could be isolated from numerous wild ruminant species, even though some shared the habitat with the NSD tick vector (*R. appendiculatus*).

The article by Zeller and Bouloy (2000), states that there has been no serological evidence or clinical cases in wildlife ruminants, which is contradictory to the above reports.

Captive wild ruminants

There is a single report of isolation of NSDV from the previously mentioned 2 blue duikers at the Ugandan zoo in 1957 (CABI, 2017). There are no other reports of NSD in wildlife ruminants.

Geographical distribution

Nairobi sheep disease was first reported in Nairobi in 1910. The disease is now enzootic in East and Central Africa (Lasecka & Baron, 2014).

The *Ganjam virus* variant has been reported in South Asia (India and Sri Lanka) (Gong et al., 2015; Lasecka & Baron, 2014).

A new strain of NSDV has also been isolated in ixodid ticks in China by viral metagenomic analysis. However, NSD in animals has not been reported (Gong et al., 2015).

The presence of the virus is unknown in European countries.

Pathogenesis

Little is known about the pathogenesis of the virus (Lasecka & Baron, 2014).

The incubation period in small domestic ruminants is 1 to 5 days, during which animals develop fever (Lasecka & Baron, 2014; Mugera & Chema, 1967). Leukopenia and viraemia coincides with the febrile reaction (Mugera & Chema, 1967).

Observations in an experimental study of sheep and goats revealed that the virus also caused glomerular-tubular nephritis in the kidneys, hyaline degeneration and fragmentation of the myocardial fibres and coagulative necrosis of the gallbladder, in addition to haemorrhage in various organs (Mugera & Chema, 1967).

All age groups are susceptible to infection (CABI, 2017).

Sheep and goats are the only known mammalian reservoir of NSDV (Lasecka & Baron, 2014).

Clinical signs

Nairobi sheep disease has limited effect on animals from enzootic areas but can be clinically significant in animals moved from NSD-free areas to enzootic areas. Mortality is high in these cases (Lasecka & Baron, 2014).

Clinical manifestation is dependent on the susceptibility of the host and strain of virus (Lasecka & Baron, 2014).

Disease manifests as an acute haemorrhagic gastroenteritis in sheep and goats (Yadav et al., 2011).

The mortality rate in domestic small ruminants ranges from 40% to 90% (Davies, 1997; OIE Terrestrial Manual, 2018j).

In peracute cases, mortality can occur within 12 hours of pyrexia. Thereafter, mortality occurs 3 to 7 days after the febrile reaction (OIE Terrestrial Manual, 2018j).

Clinical signs in domestic small ruminants include pyrexia, hypersalivation and anorexia. Animals are reluctant to move and stand with lowered heads. They develop conjunctivitis, serosanguineous nasal discharge and lymphadenitis of the superficial lymph nodes (OIE Terrestrial Manual, 2018j).

A diarrhoea develops within 36 to 56 hours of the onset of pyrexia. The diarrhoea is initially profuse, watery and fetid. It progresses to a haemorrhagic and mucoid diarrhoea with abdominal pain and tenesmus (OIE Terrestrial Manual, 2018j).

Abortion often occurs.

Ganjam virus causes an NSD-like syndrome in sheep (CABI, 2017), which is less pathogenic than NSDV (Yadav et al., 2011).

Transmission

Nairobi sheep disease does not appear to be contagious (Lasecka & Baron, 2014).

The main transmission route is tick vectors. In Africa, NSDV is primarily transmitted by *Rhipicephalus appendiculatus*.

Other ticks suggested as competent vectors include the *Rhipicephalus* genus and *Amblyomma variegatum* (OIE Terrestrial Manual, 2018j).

In South Asia (India and Sri Lanka), *Ganjam virus* has been isolated in *Haemaphysalis intermedia*, *H. wellingtoni* and *R. haemaphysaloides.*

In China, a novel NSDV strain is likely transmitted by *H. longicornis* (Gong et al., 2015).

Diagnosis

Post-mortem findings in animals are not pathognomonic, and therefore, a presumptive diagnosis cannot be based on this due to other differential diagnoses.

Virus isolation in animals or cell culture may be performed on field samples.

The agar gel diffusion test can be used to detect antigens in tissues.

The indirect fluorescent antibody test has been used but may report cross-reactions with viruses of the same group.

An ELISA using a tissue culture antigen may be used for serological surveys.

An NSD-specific real-time RT-PCR has been used with a higher sensitivity than VI. However, validation has not been completed (OIE Terrestrial Manual, 2018j).

Treatment, control and prevention

There is no specific treatment for NSD.

Epizootics of NSD occur due to introduction of susceptible animals into a NSD enzootic area, introduction of infected ticks into a receptive area or ecological changes that result in extension of the tick vector distribution (Zeller & Bouloy, 2000).

Vaccines have been developed for NSD. However, they are not widely used due to lack of demand and validation (OIE Terrestrial Manual, 2018j).

Outbreaks of disease do not occur in enzootically stable areas. Therefore, preventing the entry of new naïve animals into these areas and limiting the distribution of the tick vectors can help prevent the disease.

Semen

There is no published evidence demonstrating the presence of NSDV in semen of domestic or wildlife ruminants.

16.1.6 Hazard identification conclusion

Nairobi sheep disease is an OIE-listed disease affecting sheep and goats, with only a single report of NSD in 1 species within the scope of this IRA.

There is no evidence of NSDV in semen of domestic and wildlife ruminant species.

Nairobi sheep disease orthonairovirus is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Nairobi sheep disease orthonairovirus is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

16.2 Risk assessment

16.2.1 Entry assessment

Nairobi sheep disease has been reported in East and Central Africa, the *Ganjam virus* variant has been reported in South Asia, and a new strain of NSDV has been isolated in ixodid ticks in China.

According to WAHIS, NSDV (including the *Ganjam virus* variant) has not been reported in Australia, Canada, Japan, Singapore, the RSA, the UAE or the USA. However, its presence in European countries is uncertain.

Therefore, the likelihood of entry of NSDV via captive wild ruminants (within the scope of this IRA) from the approved countries (with the exception of Europe) is assessed as negligible.

The evidence of NSDV in wildlife ruminants within the scope of this IRA is contradictory and insufficient to conclude that NSDV is a risk in wildlife ruminants.

There is a single report from a personal communication from 1957 of clinical disease in 2 captive blue duikers in Uganda. Further publications cite this communication, stating there has been natural infection and mortality of NSD in wild and captive wild duikers.

Davies (1978b) reported low-level antibodies of NSDV in various wild ruminants but concluded these findings may be due to cross reactions with related viruses and not a true reflection of evidence of NSDV.

Davies (1978a) also attempted isolation of NSDV from a number of wildlife ruminants including 2 duikers but was unsuccessful.

The article by Zeller and Bouloy (2000) stated that wildlife ruminants are refractory to infection with NSDV and that there has been no evidence of antibodies in these species.

There are no further articles on NSDV in wildlife ruminants.

For the above reasons it is concluded that wildlife ruminants do not play a significant role in the epidemiology of NSD.

Therefore, the likelihood of entry of NSDV via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) is assessed as negligible.

16.2.2 Risk estimation

Since entry is assessed as negligible, the risk estimate for NSD is negligible, and it is not a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures are not warranted.

17 Peste des petits ruminants

17.1 Technical review

17.1.1 Aetiological agent

Family: *Paramyxoviridae*
Genus: *Morbillivirus*
Species: *Peste des petits ruminants virus* (PPRV) (Parida et al., 2015)

Peste des petits ruminants virus (PPRV) is the causative agent of peste des petits ruminants (PPR).

17.1.2 OIE list

Peste des petits ruminants is an OIE-listed disease of sheep and goats (OIE, 2020d).

17.1.3 New Zealand status

New Zealand is regarded as PPR-free (OIE, 2020c).

Peste des petits ruminants is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

17.1.4 Zoonotic potential

Peste des petits ruminants is not a zoonotic disease (OIE Terrestrial Manual, 2019b).

17.1.5 Epidemiology

Host range

Peste des petits ruminants primarily affects sheep and goats. Cattle and pigs are susceptible to infection. However, they are dead-end hosts (Banyard et al., 2010).

The existence of sylvatic reservoirs has been suggested as a result of PPR in various captive wild ruminants. However, some authors contradict this statement.

Species reported to be susceptible to infection are gazelle, bushbuck, impala, springbuck, ibex, gemsbok and nilgai (Bovidae) (Elzein et al., 2004; Furley et al., 1987; Kinne et al., 2010; Li et al., 2018).

Peste des petits ruminants virus DNA has been detected in nasal swabs of subclinically infected defassa waterbuck, bubal hartebeest, buffalo and kob (Couacy-Hymann et al., 2005).

Antibodies to PPR were demonstrated in grey duikers (Ogunsanmi et al., 2010), waterbuck and buffalo (Couacy-Hymann et al., 2005).

Peste des petits ruminants has also been reported in wild small ruminants, such as Tibetan antelope and bharal in Tibet, as well as wild ibex in China (Li et al., 2018). These species together with buffalo, sheep and goats do not fall within the scope of this IRA.

A serological survey of wild grey duikers in a rain forest in Nigeria demonstrated that 4 (10.5%) out of 38 duikers were seropositive for antibodies to PPR (Ogunsanmi et al., 2010).

Wild ruminants from game parks in Côte-d'Ivoire were sampled. Eight species of wild Bovidae consisting of 260 animals were included in the survey. Of the 247 sera tested, only 2 (buffalo and waterbuck) showed antibodies to PPRV. This was a less than 1% prevalence. Nasal swabs were also taken from these animals and pooled for testing. Animals that were positive on RT-PCR for PPR DNA were defassa waterbuck, bubal hartebeest, buffalo and kob (Couacy-Hymann et al., 2005).

The death of 900 saiga antelope (Bovidae) in Mongolia was reported in 2017 (Anonymous, 2017). The cause of the deaths was later confirmed to be PPR. In the same incident, PPR was also reported for the first time in wild Siberian ibex and goitered gazelle (Bovidae) (The Saiga Resource Centre, 2018).

There is no literature evidence of PPR in species of the Tragulidae or Giraffidae family. However, another *Morbillivirus, Rinderpest virus,* has been reported in chevrotains in a zoological collection (Plowright, 1968) and in giraffes.

Captive wild ruminants

The UAE has reported outbreaks of PPR in captive wild ruminants (Elzein et al., 2004; Kinne et al., 2010).

An outbreak of PPR was reported in a zoological collection in 1983 in the Arabian Peninsula. A number of captive wild Bovidae species were affected, with mortality of 59 animals (Furley et al., 1987).

In 2002, an outbreak of PPR affected gazelles in a semi-free-range private farm in Saudi Arabia (Elzein et al., 2004).

Outbreaks of PPR were reported in 2 captive wild animal collections in the UAE in 2005/2006 and 2008/2009. The animals were kept under semi-free-range conditions. Various wild antelope and domestic ruminants were affected. The strain isolated in these outbreaks was different to strains previously identified in UAE. It was suggested that the likely source of the virus was through importation of domestic small ruminants from PPR-affected countries (Kinne et al., 2010).

An outbreak of PPR in a herd of 25 captive chousingha (Bovidae) was reported at a zoological park in India. Animals were maintained in captivity with no contact with domestic animals. The suggested sources of infection included fomites, fodder contaminated with excretions or secretions of infected animals or through the airborne route (Jaisree et al., 2018).

Geographical distribution

The distribution of PPR is widespread over west and central Africa, Arabia, the Middle East and southern Asia (Shaila et al., 1996).

In terms of the approved countries, Australia, Singapore, the USA, Canada, the RSA and the UK are officially PPR free (OIE, 2020c).

Peste des petits ruminants was initially detected in Turkey in 1996. Since then there have been numerous outbreaks of the disease spanning most of the country (Banyard et al., 2010). In 2018, PPR outbreaks were reported in Bulgaria in domestic small ruminants (BFSA (Bulgarian Food Safety Agency), 2018).

The UAE has reported outbreaks of PPR in domestic small ruminants and captive wild ruminants (Elzein et al., 2004; Kinne et al., 2010).

Pathogenesis

The pathogenesis of PPR has not been studied in detail. It is assumed that disease progression is similar to rinderpest. Infection likely occurs via the oropharynx with viral multiplication in draining lymph nodes. A viraemia results in dissemination of the virus to other lymph nodes and epithelia throughout the body. Here the virus multiplies, causing cytopathology resulting in lesions and disease (Rossiter, 2004).

The incubation period in domestic small ruminants is 2 to 6 days (Rossiter, 2004) but may range from 3 to 10 days (OIE Terrestrial Manual, 2019b). Incubation periods observed in gazelles were between 12 and 17 days (Furley et al., 1987).

In a study to investigate persistent infection in domestic small ruminants, carrier status was suggested. Goats that had recovered from PPRV infections and were healthy shed PPRV haemagglutinins in faeces for 12 weeks post recovery. However, the decrease in haemagglutinin antigens over time suggested that the animals' immune system eliminated the infection with time following recovery (Ezeibe et al., 2008).

In a second study, PPRV RNA could be detected in goat faeces at up to 16 weeks post recovery (Wasee Ullah et al., 2016).

Further studies are required to determine the role played by post-recovery carrier animals in the epidemiology of PPR.

In experimentally infected domestic small ruminants, diarrhoea was observed between 5 and 8 days post infection, with death within 10 days (Balamurugan et al., 2014). Animals that survive infection develop lifelong immunity (Anderson, 1995).

Peste des petits ruminants has the ability to circulate uncontrolled in wildlife ruminants and may act as a potential source of virus for domestic ruminants (Kinne et al., 2010).

Clinical signs

Clinical signs in domestic small ruminants begin with pyrexia followed by depression, inappetence, and serous ocular and nasal discharges that become progressively purulent. Hyperaemia of the mucosa of the oral cavity is followed by necrosis and sloughing of the epithelium, leaving erosions. A watery blood-stained diarrhoea (OIE Terrestrial Manual, 2019b) begins 2 to 3 days after the onset of pyrexia, and mortality may occur 3 to 7 days later (Rossiter, 2004).

In domestic small ruminants, mortality rates have been recorded to range from 20% to over 90% in epizootics (Opasina & Putt, 1985), with morbidity reaching 100% (OIE Terrestrial Manual, 2019b). The mortality rates in enzootic areas range from 4% to 5% (Opasina & Putt, 1985).

When naïve populations of domestic and wildlife ruminants are infected by virulent strains of PPR, mortality rates are very high (Anderson, 1995; Kinne et al., 2010). In milder forms of the disease, the clinical signs are less severe and morbidity and mortality rates are lower (OIE Terrestrial Manual, 2019b).

In enzootic areas, such as parts of Africa and India, subclinical infections of PPR in domestic and wild ruminants have been reported. Subclinical cross-infections are also known to occur between these animals (Couacy-Hymann et al., 2005).

Reported clinical signs in antelope species in the 2005/2006 and 2008/2009 PPR outbreaks in UAE were diarrhoea and high mortality rates. There was a 100% mortality rate in the second outbreak (Kinne et al., 2010).

In the outbreak of captive chousingha, animals showed signs of acute respiratory disease with frothy nasal discharge (1 to 2 days) and mortality of 20 animals within 48 hours (Jaisree et al., 2018).

In the outbreak at a zoological collection in the Arabian Peninsula, most captive wild ruminants (gazelles, ibexes and gemsboks) were found dead without showing any clinical signs, while nilgai experienced subclinical infections. Clinical signs that were observed included diarrhoea that was blood-stained, dark and putrid. All animals that developed clinical signs died (Furley et al., 1987).

In the outbreak of semi-free-range gazelles in the UAE, morbidity was 51% and the case fatality rate was 100%. Clinical signs described in the gazelles were similar to those experienced in infected domestic small ruminants (Elzein et al., 2004).

The studies of PPR in wildlife ruminants have shown that PPR may cause severe disease with high mortality in captive wild ruminants but that PPRV may circulate silently at low prevalence rates in wild ruminant populations. These studies suggest that PPR cannot be self-sustained in wild ruminant populations as some authors propose. Epizootics in wildlife ruminants usually occur as a result of neighbouring domestic small ruminants as a source of infection.

Transmission

Transmission is horizontal via direct and indirect contact.

Direct contact between infected and susceptible animals, and inhalation of aerosols are the most common routes of transmission (Rossiter, 2004).

Indirect transmission via contaminated bedding, feed and water troughs may occasionally occur (Rossiter, 2004).

The virus has been isolated from blood and faeces and oral, nasal, ocular and pharyngeal swabs during acute disease. The virus is temperature-sensitive, and outside the body, it is readily inactivated in dry environments (Rossiter, 2004).

The spread of PPR infections are affected by host density and birth rates (Anderson, 1995).

PPR has demonstrated interspecies transmission between domestic and wildlife ruminants that mingle together (Kinne et al., 2010).

The source of infection in the outbreak of 900 saiga antelope in Mongolia was due to spillover from livestock (The Saiga Resource Centre, 2018).

The hypothesised source of infection in the zoo outbreak in the Arabian Peninsula was the movement of a zookeeper between the government goat farm that was initially infected and the captive wild ruminant paddocks (Furley et al., 1987).

Diagnosis

There are various tests available for agent identification of PPRV. These include RT-PCR, real-time RT-PCR, virus isolation, immunocapture ELISA, penside test (lateral flow device), AGID and counterimmunoelectrophoresis. Of these tests, the OIE recommends immunocapture ELISA, RT-PCR and real-time RT-PCR for the confirmation of clinical cases and real-time RT-PCR for eradication.

Virus neutralisation is the recommended test to determine individual animal and population freedom from infection. Competitive ELISA may be used for multiple purposes but not for the confirmation of clinical cases, as it may not be reliable in this instance (OIE Terrestrial Manual, 2019b).

Treatment, control and prevention

There is no effective treatment for animals infected with PPRV. The administration of hyperimmune serum during the acute stages has been reported to assist with recovery (Rossiter, 2004).

Valuable animals may be treated with fluid replacement and appropriate nursing.

In countries that are affected with PPR, susceptible animals may be protected by vaccination. Domestic small ruminants vaccinated with an attenuated strain of PPR develop lifelong immunity (OIE Terrestrial Manual, 2019b). The 2 commonly used vaccine strains (Nigeria/75/1 and Sungri/96) have been shown to protect animals against PPRV isolates of all lineages (Hodgson et al., 2018).

In countries that are PPR-free, restricting the importation of infected domestic small ruminants from PPR-affected countries would serve as the main prevention strategy.

Semen

Various authors state that PPRV is present in all excretions and secretions (Rossiter, 2004). However, there are no studies demonstrating viral presence in semen. Domestic small ruminants do become viraemic and shed virus in faeces and oral, nasal and ocular secretions. Therefore, it is plausible that the virus could be shed in semen during the viraemic period.

The OIE considers germplasm of domestic small ruminants a risk commodity.

There is also no evidence in the literature of venereal transmission of PPRV.

There is no published evidence demonstrating the presence of PPRV in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with PPRV-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

17.1.6 Hazard identification conclusion

Peste des petits ruminants is an OIE-listed disease of sheep and goats. Peste des petits ruminants has been reported in a large number of wildlife ruminants.

The presence of PPRV has not been demonstrated in semen. However, the OIE considers semen a risk commodity for PPRV.

Peste des petits ruminants virus is identified as a hazard in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

17.2 Risk assessment

17.2.1 Entry assessment

Peste des petits ruminants virus has never occurred in Australia, Singapore, the USA, Canada, the UK, Japan or the RSA. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

There is evidence of PPR in wildlife ruminants both in the wild and in captive collections in Saudi Arabia and the UAE. From these outbreaks and surveillance studies of PPR in wildlife ruminants, it has been noted that infection in captive wild ruminants is likely to be severe with high mortality rates, whereas infection in wild ruminants may be subclinical and present at low levels. Some authors suggest certain species of wild ruminants may act as reservoir hosts. However, this has been contradicted by other authors who state that PPR cannot be sustained in wildlife without domestic small ruminants as a source of infection. The latter hypothesis seems more plausible given the suggested sources of infection in the wildlife ruminant PPR outbreak case studies.

Peste des petits ruminants has an incubation period of up to 17 days in wildlife ruminants. Some species may experience subclinical infections or be healthy carriers. It is plausible that subclinically infected animals, not exhibiting signs of PPR, could be passed as clinically sound for export or as donors for semen collection.

There is currently no published evidence to demonstrate the presence of PPRV in semen. Authors of various studies state that PPRV is found in all secretions and excretions, and the OIE considers germplasm to be a risk commodity.

Therefore, the likelihood of entry of PPRV via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from PPR-affected countries is assessed as low.

The likelihood of entry of PPRV via semen of captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from PPR-affected countries is assessed as very low.

17.2.2 Exposure assessment

Transmission routes include direct and indirect contact with infected animals, via aerosols and contaminated bedding, water or feed.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

Imported infected animals are likely to act as a source of infection for susceptible captive wild ruminants within zoos if they are in direct contact in the same enclosures. Susceptible animals may also become infected via fomites if the same zookeepers manage enclosures as described in previous captive collection outbreaks.

As suggested, PPR is likely to result in the death of infected captive wild ruminants, and therefore, establishment is very unlikely. However, if domestic small ruminants are kept at the zoo, PPRV could establish in these populations and become a continuous source of infection for other susceptible animals within the zoo.

Peste des petits ruminants virus can be transmitted via aerosols between animals in close contact. The survival time of the virus would be a function of distance and time outside the body. Since the virus is temperature-sensitive and cannot survive outside the body for long periods, it is unlikely to spread to susceptible wild or domestic small ruminant populations outside the zoo via this route.

However, if domestic small ruminants are released into New Zealand farms, PPRV could be transmitted to other domestic small ruminants, as well as cattle and pigs. Cattle and pigs are dead-end hosts, but the disease could establish in the small ruminant population in New Zealand.

As mentioned, the likelihood of entry of PPRV in semen is very low. However, if the semen is contaminated with PPRV, the likelihood of infection resulting from insemination is unknown due to a lack of research within this area. There have been no reports of venereal transmission of PPRV or infection of naïve dams after AI with contaminated semen in domestic or wildlife ruminants.

There are limited numbers of captive wild female ruminants in New Zealand zoos, thus the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore the likelihood of PPRV exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate, the likelihood of exposure and establishment outside the zoo is assessed as very low and likelihood of exposure and establishment via contaminated semen of captive wild Bovidae, Giraffidae and Tragulidae is assessed as very low.

17.2.3 Consequence assessment

Peste des petits ruminants is an OIE-listed disease of sheep and goats.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of PPR would be due to infected imported animals. These animals are likely to become ill and succumb to death. During this time, they may spread the disease to susceptible captive wild and domestic small ruminants within the zoo resulting in further loss of valuable captive wild species. If there are no domestic small ruminants within the zoo, PPR is likely to cause death of all infected captive wild ruminants without establishment in these populations. If domestic small ruminants are present, establishment in these populations may occur with a continuous source of infection for other animals.

The likelihood of PPRV exposure and establishment in domestic small and wild ruminants outside the zoo is assessed as very low and is only likely to happen if domestic ruminants are released from the zoo onto New Zealand farms. Peste des petits ruminants could then spread and establish in the New Zealand sheep and goat population. Morbidity and mortality in naïve herds tend to be higher than in enzootic areas. Mortality could range from 20% to over 90%, with morbidity reaching 100%.

Trade impacts as a result of a PPR incursion at the zoo are likely to be minimal if New Zealand can prove the outbreak has been contained in the zoo. However, this may be difficult to prove if domestic animals are released from the zoo.

Peste des petits ruminants is not a zoonotic disease, and therefore, the consequences for human health are negligible.

Indirect consequences would include the costs for testing and surveillance of susceptible captive wild ruminants. Animals with antibodies to PPRV are likely to be euthanased due to the probability of becoming reservoir hosts.

There would be additional costs to New Zealand for PPR control in the event of an incursion in domestic ruminants.

Therefore, the overall consequences as a result of a PPR incursion are assessed as low.

17.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for PPR is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and their semen.

Therefore, risk management measures can be justified.

17.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Peste des petits ruminants is an OIE-listed disease of sheep and goats.

- New Zealand is free from PPR, and PPR is a notifiable disease.
- Peste des petits ruminants is not a zoonotic disease.
- Peste des petits ruminants primarily affects sheep and goats, but wildlife ruminants are susceptible to infection.
- Peste des petits ruminants has never occurred in Australia, Singapore, the USA, Canada, the UK or the RSA.
- An incubation period of 12 to 17 days has been reported in wildlife ruminants.
- Transmission routes include direct contact between infected and susceptible animals, inhalation of aerosols, and fomites such as contaminated bedding, feed and water.
- Diagnosis includes RT-PCR, real-time RT-PCR, VI, immunocapture ELISA, penside test (lateral flow device), AGID, counterimmunoelectrophoresis, VN, and competitive ELISA.
- *Peste des petits ruminants virus* is present in all excretions and secretions.
- The OIE considers germplasm of ruminants a risk commodity.

17.3.1 Options

One or a combination of the following options may be used:

Option 1

1. Country freedom for PPR; AND
2. the animal(s)/donor male(s) was resident in PPR free countries since birth; AND
3. the animal(s)/donor male(s) showed no clinical signs of disease on the day of export or semen collection.

Option 2

Animals:

1. The animal(s) showed no clinical signs suggestive of PPRV infection for at least 21 days prior to export; AND
2. within 21 days of export, the animal(s) was tested for PPR with negative results; AND
3. the animal(s) was kept in pre-export isolation for at least 21 days.

The recommendations are similar to those of the OIE Code for the importation of wild ruminants from countries or zones infected with PPRV.

Semen:

1. The donor male(s) showed no clinical signs suggestive of PPRV infection for at least 21 days prior to collection of semen and during the following 21 days; AND
2. the donor male(s) were kept, for at least 21 days prior to collection, in an establishment where no case of PPR was reported during that period, which was not situated in a PPRV infected zone and to which no animals had been added during the 21 days prior to collection; AND
3. the donor male(s) was tested for PPR with negative results, at least 21 days prior to collection of semen; OR

These recommendations are similar to those of the OIE Code for the importation of semen of domestic sheep and goats from countries or zones infected with PPRV.

4. A straw of semen from each collection batch was tested for PPRV with an MPI-approved test and was negative.

18 Rabies

18.1 Technical review

18.1.1 Aetiological agent

Family: *Rhabdoviridae*
Genus: *Lyssavirus*
Species: *Rabies lyssavirus* (ICTV, 2019)

Rabies lyssavirus (RABV) is the causative agent of rabies.

18.1.2 OIE list

Rabies is an OIE-listed disease affecting multiple species (OIE, 2020d).

18.1.3 New Zealand status

New Zealand is free from rabies (WAHIS, 2019d), as reported to the OIE.

Rabies is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

18.1.4 Zoonotic potential

Rabies is a zoonotic disease (OIE Terrestrial Manual, 2018v). If treatment is not administered within 48 hours post exposure, fatality is almost definite.

18.1.5 Epidemiology

Host range

Rabies lyssavirus can infect and cause rabies in all mammals (OIE Terrestrial Manual, 2018v).

Of the species covered under this IRA, rabies has been reported in wild greater kudus (Scott et al., 2012), elands (Hübschle, 1988) and a captive-bred addax (David et al., 2007).

The first reported cases of rabies in greater kudus were in Namibia in the early 1970s (Hikufe et al., 2019).

Rabies epizootics have also been reported in deer (Scott et al., 2012); however, deer do not fall within the scope of this IRA.

Captive wild ruminants

Rabies in zoological collections are rare. However, a handful of cases have been reported in non-ruminant species (bats, rhinoceroses and bears) (David et al., 2007).

In a survey carried out on 32 zoos in the USA, 1 zoo reported an incident of rabies in their animal collection and 7 zoos reported rabies in wild animals caught on zoo grounds (Zelepsky & Harrison, 2010).

In 2004 at a zoo in Israel, an addax that was kept in an “African exhibit” with zebras, giraffes, rhinoceroses, ostriches and marabous began showing signs of distress. The animal became recumbent the next day and died. A brain sample from the addax tested positive for RABV. It was revealed that a month before the addax rabies case, a fox carcass was found in the zoo (David et al., 2007).

Geographical distribution

Rabies affects Africa, Asia, Europe, North America and South America (WAHIS, 2019c).

Rabies was last detected in Australia in 1867, in Singapore in 1953, in the UK in 1970 and the UAE in 1999 (WAHIS, 2019d).

Clinical rabies has been reported in the USA, Canada and the RSA in domestic and wild animals (WAHIS, 2019d).

Dog-mediated rabies has been eliminated from western Europe, Canada, the USA, Japan and some Latin American countries (WHO (World Health Organization), 2019).

Pathogenesis

Rabies is a highly fatal disease affecting all warm-blooded vertebrates and humans.

The OIE Code indicates an incubation period of 6 months in animals infected with RABV. The incubation period in kudus ranges from 12 to 250 days (Hassel et al., 2018; Hikufe et al., 2019).

The virus enters the site of infection, most commonly through the sensory nerve endings of the epithelial and subepithelial tissues of the skin or mucous membranes. The virus may also enter the nervous systems through cells of the eye, muscles and other tissues of the body. The virus then travels via the nerves toward the central nervous system. The virus is able to reach the brain, hidden from the body's immune system, where it eventually leads to encephalomyelitis with behavioural changes of the animal. By this time, the virus is also present in the saliva of the animal (Swanepoel, 2004). Behavioural changes most often result in altered behaviour: aggression, with subsequent biting/attacking of other animals or humans thus leading to propagation of rabies.

Sequestration of the virus and replication in non-neural tissue may result in protracted incubation periods (Swanepoel, 2004).

Wild and domestic carnivores are regarded as maintenance hosts (Scott et al., 2012). Most cases of rabies in other animals are usually a result of spillover from carnivores.

In countries like the RSA, the most important vectors and reservoirs for rabies are dogs and jackals. Sporadic rabies cases in greater kudus are the norm, as these animals come into contact with rabid carnivores. However, during 1978 and 1979, there was an alarming increase in the number of rabid greater kudus and cattle, thus pointing to an epizootic (Barnard & Hassel, 1981).

By 1985, there were an estimated 30,000 to 50,000 greater kudu deaths due to rabies. This was approximately 20% of the Namibian greater kudu population (Barnard & Hassel, 1981).

In 2011, greater kudu farmers reported losses of 30% to 68% due to rabies (Scott et al., 2012).

Epidemiological studies noted that the rise in greater kudu and cattle rabies cases paralleled that of rabies cases in jackals. However, the disparity was too large to conclude that the ruminant cases were solely due to spillover from carnivorous animals (Scott et al., 2012).

There are 2 differing theories on the cause of the 2 major rabies epizootics in greater kudus. One theory involves 2 point sources from rabid carnivores and the other a continuous cycle with transmission via horizontal means from rabid kudus (Scott et al., 2012). More recent molecular studies have demonstrated that the rabies isolates found in kudu are genetically separable from isolates found in jackals within the same area, suggesting the latter theory is more plausible.

Clinical signs

Common clinical signs in greater kudus included docility, excess salivation (Scott et al., 2012), losing their fear of humans and visiting domestic households (Hübschle, 1988).

In the experimental study by Barnard and Hassel (1981), only 5% of 80 greater kudus suspected of rabies showed aggressive behaviour. In the later stages, neurological signs indicative of paresis of the hindquarters, ataxia and incoordination were experienced.
Other signs observed included tail wagging, increased sexual activity, collisions with obstacles and walking in circles (Hübschle, 1988).

The disease in these animals is usually peracute to acute, as death ensues within 48 hours from the onset of observable signs.

Clinical signs noted in rabid deer were aggression, attacking other animals, what appeared to be mental hallucination, and hypersensitivity to noise. The deer would lick their companions and then attack them by biting their shoulders and tearing off hair and skin.

In early disease, animals experienced fits of excitement with bouts of syncope (Cope, 1888).

Sites of bites became pruritic and deer were found rubbing these parts on objects till they were raw. The signs ended in coma and paralysis with impending cardiac failure (Cope, 1888).

Transmission

Transmission in wildlife ruminants, as in most animals, is via saliva (Cope, 1888). *Rabies lyssavirus* is present in the saliva of infected animals and is transmitted through bite wounds (Scott et al., 2012).

Transmission results from superficial bites, licking of mucous membranes or shallow skin wounds and abrasions, and ingestion or inhalation of infected material (Swanepoel, 2004).

Experimental infection of laboratory animals via the intranasal route has been demonstrated (Swanepoel, 2004). Aerosol transmission has only been reported in rare occasions in laboratory animals and under field conditions in humans and animals in caves housing bats (Constantine, 1962).

In the majority of cases, spillover to ruminants results in termination of the transmission route. However, there have been cases where ruminants were able to transmit the virus horizontally and maintain an epidemiological cycle (Scott et al., 2012).

The horizontal transmission in greater kudus is dependent on environmental and behavioural factors. In the first epizootic, high rainfall led to an abundance of foliage and water with an exponential increase in the kudu population. The number of kudus continued to increase as a result of protection from predators by farmers for trophy hunting and tourism (Scott et al., 2012).

There are 2 hypotheses for the horizontal transmission between greater kudus.
The first is that animals browse on thorny acacia trees, which may cause oral lesions. Rabid kudus exhibit hypersalivation and may deposit copious amounts of saliva on these thorny branches. Since kudus are social browsers, susceptible kudus may browse on the same branches, thus coming into contact with contaminated saliva (Barnard & Hassel, 1981). An experimental trial by Barnard et al. (1982) demonstrated that contact between contaminated saliva and intact mucous membranes in kudus resulted in the infection and death of all kudus.

The other hypothesis is that of mutual grooming. Kudus lick themselves as well as the head, neck and shoulders of herd mates. This type of contact between susceptible and rabid kudus could result in the transmission of rabies (Mansfield et al., 2006).

Diagnosis

There are a number of OIE-recommended diagnostic tests for agent identification for various purposes.

To determine population freedom from infection, direct fluorescent antibody test (DFAT), direct rapid immunohistochemistry test (dRIT) for antigen detection, and conventional RT-PCR and real-time RT-PCR for RNA detection. Direct fluorescent antibody test, dRIT, VI, conventional RT-PCR and real-time RT-PCR can be used for eradication, confirmation of clinical cases and surveillance.

Virus neutralisation and ELISA tests are usually used to determine the immune status in individual animals or populations post-vaccination and for eradication. Virus neutralisation is the preferred choice for determining the animal's RABV immune status prior to movement (OIE Terrestrial Manual, 2018v).

Neurological signs may be indicative of rabies in enzootic areas. However, a definitive diagnosis is made after post-mortem examination and testing of brain tissue.

Treatment, control and prevention

In rabies enzootic countries, measures are put in place to control and limit the risk of infection in vulnerable populations such as wildlife, stray and domestic animals. Further measures include creating a barrier between the sources of infection and humans.

For these measures to be achieved, the World Organisation for Animal Health's (OIE's) strategy is mass dog vaccination campaigns, public awareness campaigns and the improvement of access to

human medical care (rabies vaccines, post-exposure prophylaxis and treatment with immunoglobulins).

A potential control measure for kudu rabies in Namibia would be the development of a safe, stable and suitable bait vaccine (Scott et al., 2012). Parenteral vaccines may also be used to protect endangered species.

For the valuable but limited number of animals in zoological collections, parenteral killed rabies vaccines may be warranted to protect at risk animals in rabies enzootic areas.

However, the bigger picture in control and prevention of rabies would be to target all vector and reservoir species.

In countries that hold a rabies-free status, prevention strategies would include limiting importation of animals, more especially rabies vectors or reservoir species, from countries that are affected by rabies. If importation is required, as in the case of domestic companion animals, rabies vaccination should be a requirement coupled with a rabies neutralisation antibody titre test to determine an appropriate level of protection.

Semen

There is no published evidence demonstrating the presence of RABV in semen of domestic or wildlife ruminants.

18.1.6 Hazard identification conclusion

Rabies is an OIE-listed disease affecting multiple species.

There is no evidence demonstrating the presence of RABV in semen of domestic or wildlife ruminants.

Rabies lyssavirus is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Rabies lyssavirus is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

18.2 Risk assessment

18.2.1 Entry assessment

Rabies lyssavirus was last detected in Australia in 1867, in Singapore in 1953, in the UK in 1970 and in the UAE in 1999. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Rabies can affect all mammals. Rabies has been reported in captive and wild Bovidae species within the scope of this IRA. Epizootics with suggested horizontal transmission have been reported only in wild Bovidae species. There have been limited cases of rabies in enzootic areas in captive collections with sources suggested to be rabid carnivores. Horizontal transmission has not been reported in these cases.

Although the OIE indicates an incubation period of 6 months, incubation periods in infected kudus can be as long as 250 days. If these animals are imported from rabies-affected countries and are incubating the disease, they would not show any clinical signs. It is plausible that subclinically infected animals, not exhibiting signs of rabies, could be passed as clinically sound for export.

Therefore, the likelihood of entry of *Rabies lyssavirus* via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from rabies-affected countries is assessed as low.

18.2.2 Exposure assessment

The transmission route of rabies is most often through bite wounds that are infected with contaminated saliva. Horizontal transmission in wild ruminants through licking mucous membranes or shallow skin wounds and abrasions, and ingestion or inhalation of infected material has been suggested in rabies epizootics in high-density kudu populations. This scenario is unlikely to be present in captive collections, as stocking density of animals in zoos in New Zealand is of importance for health and behavioural management.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If a captive wild ruminant that is incubating rabies is imported, the outcome once clinical disease manifests could be peracute to acute death, or the animal could become depressed for some time isolating itself from other animals, or the animal could become aggressive and attack or bite other animals in the enclosure.

If the animal shows aggressive clinical signs, exposure and infection of other animals is likely. These animals could also attack humans, such as zookeepers or vets, exposing them as well.

Establishment in the infected animals is unlikely because all animals that become infected and begin showing clinical signs will die. These animals will not be able to expose or infect carnivores that are the main reservoir hosts. Since New Zealand does not have a stray dog population or wild carnivores such as jackals or foxes that could enter zoos, rabies cannot spread further than the infected captive ruminants.

Therefore, the only animals or humans that will be exposed are those that come into direct contact with the infected animal or its saliva. Members of the public are unlikely to be exposed unless there are “close encounters” with these animals.

Rabies is unlikely to spread to animals or humans outside the zoo.

Therefore, the likelihood of *Rabies lyssavirus* exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate, the likelihood of exposure and establishment outside the zoo is assessed as negligible.

18.2.3 Consequence assessment

Rabies is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of rabies would be due to infected imported animals. These animals will succumb to clinical disease. Depending on the type of clinical signs experienced, these animals may spread the disease to susceptible captive wild animals in direct contact with them. The eventuality of rabies is death.

The zoos will lose valuable animals, which may negatively impact the ASMP programme.

Captive wild ruminants are not mixed with carnivore species. Therefore, the disease cannot spread to these species. As a result, rabies cannot establish in the zoo population.

Rabies is only transmitted via contact with infected and susceptible animals and in very rare cases via aerosol means in bat caves. It is very unlikely that rabies will spread to animals outside the zoo. Since an outbreak of rabies will be contained to captive wild animals only there will be no consequences for animals outside the zoo.

Rabies is a zoonotic disease. Infected animals may spread the disease to humans they come into contact with such as zookeepers or vets, through bites or exposure to saliva. If a suspicion or diagnosis of rabies is made rapidly, post-exposure prophylaxis can be administered to people that have been exposed to the rabid animal. However, if treatment is not provided in time and clinical signs

develop, death is likely to be the end result. There have been rare human survival cases; however, the majority of cases result in death.

In the absence of zoo “animal encounters” with infected captive wild ruminants, there would be no consequences for the public, as they would not be exposed to rabies.

Indirect consequences would include the costs for testing and surveillance of susceptible captive wild ruminants that were in direct contact with the rabid animal(s) within the zoo only, as well as the costs of post-exposure prophylaxis for exposed people.

Since rabies has never occurred in New Zealand, there could be negative trade impacts. However, these may be limited if New Zealand can prove that the disease was contained in the zoo without spreading to carnivores inside or outside the zoo.

Therefore, the overall consequences as a result of a rabies incursion are assessed as moderate.

18.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for rabies is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

18.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Rabies is an OIE-listed disease affecting multiple species.
- New Zealand is free from rabies.
- Rabies is a zoonotic disease.
- *Rabies lyssavirus* can infect and cause rabies in all mammals.
- New Zealand recognises Singapore and Australia as countries free from rabies.
- Wild and domestic carnivores are regarded as maintenance hosts.
- The incubation period in greater kudus ranges from 12 to 250 days.
- Transmission results from bites, licking mucous membranes or shallow skin wounds and abrasions, and ingestion or inhalation of infected material.
- Diagnostic tests for rabies include DFAT, dRIT, conventional RT-PCR, real-time RT-PCR, VN and ELISA.
- There is no evidence in the literature demonstrating the presence of RABV in semen of domestic or wildlife ruminants or the transmission of rabies to a naïve dam if inseminated with RABV-contaminated semen.

18.3.1 Options

One or a combination of the following options may be used:

Rabies lyssavirus is not identified as a hazard in semen, and therefore, risk management measures are not warranted.

Option 1

1. Country freedom for rabies; AND
2. the animal(s) were resident in rabies-free countries or zones since birth or for at least 6 months; AND
3. the animal(s) showed no clinical signs of disease on the day prior to or on the day of export.

The recommendations are similar to those of the OIE Code for the importation of domestic and captive wild mammals from countries or zones free from infection with the rabies virus.

Option 2

1. The animal(s) showed no clinical signs of disease on the day prior to or on the day of export; AND

2. the animal(s) were kept for the 6 months prior to shipment in an establishment where separation from susceptible animals was maintained and where there was no case of rabies for at least 12 months prior to shipment.

The recommendations are similar to those of the OIE Code for the importation of wildlife from countries infected with rabies.

19 Rift Valley fever

19.1 Technical review

19.1.1 Aetiological agent

Family: *Phenuiviridae*
Genus: *Phlebovirus*
Species: *Rift Valley fever phlebovirus* (ICTV, 2019)

Rift Valley fever phlebovirus (RVFV) is the causative agent of Rift Valley fever (RVF).

19.1.2 OIE list

Rift Valley fever is an OIE-listed disease affecting multiple species (OIE, 2020d).

19.1.3 New Zealand status

New Zealand is free from RVF (WAHIS, 2019d), as reported to the OIE.

Rift Valley fever is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

19.1.4 Zoonotic potential

Rift Valley fever is a zoonotic disease (OIE Terrestrial Manual, 2018w).

Rift Valley fever in humans is usually asymptomatic. Symptoms resemble those of an influenza-like illness, with fever and headaches. Occasionally there may be complications resulting in haemorrhagic syndromes, retinitis, encephalitis and death (Boushab et al., 2016).

19.1.5 Epidemiology

Host range

Rift Valley fever affects sheep, goats, cattle and camel (OIE Terrestrial Manual, 2018w).

Rift Valley fever phlebovirus seropositivity has been reported in wild springboks, wildebeest and impalas (Bovidae) in Namibia. Viral RNA has been isolated from springboks in the same study (Dondona et al., 2016).

Other wild ruminant species with reported antibodies to RVFV include, but are not limited to, kudus, gazelles, gerenuks, bushbucks (Bovidae) and giraffes (Giraffidae). The high seroprevalence suggests that some of these animals may be susceptible to infection (Evans et al., 2008).

Outbreaks of RVF and isolation of the virus have been reported in the RSA in wild buffalo and waterbuck (Bovidae) (Beechler et al., 2015; Grobbelaar et al., 2011).

Rift Valley fever has not been reported in Tragulidae species. However, due to the wide host range of RVFV, it is assumed that these species may be affected.

Captive wild ruminants

In January 1999, captive-reared buffalo near Kruger National Park (KNP) aborted after a spell of heavy rains. *Rift Valley fever phlebovirus* was isolated from the 6 aborted buffalo fetuses. During this period, RVFV was also isolated from a dead waterbuck from a nature reserve near KNP (Beechler et al., 2015). Buffalo are not included within the scope of this IRA.

There was a single report to the OIE in 2018 of RVF in gemsbok in a natural park in Chad. Of the 171 animals, 42 died of RVF. The source of infection was due to the legal introduction of new live animals (OIE Notification Report, 2019).

Springboks in Etosha National Park (Namibia) were positive for RVFV RNA (Dondona et al., 2016).

There is no published evidence of RVF in zoological collections.

Geographical distribution
Rift Valley fever is enzootic in many African countries, the Arabian Peninsula and some Indian Ocean islands (OIE Terrestrial Manual, 2018w).

Areas where RVF has never occurred include Australia, Singapore, the USA, Canada, Japan, the UK and Europe (WAHIS, 2019d).

Pathogenesis
Rift Valley fever is a peracute to acute mosquito-borne disease of domestic ruminants in Africa and Madagascar.

Rift Valley fever phlebovirus replicates at the initial site of infection and then spreads to critical organs (liver, spleen, brain). Damage to the organs is caused by lytic effects of the virus or immunopathological mechanisms (Swanepoel & Coetzer, 2004).

It is presumed that, similar to other arthropod-borne viral infections, the RVFV travels from the site of infection via the lymphatic system to regional lymph nodes. Here it replicates further and spills over into the circulation, producing the primary viraemia and systemic infection. There may be slight variations in the course of disease progression in different species (Swanepoel & Coetzer, 2004).

Viraemia in sheep, goat and cattle is observed from day 1 to 2 post infection up to day 7.

In experimental infections of buffalo, virus was detected in 4 out of 5 buffalos 2 days post infection. The detectable viraemia lasted for at least 48 hours. One buffalo did not develop viraemia or neutralising antibodies to RVF (Davies & Karstad, 1981). This demonstrates the difference in observations following RVFV infections in wild ruminants.

The study by Beechler et al. (2013) showed long-term subclinical circulation of RVFV in wild buffalo in the KNP and potentially other wildlife. A similar conclusion was reached by LaBeaud et al. (2011) and Evans et al. (2008) due to the RVFV seropositivity of numerous wild ruminants (LaBeaud et al., 2011).

A study of RVF in wild ruminants was carried out in Etosha National Park in Namibia. *Rift Valley fever phlebovirus* RNA was isolated from 18 springboks, demonstrating viraemia in these animals. A persistent immune response was also detected in 6 of 70 seropositive springboks after resampling 6 months apart (Dondona et al., 2016).

Wildlife ruminants may play a role in the epidemiology of RVF, more so in the inter-epizootic periods (Davies & Karstad, 1981). However, further research relating to the observed viraemia in these animals is required to determine the extent of the role in maintenance of the disease in the absence of domestic ruminants (Dondona et al., 2016).

Clinical signs
Some infected ruminants may experience inapparent infections, while others suffer from severe clinical disease with mortality and abortions. Generally, older non-pregnant animals do not show clinical disease (OIE Terrestrial Manual, 2018w).

Clinical signs observed in domestic small ruminants during inter-epizootic periods are usually non-specific and may go unnoticed. However, during an epizootic, animals suffer “abortion storms” (i.e. numerous abortions at one time) (Swanepoel & Coetzer, 2004).

In young domestic small ruminants, the disease is peracute to acute. Pyrexia is observed within 24 to 36 hours, followed by anorexia, weakness, regurgitation of ingesta, melaena, foetid diarrhoea and blood tinged mucopurulent nasal discharge. The disease in calves is similar (Swanepoel & Coetzer, 2004).

Older pregnant domestic small ruminants may abort at any time during pregnancy due to the febrile reaction or infection of the foetus. Infection in cattle is inapparent, however some animals may develop a fever, anorexia, lachrymation, salivation, nasal discharge, dysgalactia and bloody or fetid diarrhoea (Swanepoel & Coetzer, 2004).

In sheep, mortality and abortion rates vary between 5% and 100% during outbreaks. The mortality rate in cattle is normally lower than 10% (OIE Terrestrial Manual, 2018w).

Clinical signs in wildlife ruminants have not been described in detail.
Experimental infections of buffalo demonstrated a general loss of condition, which may be attributed to the immobilisation and altered environment rather than RVF infection. Of 2 pregnant buffalo, 1 aborted 16 days post infection. The fetus showed necrosis of the liver with isolation of RVFV (Davies & Karstad, 1981).

Another report also noted abortions in buffalo together with mortality in antelope species (Beechler et al., 2015).

The morbidity rate reported in an outbreak of RVF in a herd of 171 gemsboks was 40.35% with a mortality rate of 24.56% (OIE Notification Report, 2019).

Transmission

Rift Valley fever is a vector-borne disease.

Aedes and *Culex* mosquitoes are the main competent vectors of RVFV. They may transmit the virus transovarially, implying that the virus may survive in eggs for several years and then be transmitted to animals during favourable conditions (Linthicum et al., 1985). *Aedes* and *Culex* mosquitoes are present in New Zealand.

Other mosquitoes such as *Anopheles, Eretmapodites, Coquillettidia* and *Mansonia* are susceptible to infection with RVFV, but may not be capable of transmission (Pepin et al., 2010).

Transmission also occurs horizontally via direct contact with infected animal tissues, bodily fluids and fomites. Large volumes of virus are associated with abortion material and can be a major source of infection (Pepin et al., 2010). The virus is stable in the environment and may survive for extended periods (Craig et al., 1967).

Mechanical transmission (via needles) of RVFV has been described in epizootics, during periods of intense viraemia in animals (Swanepoel & Coetzer, 2004).

Diagnosis

A definitive interpretation can be obtained by using a combination of epidemiological, clinical and laboratory methods.

Virus isolation in cell culture and RT-PCR is the preferred method for the confirmation of clinical cases. Other methods that may be used include antigen detection and histopathology.

ELISA and plaque reduction neutralisation tests (PRNT) are the preferred methods to determine population freedom from infection (unvaccinated animals), immune status in individual animals or populations (post vaccination) and for eradication. ELISA and PRNT may also be used for surveillance and individual animal freedom from infection prior to movement, respectively (OIE Terrestrial Manual, 2018w).

Treatment, control and prevention

In newly affected countries, containment via movement control and elimination of the virus by the destruction of infected and potentially infected animals should be swift. A rapid response to an incursion is paramount to prevent the virus from establishing in the insect vector, domestic and wildlife populations (FAO (Food and Agriculture Organization), 2019).

In countries that are affected by RVF, measures that may implemented include:

- Chemical control of vectors by spraying animals with insecticides and systemic treatment of animals
- Moving animals from low-lying areas that are close to water sources, to higher well-drained areas
- Confining animals to insect-proof facilities
- Control of livestock movement
- Slaughter and disposal of infected animals.

The above measures are not always practical or cost-effective. In the face of an epizootic, vaccination is usually the best form of control if implemented rapidly.

Since RVF is a zoonotic disease, preventative measures should also be employed to protect people that may potentially come into contact with RVF-infected animals and animal products (OIE Terrestrial Manual, 2018w). Public awareness campaigns are essential to keep people informed about the dangers of RVF (FAO (Food and Agriculture Organization), 2019).

Prevention for areas and countries bordering RVF-affected regions includes surveillance over a distance of at least 10 kilometres from the affected area and vaccination to protect susceptible animals (FAO (Food and Agriculture Organization), 2019).

Trade restrictions on domestic ruminants and their products from RVF-affected countries would complement the prevention strategies of RVF-free countries.

Semen

Rift Valley fever phlebovirus has been listed as a pathogen that is *likely* to be present in semen of domestic ruminants and *could* be transmitted by semen (Hare, 1985). However, there is no literature evidence to demonstrate the presence of RVFV in semen or the transmission of disease to naïve dams.

Domestic small ruminants do become viraemic and may shed virus in bodily fluids. Therefore, it is plausible that the virus could be shed in semen during the viraemic period. The OIE considers germplasm of ruminants a risk commodity.

There is no published evidence demonstrating the presence of RVFV in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with RVFV-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

19.1.6 Hazard identification conclusion

Rift Valley fever is an OIE-listed disease affecting multiple species.

The presence of RVFV has not been demonstrated in the semen of domestic or wildlife ruminants. However, the OIE considers semen to be a risk commodity for RVFV.

Rift Valley fever phlebovirus is identified as a hazard in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

19.2 Risk assessment

19.2.1 Entry assessment

Rift Valley fever has never occurred in Australia, Singapore, the USA, Canada, Japan, the UK or Europe. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Captive and wild ruminants are susceptible to infection with RVFV. There is evidence that wild ruminants within the scope of this IRA are capable of developing viraemia in the absence of clinical disease, implying subclinical infection. It has also been proposed that some wildlife ruminants may be able to maintain the virus during inter-epizootic periods. Rift Valley fever in wildlife ruminants has been reported in the RSA and the UAE.

The incubation and viraemic periods in experimentally infected wildlife ruminants is short (1 to 2 days). It is, however, plausible that subclinically infected animals, not exhibiting signs of RVF, could be passed as clinically sound for export or as donors for semen collection.

There is no published evidence to demonstrate the presence of RVFV in semen. *Rift Valley fever phlebovirus* has been listed as a pathogen that is *likely* to be present in semen of domestic ruminants and *could* be transmitted by semen. The OIE considers germplasm of ruminants to be a risk commodity. Extrapolation is therefore made from domestic to wildlife ruminants.

Therefore, the likelihood of entry of RVFV via captive wild Bovidae and Giraffidae (within the scope of this IRA) from RVF-affected countries is assessed as low.

The likelihood of entry of RVFV via semen of captive wild Bovidae and Giraffidae (within the scope of this IRA) from RVF-affected countries is assessed as very low.

19.2.2 Exposure assessment

The main route of transmission is via mosquito vectors. The 2 known competent vectors are *Aedes* and *Culex* species. These genera are present in New Zealand. Transmission may also occur horizontally via direct or indirect contact with infected animal tissues, bodily fluids, fomites and mechanically in rare occasions.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

It has been reported that wild ruminants have the ability to maintain RVFV in inter-epizootic periods. It is uncertain whether animals that are harbouring the virus are able to produce a viraemia high enough to infect feeding mosquitoes. In the absence of this information, it is assumed that this scenario is probable. If imported captive wild ruminants still have viraemia on entering New Zealand, they could be a source of virus to mosquitoes in New Zealand. Infected mosquitoes could then bite other animals within the zoo. Arthropod vectors may travel long distances, and therefore, there is a likelihood that they could reach domestic or wildlife ruminants outside the zoo. They could also bite humans, thereby transmitting the virus to animals and humans within and outside the zoo. Infected domestic ruminants could then become a source for further spread of the disease.

Captive-reared buffalo have aborted due to RVF, while wild buffalo may or may not succumb to clinical disease. Viraemic wild antelope did not show signs of clinical disease.

Translocation is a very stressful time for animals. Captive wild ruminants that are subclinically infected with RVFV could develop clinical disease and suffer abortions once they arrive at zoos in New Zealand. If these animals are mixed with other animals in their enclosure, the abortion material, if not detected and removed fast enough, could be a source of infection to other in-contact susceptible animals and humans. Since the virus is quite stable in the environment, fomites could also be a source infection for a longer period.

The likelihood that RVF would establish in captive wild ruminants in zoos is very low. However, they could establish in domestic small ruminants in zoos (if any) and outside zoos.

Containment of outbreaks within the zoo may be easier to control. All animals are likely to be tested to determine exposure. However, if RVF spreads to domestic and wild animals outside the zoo containment would be harder in the absence of vaccination.

As mentioned, the likelihood of entry of RVFV in semen is very low. However, if the semen is contaminated with RVFV, the likelihood of infection resulting from insemination is unknown due to a lack of research within this area. There have been no reports of venereal transmission of RVFV or infection of naïve dams after AI with contaminated semen in domestic or wildlife ruminants.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also expected to be very low.

Therefore, the likelihood of RVFV exposure and establishment within the zoo via infected captive wild Bovidae and Giraffidae is assessed as low to moderate, the likelihood of exposure and establishment outside the zoo is assessed as low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae and Giraffidae is assessed as very low.

19.2.3 Consequence assessment

Rift Valley fever is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

There may be direct consequences for imported captive wild ruminants that may have been incubating the disease. As reported, the clinical manifestation in wildlife ruminants are rare and varied. Some may be seropositive and subclinically infected, and some may experience abortions, while mortality may be the end result in others.

Captive wild ruminants experiencing abortions and mortality will be investigated rapidly. These animals are likely to be contained until a diagnosis is obtained. However, arthropod vectors cannot be contained. These vectors could spread the virus to other susceptible species within the zoo as well as susceptible animals outside the zoo.

If the disease spread to susceptible species outside the zoo, the direct consequences for New Zealand would be the abortion storms in domestic small ruminants. Mortality and abortion rates in the naïve sheep population could vary between 5% and 100% in an outbreak. The cattle population is likely to be less severely affected with mortalities of less than 10%.

Rift Valley fever is a zoonotic disease. Humans could become infected either from mosquito bites or more commonly from contact with infected animals and animal products such as blood and abortion material. Zoo staff would be at a higher likelihood of exposure to infected animals and animal products than the public visiting the zoo. The likelihood that humans outside the zoo would be infected is very low, as transmission could only occur via mosquito bites. However, this likelihood could increase if disease spread to susceptible species outside the zoo.

Indirect consequences would entail the costs for control and surveillance within the affected zoos.

Should the disease spread further than expected, loss of valuable species within zoos and abortions of pregnant animals may also occur. This would negatively impact their ASMP and conservation efforts.

If RVF spreads to the domestic small ruminant population outside the zoo, establishment is likely to occur. Considerable costs could be incurred in order to contain and control the disease. Disease transmission is mostly due to mosquito vectors, which are currently abundant in New Zealand.

New Zealand experiences high rainfall in some areas, which leads to the mass multiplication of mosquitoes. Rift Valley fever outbreaks are often seen after heavy rains. Containment and control of RVF in the absence of vaccination would be difficult. If disease transmission is attributed to arthropods present in New Zealand, eradication may also be difficult to achieve unless vaccination is implemented.

Rift Valley fever is an OIE-listed disease, and therefore, negative trade impacts are likely to occur.

Therefore, the overall consequences as a result of an RVF incursion are assessed as high.

19.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for RVF is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and their semen.

Therefore, risk management measures can be justified.

19.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Rift Valley fever is an OIE-listed disease affecting multiple species.
- New Zealand is free from RVF, and RVF is a notifiable disease.
- Rift Valley fever is a zoonotic disease.

- Rift Valley fever has not been reported in Tragulidae species.
- Areas where RVF has never occurred include Australia, Singapore, the USA, Canada, the UK and Europe.
- The incubation period in wildlife ruminants may be 1 to 2 days.
- Long-term subclinical infection has been described in wild Bovidae species.
- Transmission of RVFV is via mosquito vectors (*Aedes* and *Culex*) and direct and indirect contact with infected animal tissues, bodily fluids and fomites. Mechanical transmission has occurred in rare cases.
- Diagnostic tests include VI in cell culture, RT-PCR, antigen detection, histopathology, ELISA and PRNT.
- *Rift Valley fever phlebovirus* has been listed as a pathogen that is *likely* to be present in semen of domestic ruminants and *could* be transmitted by semen.

19.3.1 Options

One or a combination of the following options may be used:

Option 1

1. Country freedom for RVF; AND
2. the animal(s)/donor male(s) were resident in RVF-free countries or zones since birth or for at least 14 days prior to export or semen collection; AND
3. the animal(s)/donor male(s) showed no clinical signs of disease on the day of export or semen collection, AND
4. the animal(s)/donor male(s) did not transit through an area experiencing an epizootic during transportation to the place of export; OR
5. the animal(s)/donor male(s) were protected from vector attacks when transiting through an area experiencing an epizootic.

The recommendations are similar to those of the OIE Code for the importation of ruminants from countries or zones free from RVF.

Option 2

Animals:

1. The animal(s) showed no sign of RVF on the day of export; AND
2. the animal(s) did not originate from the area of the epizootic; AND
3. the animal(s) were held for at least 14 days prior to export in a vector-protected quarantine station, which is located in an area of demonstrated low vector activity outside the area of the epizootic. During this period the animals showed no sign of RVF; AND
4. the animal(s) did not transit through an area experiencing an epizootic during transportation to the place of export; OR
5. the animal(s) were protected from vector attacks when transiting through an area experiencing an epizootic.

The recommendations are similar to those of the OIE Code for the importation of ruminants for importation from countries or zones infected with RVFV during an epizootic.

Semen:

1. Donor male(s) showed no sign of RVF within the period from 14 days prior to and 14 days following collection of the semen; AND
2. Donor male(s) were tested via paired serum samples, and results demonstrated that seroconversion did not occur between semen collection and 14 days after.

The recommendations are similar to those of the OIE Code for the importation of semen of ruminants from countries or zones not free from RVF.

20 Anthrax

20.1 Technical review

20.1.1 Aetiological agent

Family: *Bacillaceae*
Genus: *Bacillus*
Species: *Bacillus anthracis*

Bacillus anthracis (BA) is the causative agent of anthrax.

20.1.2 OIE list

Anthrax is an OIE-listed disease affecting multiple species (OIE, 2020d).

20.1.3 New Zealand status

New Zealand is free from anthrax (WAHIS, 2019d), as reported to the OIE. Anthrax was last detected in New Zealand in 1954.

Anthrax is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

20.1.4 Zoonotic potential

Anthrax is a zoonotic disease. The main source of natural human infections is direct and indirect contact with infected animals or occupational exposure to contaminated animal products (World Health Organization, 2008).

Humans may develop cutaneous anthrax (OIE Terrestrial Manual, 2018b) or, in rare cases, enteric anthrax from ingesting contaminated meat. Enteric anthrax is often fatal (World Health Organization, 2008).

20.1.5 Epidemiology

Host range

Bacillus anthracis affects most warm-blooded vertebrates. Mortality associated with anthrax has been reported in most mammals and several bird species (de Vos & Turnbull, 2004).

Anthrax has been reported in wild Bovidae and Giraffidae in the RSA (Ebedes, 1976).

Due to the wide host range of BA, it is assumed that species of the Tragulidae family may be susceptible.

Captive wild ruminants

The number of reported anthrax cases in captive collections are limited.

There are a few cases of anthrax in captive wild animals (mostly carnivores and omnivores) in zoos in the USA, Turkey and Nigeria (Ikede et al., 1976; Jordan, 1964; Lyon, 1973). The source of BA in these cases was suspected to be due to BA-contaminated meat or contaminated sand brought in from outside.

There are no known reports of anthrax cases in captive wild ruminants in zoos.

Many of Africa's wildlife reserves experience cyclic anthrax outbreaks. Kruger National Park in South Africa is an example of one. The cyclical pattern of anthrax epizootics is roughly 10 years and coincides with a dry spell. Wild ruminants affected by such outbreaks include buffaloes, elands, greater kudus, impalas and wildebeest (de Vos, 1990).

Geographical distribution

Anthrax is enzootic in most countries of Africa and Asia, a number of European countries and countries/areas of North America (including Canada) and South America.

The disease is restricted to certain zones/regions in Australia, South Africa and the USA (WAHIS, 2019d; World Health Organization, 2008).

Anthrax has never occurred in the UAE and Singapore (WAHIS, 2019d).

Pathogenesis

Bacillus anthracis is not an invasive organism. Natural infection in animals can be acquired mainly from ingestion of spores. After infecting the host, the spores germinate to form actively dividing encapsulated vegetative bacilli within the host animal. From the primary site of infection, the bacilli travel via the lymphatics to the regional lymph nodes where they multiply, and the vegetative bacilli enter the bloodstream. The antiphagocytic capsule of the bacilli helps the bacteria evade the host's immune system. The bacteria also produce a tripartite protein toxin. Together with the capsule, the toxin increases capillary permeability and delays blood clotting, resulting in oedema and haemorrhage of many tissues and organs (de Vos & Turnbull, 2004).

Infected hosts shed the vegetative bacilli, which sporulate on exposure to air. The spores can remain viable in the soil for decades until gaining access to another host where they can germinate and multiply (World Health Organization, 2008). *Bacillus anthracis* only multiplies in animals. If a carcass is opened, it sporulates, resulting in contamination of soil and the environment. In unopened carcasses, the organism does not sporulate and is destroyed by putrefaction (de Vos & Turnbull, 2004).

The variation in severity and disease progression is dependent on virulence of the bacteria, susceptibility of the animal, infective dose and the route and site of infection (de Vos & Turnbull, 2004).

The incubation period under natural conditions is thought to be 1 to 14 days (de Vos & Turnbull, 2004).

Wild ruminants such as kudus, nyalas, waterbucks and roan antelopes consistently show high terminal septicaemia. This corresponds to the animals' susceptibility to anthrax, i.e. animals with high terminal septicaemia are very susceptible to anthrax (de Vos & Turnbull, 2004).

There is evidence to suggest that a carrier state may exist in some individuals and some species. Viable, virulent BA has been isolated from apparently healthy impala that were slaughtered 4 weeks after experimental infection (de Vos, 1990).

Viable bacteria has also been isolated from lymph nodes of healthy cattle in enzootic areas in Chad (de Vos & Turnbull, 2004; World Health Organization, 2008).

However, the World Health Organization (2008) clarifies the use of terminology in older reports, stating that a carrier state has not been truly demonstrated in animals. There are, however, prolonged incubation periods, chronic infections and latent infections.

Some authors who have suggested that carrier animals may subclinically harbour the dormant bacteria at low levels and develop peracute disease when immunosuppressed, for example, due to environmental stresses such as drought (Gainer, 1987), may in fact be referring to latent infections (World Health Organization, 2008).

Clinical signs

Anthrax manifests as 3 forms, namely: peracute or apoplectic, acute and subacute to chronic (de Vos & Turnbull, 2004).

Cattle, sheep and goats normally exhibit peracute and acute disease.

Wild ruminants such as greater kudus, roan antelopes and impalas also experience peracute and acute anthrax.

In the peracute form, the course of disease is usually less than 2 hours. Animals are often found dead prior to signs of illness.

In animals that do show clinical signs, the following signs have been noted: pyrexia, restlessness, muscle tremors, dyspnoea, congestion of mucous membranes and terminal convulsions. Bloodstained fluid often exudes from the mouth, nostrils and anus.

Acute anthrax occurs in less than 72 hours. Animals are anorexic, depressed and listless. They lag behind and display laboured breathing and petechiae of the mucous membranes and skin. They also sometimes develop a haemorrhagic diarrhoea. Milk production decreases in lactating animals, and small amounts of blood-tinged or yellow milk may be secreted. Pregnant animals may abort. Oedema of the ventral parts of the body may be visible.

Subacute to chronic disease lasts at least 3 days before recovery or death. The most frequent sign is oedema of the face, throat and neck. These signs are often seen in pigs and carnivores, with some animals recovering due to a degree of natural resistance to anthrax (de Vos & Turnbull, 2004).

Impalas and other wild ruminants rarely show clinical signs until shortly before death. They are usually afebrile and severely dyspnoeic due to lung oedema and display convulsions, paddling movements of the legs and opisthotonos with forelimb rigidity (de Vos & Turnbull, 2004).

Anthrax causes high mortality in affected animals, primarily in domestic and wild ruminants (World Health Organization, 2008).

Susceptibility in animals may vary depending on the different routes of infection (de Vos & Turnbull, 2004).

Transmission

Domestic and wild ruminants can become infected when they inhale or ingest spores from contaminated soil or other contaminated material like vegetation or water.
Relatively large numbers of spores are required to cause infection via the oral route – 15 million spores are required to cause infection in impalas (de Vos & Turnbull, 2004).

Biting and non-biting flies and other insects such as mosquitoes and ticks can disseminate BA mechanically when they feed on carcasses. In many cases, these flies only spread organisms to nearby vegetation. Biting flies have been suggested to transmit BA to animals during some widespread outbreaks. Infection may also occur through abraded skin, other cutaneous lesions and through insect bites (de Vos & Turnbull, 2004; Radostits et al., 2007; World Health Organization, 2008).

Direct transmission between living animals is insignificant, but carcasses are an important source of spores. Opening an infected carcass can result in the sporulation of anthrax bacilli, resulting in contamination of soil and the environment (de Vos & Turnbull, 2004). The spores can remain viable in the soil for decades until gaining access to another host where they can germinate and multiply (World Health Organization, 2008).

Natural transmission to humans has been reported through direct and indirect contact with infected animals. Humans may contract anthrax after exposure to contaminated animal products (World Health Organization, 2008).

Diagnosis

Anthrax can be diagnosed by examining blood or tissue smears for the presence of the rod-shaped bacteria with a capsule. Samples must be collected carefully to avoid contamination of the environment and to prevent human exposure to the bacteria.

For suspected cases of anthrax, confirmation can be done through bacteriological methods. Suitable samples such as blood, mesenteric fluid, other oedematous fluid and small tissue excisions from relatively fresh carcasses can contain large numbers of bacilli, which can be seen under a microscope, cultured and isolated in a laboratory, or detected by rapid tests such as RT-PCR (de Vos & Turnbull, 2004; OIE Terrestrial Manual, 2018b).

Real-time PCR is the OIE-recommended diagnostic technique for surveillance (OIE Terrestrial Manual, 2018b).

Treatment, control and prevention

Anthrax is responsive to antibiotic therapy. Antibiotics such as penicillin and streptomycin can be used as a measure to treat animals and humans with anthrax. The clinical course of the disease in animals is often so rapid that a veterinarian may be unable to treat affected animals (OIE Terrestrial Manual, 2018b).

Treatment is, however, recommended, even in advanced stages of the disease, in order to reduce the bacterial load so that subsequent environmental contamination can be prevented. Treatment is recommended in zoological gardens for animals suspected of anthrax exposure (de Vos & Turnbull, 2004).

In anthrax enzootic countries, control is aimed mainly at domestic ruminants.

Control measures for anthrax include treatment of infected animals, surveillance, vaccination, quarantine of potential sources of infection, proper disposal of infected carcasses and disinfection.

These measures are often not practical for free-ranging wildlife. Other control measures that have been used in wildlife situations include fencing off or burning known contaminated vegetation, location, cover or incineration of carcasses as soon as possible and the replacement of natural water holes with concrete troughs that can be drained and disinfected (de Vos & Turnbull, 2004).

To ensure the safety of animal products used for human consumption, control measures such as early detection of outbreaks, quarantine of affected premises, proper destruction of diseased animals and fomites, and implementation of appropriate sanitary procedures at abattoirs and dairy factories have been proved to be effective (OIE Terrestrial Manual, 2018b).

Semen

There is no published evidence demonstrating the presence of BA in semen of domestic or wildlife ruminants.

The OIE Code considers semen a safe commodity.

20.1.6 Hazard identification conclusion

Anthrax is an OIE-listed disease affecting multiple species. Cases of anthrax in captive collections have only been reported in carnivores and omnivores.

There is no evidence demonstrating the presence of BA in semen of domestic or wildlife ruminants, and the OIE Code considers semen a safe commodity.

Bacillus anthracis is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Bacillus anthracis is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

20.2 Risk assessment

20.2.1 Entry assessment

Anthrax has never occurred in Singapore and the UAE. Disease is present in all other approved countries.
There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Anthrax has been reported in both domestic and wildlife ruminants in all other approved countries. There are several reports of anthrax in wild ruminants. However, no reports could be found of anthrax in captive wild ruminants. Animals affected by anthrax in zoological collections were carnivorous and omnivorous animals that were fed contaminated meat.

However, wild animals may be captured from national parks and game reserves and brought to captivity. Wild ruminants are susceptible to anthrax. The course of disease in wild ruminants is often peracute to acute with a very short incubation period of 1 to 3 days, ending in mortality. The subacute to chronic forms of disease are usually described in carnivores and pigs. There are rare cases where suggested latent infections of up to 4 weeks have been reported in experimentally infected impala and naturally infected cattle in enzootic areas. If these latently infected animals exist in captive collections, it is plausible that they could be passed as clinically sound for export. However, the role that these animals play in epidemiology is uncertain. Authors suggest that these animals may succumb to peracute disease after a stressful period, which could include the stress of handling and transport.

Under these circumstances, animals would die during travel. If these animals survive the travel, they could enter New Zealand carrying anthrax.

Therefore, the likelihood of entry of *Bacillus anthracis* via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from anthrax-affected countries is assessed as very low.

20.2.2 Exposure assessment

The main route of transmission is via inhalation or ingestion of spores from contaminated soil or other contaminated material like vegetation or water. Infection may also occur through abraded skin, other cutaneous lesions and through insect bites.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

Live animals do not transmit BA. However, should latently infected animals survive relocation, they are likely to succumb to disease at the zoo. In peracute to acute disease, mortality occurs within 1 to 3 days with or without clinical signs. Carcasses will be removed in order to conduct a post-mortem. Due to haemorrhaging, blood-tinged fluid may ooze from the orifices of the carcass, this fluid may contain vegetative bacilli that sporulate on exposure to air and could contaminate the environment. If the carcass is not removed prior to this happening or the area is not properly disinfected, other animals in the enclosure could be exposed to spores and become infected. Infection will likely result in death. If these animals do not die, they are likely to be isolated by the zoo, tested, monitored and treated or euthanased if there is any doubt about the infection status. Therefore, establishment within the zoo is unlikely.

The only animals likely to be exposed to spores would be those in direct contact with the infected animals. If domestic ruminants are kept in the same enclosure as infected captive wild ruminants and then released into New Zealand farms, they could be a source of infection to other animals and humans. However, infected animals will die. Therefore, the likelihood of establishment outside the zoo is assessed as negligible.

The scenario of blowflies transmitting the spores to nearby vegetation is usually seen in outbreaks involving large numbers of infected animals and is unlikely to occur in the event of a single or handful of animals dying of anthrax. Therefore, anthrax is unlikely to spread or establish in domestic or wildlife animals within or outside the zoo.

Carcasses would require a post-mortem examination to ascertain the cause of death. If the carcass is opened, the veterinary or keeping staff would be exposed to BA spores and could contract anthrax. However, if anthrax is later suspected or diagnosed, all exposed personnel can be treated with antibiotics.

There would be no exposure of the general public to anthrax spores, as they are unlikely to get close enough to a carcass or the contaminated environment.

Therefore, the likelihood of *Bacillus anthracis* exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as very low and the likelihood of exposure and establishment outside the zoo is assessed as negligible.

20.2.3 Consequence assessment

Anthrax is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand. These imports are also likely to be infrequent.

There may be direct consequences for imported captive wild ruminants that could be latently infected, incubating disease. As reported, the clinical manifestation in wildlife ruminants is usually peracute to acute, and infected ruminants will eventually succumb to mortality unless treated with antibiotics. Animals in direct contact with the carcass or contaminated environments may also be exposed and suffer the same fate. As a result, the zoo may lose valuable animals.

The likelihood of anthrax exposure to animals outside the zoo is assessed as very low. However, the likelihood of establishment is assessed as negligible. Infected animals that are released would die.

Anthrax is a zoonotic disease. If animals at the zoo die, staff could be exposed to anthrax spores and could contract the disease. However, if a diagnosis is made rapidly, all exposed staff could be treated with antibiotics.

If domestic ruminants at the zoo become infected, are released to New Zealand farms and then sent for slaughter, processing the carcass could result in a large number of processing plant staff being exposed to anthrax.

Indirect consequences would entail the costs for control and surveillance within the affected zoos. The loss of animals could negatively impact the zoos' ASMP and conservation efforts.

There may be additional costs to New Zealand for control in the event of an anthrax detection.

Anthrax is an OIE-listed disease, and therefore, negative trade impacts are likely to occur.

Therefore, the overall consequences as a result of an anthrax incursion is assessed as moderate.

20.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for anthrax is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

20.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Anthrax is an OIE-listed disease affecting multiple species.
- Anthrax was last detected in New Zealand in 1954, and it is a notifiable disease.
- Anthrax is a zoonotic disease.
- Anthrax has never occurred in the UAE or Singapore.
- Incubation period is between 1 to 14 days.
- Anthrax may exhibit prolonged incubation periods, chronic and latent infections.
- Transmission occurs via inhalation or ingestion of spores from contaminated soil or other contaminated material like vegetation or water. Infection may also occur through abraded skin, other cutaneous lesions and insect bites.
- Diagnosis can be achieved by microscopic examination of blood and tissue impression smears, laboratory culture and isolation and RT-PCR.
- The OIE Code considers semen a safe commodity.

20.3.1 Options

One or a combination of the following options may be used:

Bacillus anthracis is not identified as a hazard in semen; therefore, risk management measures are not warranted.

Option 1

1. Country freedom for anthrax; AND
2. the animal(s) were resident in anthrax free countries since birth; AND
3. the animal(s) showed no clinical signs of anthrax on the day of export.

Option 2

1. The animal(s) were kept for the 20 days prior to export in an establishment where no case of anthrax was officially declared during that period; AND
2. the animal(s) showed no clinical signs of anthrax on the day of export.

The recommendations are similar to those of the OIE Code for importation of ruminants, equids and pigs.

21 Brucellosis

21.1 Technical review

21.1.1 Aetiological agent

Family: *Brucellaceae*
Genus: *Brucella*
Species: *Brucella abortus, Brucella melitensis, Brucella ovis, Brucella suis, Brucella canis* and *Brucella neotomae* (Godfroid, 2002)

Brucella abortus (*B. abortus*), *Brucella melitensis* (*B. melitensis*), *Brucella ovis* (*B. ovis)* and *Brucella suis* (*B. suis*) are the causative agents of bovine brucellosis, caprine and ovine brucellosis, ovine epididymitis and porcine brucellosis, respectively.

Brucella suis, B. canis and *B. neotomae* have not been included as potential hazards, as there is no evidence to suggest that disease is caused by these pathogens in species within the scope of this IRA.

Brucella ovis is present in New Zealand (WAHIS, 2019d).

Therefore, *B. ovis, B. suis, B. canis* and *B. neotomae* will not be assessed further.

21.1.2 OIE list

Brucellosis (infection with *B. abortus, B. melitensis* and *B. suis*) is an OIE-listed disease affecting multiple species (OIE, 2020d).

21.1.3 New Zealand status

New Zealand is free from bovine brucellosis *(B. abortus)* (Hellstrom, 1991; WAHIS, 2019d), as reported to the OIE. Bovine brucellosis was last detected in New Zealand in 1989.

New Zealand is free from caprine and ovine brucellosis (*B. melitensis*) (WAHIS, 2019d), as reported to the OIE.

Bovine brucellosis (*B. abortus)* and caprine and ovine brucellosis (*B. melitensis)* are notifiable diseases under the Biosecurity (Notifiable Organisms) Order 2016.

21.1.4 Zoonotic potential

Brucellosis (caused by *B. abortus* and *B. melitensis*) is a zoonotic disease. *Brucella abortus* and *B. melitensis* are highly pathogenic to humans (OIE Terrestrial Manual, 2018i).

Human brucellosis is also known as undulant fever. In humans, brucellosis is often an occupational disease in people who have direct contact with infected animals. Ingestion of unpasteurised milk or cheese produced from contaminated milk can be the source of infection in humans with no direct contact with infected animals (Godfroid et al., 2004a; Wallach et al., 1997).

The most common symptoms include fever, night sweats, asthenia, insomnia, anorexia and headaches. Infections may also lead to peripheral arthritis, sacroiliitis and epididymoorchitis (Roushan & Ebrahimpour, 2015; Wallach et al., 1997).

21.1.5 Epidemiology

Host range

Brucella abortus and *B. melitensis* infections have been reported in both domestic and wildlife ruminants.

B. abortus
Brucella abortus mainly affects cattle. Other domestic ruminants affected include gayals, yaks, llamas, alpacas, sheep and goats (DAWR, 2017; OIE Terrestrial Manual, 2018i).

Dogs, horses, pigs and camels are also susceptible to infection (DAWR, 2017; OIE Terrestrial Manual, 2018i).

Brucella abortus has been isolated worldwide from various wildlife ruminants including bison, elands, waterbucks (Bovidae), elks/wapiti, reindeer and caribous (Cervidae) (Godfroid, 2002).

Various African antelopes are also susceptible to infection (OIE Terrestrial Manual, 2018i).

Giraffes in Botswana tested serologically positive on the rose bengal test (RBT) and fluorescence polarization assay (FPA) for *Brucella spp.* (Alexander et al., 2012).

B. melitensis
Brucella melitensis is common in domestic small ruminants and is rarely reported in wildlife ruminants. Cases have, however, been documented in chamois and ibexes (Caprinae) in Europe (Godfroid, 2002; Godfroid, 2017), gazelles and Arabian oryxes (Bovidae) (Ostrowski et al., 2002; Soares et al., 2019).

It is noteworthy that chamois and ibexes, along with Cervidae, are not included within the scope of this IRA.

Due to the wide host range of *B. abortus* and *B. melitensis*, it is assumed that species of the Giraffidae and Tragulidae families are susceptible to infection.

Captive wild ruminants
There are limited cases of brucellosis in captive wild animal collections.

A single case of a male Arabian oryx diagnosed with *B. melitensis* was reported at the National Wildlife Research Centre in Saudi Arabia (Ostrowski et al., 2002).

An outbreak of *B. melitensis* was reported in 47 captive gazelles at the King Khalid Wildlife Research Centre in Saudi Arabia and 726 gazelles at the Prince Mohammed Al-Sudairi Gazelle Breeding Centre (PMSGBC). The gazelles in the PMSGBC outbreak were tested for *Brucella* prior to translocation and were negative but began showing clinical signs during translocation (Soares et al., 2019).

The paucity of reports of brucellosis in captive animal collections may indicate that brucellosis in captive collections is not a common occurrence.

Geographical distribution
B. abortus
Bovine brucellosis occurs worldwide except in those countries where the disease has been eradicated. Eradication in some of these countries pertain to domestic ruminants, while disease may still be present in wildlife ruminants.

Canada, Japan, New Zealand, the UK and several countries in western and northern Europe are free from the disease (OIE Technical Disease Cards, 2018).

Australia has been free from bovine brucellosis since 1989 (DAWR, 2017; WAHIS, 2019a), and it is nationally notifiable in Australia (DAWR, 2015).

According to the OIE, bovine brucellosis in domestic animals has been absent in the USA since November 2018 (WAHIS, 2019a). However, bovine brucellosis is present in the USA in the wild and is limited to free-ranging bison (Bovidae) and wapiti (Cervidae) in the Greater Yellowstone National Park area (WAHIS, 2019d).

In the RSA, several species of wild ruminants have tested serologically positive for *B. abortus* (Gradwell et al., 1977).

B. melitensis
Caprine and ovine brucellosis has a worldwide distribution, albeit more restricted than bovine brucellosis. It occurs mainly in the Mediterranean region, west and central Asia, Central America, South America and Africa.

Caprine and ovine brucellosis was last reported in the USA in 1999 (WAHIS, 2019d).

Caprine and ovine brucellosis has never occurred in Australia, the UK and Canada (WAHIS, 2019d) and is nationally notifiable in Australia.

Brucella melitensis has been reported in chamois and ibexes in Europe and in camels in the Middle East (Godfroid, 2002).

Brucellosis (*B. abortus* and *B. melitensis*) has never occurred in Singapore (WAHIS, 2019d).

Pathogenesis

Brucellosis is a highly contagious disease that can cause severe production losses to the livestock industry.

In domestic ruminants, the incubation period for brucellosis is variable and depends on factors such as the animal's age, sex, sexual maturity and stage of pregnancy (Godfroid et al., 2004b). The incubation period can vary from 2 weeks to a year or even longer (USDA APHIS, 2010). The most prolonged recorded incubation period in a cow is 9 years (Godfroid et al., 2004a).

Calves infected in utero or at birth can remain latent for 18 months or more till pregnancy and then start showing clinical signs. During the latency period, infected heifers can remain seronegative (Godfroid et al., 2004a).

Entry and multiplication of *Brucella* in infected animals can be followed by bacteraemia. Bacteraemia can occur periodically and transiently (Olsen & Tatum, 2010) or sometimes last for several weeks or months in chronically infected animals (DAFF, 2016). During the bacteraemia, organisms are carried inside neutrophils and macrophages or can be transported free in plasma to various organs (Godfroid et al., 2004a).

Brucellosis is a spillover or self-limiting disease in wildlife ruminants (Godfroid, 2002). There are a few exceptions where wildlife ruminants are regarded as reservoirs for brucellosis: bison and elk (*B. abortus*) in the USA and Canada; Cape buffalo (*B. abortus*) in the RSA and the Alpine ibex (*B. melitensis*) in the French Alps (Godfroid, 2002; Godfroid, 2017). These species are however not included within the scope of this IRA.

Wildlife ruminants were reported to be dead end hosts of *B. melitensis*. The disease in these animals has been known to disappear once eliminated from domestic species they are in contact with (OIE Terrestrial Manual, 2018i).

However, a 12-year study of ranched sable antelope in the RSA concluded that infection with *B. melitensis* was maintained in the sable herd for at least 12 years in the absence of apparent spillover from livestock (Glover et al., 2020).

Clinical signs

Duration of brucellosis is variable. Some infected animals can rid themselves of disease within months while others may experience chronic disease (FAO, 2003; Godfroid et al., 2004a).

Brucellosis is usually subclinical in young animals and non-pregnant heifers. The clinical signs in pregnant adult domestic ruminants include abortion, stillbirth, the birth of weak offspring, retained placenta, reduced milk yield and, rarely, arthritis (Godfroid et al., 2004a; OIE Terrestrial Manual, 2018i).

The abortion rate in cattle varies from 30% to 80% in infected herds (Godfroid et al., 2004a).

In bulls, orchitis, epididymitis and seminal vesiculitis occur (Godfroid et al., 2004a).

Mortality is rare, except in fetuses and newborns. Hygromas and non-suppurative arthritis have also been reported in infected cattle (Godfroid et al., 2004a; OIE Terrestrial Manual, 2018i).

Clinical signs of bovine brucellosis in wildlife ruminants are similar to those of domestic ruminants (Godfroid, 2002; Gradwell et al., 1977).

Manifestations of caprine and ovine brucellosis in wildlife ruminants are similar to those experienced in domestic ruminants (Ostrowski et al., 2002; Soares et al., 2019). However, there are several species that suffer from purulent or calcified arthritis and orchitis, uveitis and neurological signs (Godfroid, 2002; OIE Terrestrial Manual, 2018i).

Transmission

Transmission is mainly via direct contact (ingestion, inhalation, through skin abrasions and through mucous membranes) with fluids and tissues (fetal membranes, lochia, post-parturient discharges) from infected animals (Olsen & Tatum, 2010).

Aerosol transmission is not a major route of transmission. Aerosol transmission has been the cause of outbreaks in confined spaces such as laboratories or abattoirs rather than open environments (Kaufmann et al., 1980).

Indirect transmission through ingestion of contaminated feed and water has been reported, as well as through fomites from the environment and contaminated equipment used for artificial insemination and milking (Aparicio, 2013; Godfroid et al., 2004a).

Face flies can be transient transmission vectors (Cheville et al., 1989).

The PMSGBC outbreak in gazelles was suspected to have stemmed from indirect contact with positive *B. melitensis* domestic ruminants grazing outside the centre. This could have been via scavenger species like feral dogs and foxes that tested positive for *Brucella* (Soares et al., 2019).

In the Arabian oryx case, it was suspected that a raven may have carried the bacteria from infected domestic ruminants surrounding the centre into the oryx's pen (Ostrowski et al., 2002).

Vertical transmission occurs in utero (Godfroid et al., 2004a) and through the shedding of *Brucella* in colostrum and milk (Olsen & Tatum, 2010).

Infected semen may act as a source for the transmission of the disease (Aparicio, 2013; Godfroid et al., 2004a).

Transmission between domestic and wild ruminants has been demonstrated (Godfroid, 2002).

The bacteria can survive in the environment for up to 60 days at low temperatures and in highly organic material (American Association of Zoo Veterinarians Infectious Disease Committee Manual, 2013; Soares et al., 2019).

Diagnosis

A definitive identification of the agent can be achieved by bacteriological, serological and molecular methods.

It is important that diagnosis also focuses on early detection of preclinical and subclinical infections (Godfroid, 2002). Latent infections are problematic in eradication efforts, as animals remain serologically negative until close to first calving, during abortion, or during a stressful event (Soares et al., 2019).

Bacteriological diagnosis is made by isolating and identifying the causative organisms (Nielsen, 2002; OIE Terrestrial Manual, 2018i). This may include microscopic examination, culture and typing, and PCR. The OIE recommends culture and the brucellin skin test (BST) for the confirmation of suspected or clinical cases (OIE Terrestrial Manual, 2018i).

OIE-recommended methods for population freedom from infection and eradication are the rose bengal test (RBT) and buffered plate agglutination test (BPAT), indirect ELISA and bulk milk tests (i.e. milk I-ELISA or milk ring-test).

The RBT and BPAT, CFT, I-ELISA and bulk milk tests are recommended for surveillance purposes (OIE Terrestrial Manual, 2018i).

Treatment, control and prevention

No effective treatment is available to completely cure *B. abortus* infected animals (Godfroid et al., 2004a; Soares et al., 2019) due to the persistence of the bacteria in lymph nodes and other tissues.

In humans, treatment with various antibiotic combinations is recommended (Roushan & Ebrahimpour, 2015; Wallach et al., 1997).

Vaccination may reduce the prevalence of bovine brucellosis (DAWR, 2017) and help control the clinical signs. The *Brucella abortus* strain 19 and strain RB51 vaccines are the most common vaccines available for use.

In the zoological setting, prevention and control of brucellosis would be aimed at preventing entry by quarantining animals prior to introduction and returning serological reactors. For animals present at the zoo, testing and slaughtering of serological reactors would be required.

Semen

Infected domestic ruminant bulls can shed *B. abortus* in their semen and seminal fluid (Eaglesome & Garcia, 1997; Godfroid et al., 2004a).

Brucella melitensis in the seminal fluid fraction and non-sperm fractions of semen of bulls and rams has been identified by both PCR and direct culture methods (Amin et al., 2001).

Although *B. melitensis* has not been demonstrated in the semen of sable antelope, venereal transmission has been suggested. Artificial insemination with *B. melitensis*-contaminated semen can be high risk with regards to disease spread (Glover et al., 2020).

Artificial insemination of domestic ruminant cows with raw *Brucella*-contaminated semen has resulted in brucellosis (Robison et al., 1998). Artificial insemination may therefore act as a source for the transmission of the disease (Aparicio, 2013; Campero et al., 1990; Godfroid et al., 2004a).

Robison et al. (1998) demonstrated that bison are able to intermittently shed *B. abortus* in semen. This was confirmed in an experimental study where bison bulls were injected with corticosteroids to induce immunosuppression and enhance the excretion of *B. abortus* in semen. Four bison cows bred to a *B. abortus*-positive bull by natural service became pregnant and produced calves. Neither the calves nor the naïve cows showed evidence of *B. abortus* infection (Robison et al., 1998).

Brucellosis transmission to naïve dams via natural service is extremely rare (Kaushik et al., 2006; Radostits et al., 2007) and is not a significant transmission route (Robison et al., 1998).

The difference noted between AI and natural service could be explained by the site of deposition of the contaminated semen. Semen is deposited into the uterus during AI, which is an ideal environment for *B. abortus.* During natural service, semen is deposited into the vagina, which may be a harsh environment for the survival of the bacteria (Robison et al., 1998).

21.1.6 Hazard identification conclusion

Brucellosis (infection with *B. abortus, B. melitensis* and *B. suis*) is an OIE-listed disease affecting multiple species.

The presence of *B. abortus* has been demonstrated in the semen of domestic and wildlife ruminants (though not covered within the scope of this IRA). Artificial insemination with *B. abortus*-contaminated semen has resulted in the transmission of brucellosis to naïve domestic ruminants.

Brucella melitensis has only been isolated from semen of domestic ruminants. Studies have, however, suggested venereal transmission of *B. melitensis* in sable antelopes.

As a result, it is assumed that *B. abortus* and *B. melitensis* may be present in the semen of wildlife ruminants and may result in the transmission of infection to naïve ruminants after artificial insemination with contaminated semen.

Brucella abortus and B. melitensis are identified as hazards in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

21.2 Risk assessment

21.2.1 Entry assessment

Brucellosis (*B. abortus* and *B. melitensis*) has never occurred in Singapore and has been eradicated from Australia and the UK. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

There is evidence that *B. abortus* and *B. melitensis* can infect and cause disease in a range of wildlife ruminants in all other approved countries. There are a handful of cases of caprine and ovine brucellosis in captive wild Bovidae in the UAE. This implies that while brucellosis in captive wild ruminants is a rare occurrence, these animals are still susceptible to and may carry the disease.

In captive collections, clinical signs were noted in animals only once exposed to stressful conditions. The manifestations of brucellosis vary and include subclinical infections, latency and reproductive disorders. Mortality is rare. Incubation periods are also variable and may range from 2 weeks to 1 year or longer.

It is plausible that subclinically or latently infected animals, not exhibiting signs of brucellosis, could be passed as clinically sound for export or as donors for semen collection.

Brucella abortus and *B. melitensis* have been detected in the semen of domestic ruminants (although not covered in the scope of this IRA). *Brucella abortus* has also been detected in the semen of a wild Bovidae. However, excretion in semen was intermittent.

Artificial insemination with *B. abortus*-contaminated semen has resulted in the transmission of brucellosis to naïve domestic ruminants.

It is assumed that *B. abortus* and *B. melitensis* may be present in the semen of wildlife ruminants (within the scope of this IRA) and may result in the transmission of infection to naïve ruminants after artificial insemination.

Therefore, the likelihood of entry of *B. abortus* and *B. melitensis* via captive wild Bovidae, Giraffidae, Tragulidae (within the scope of this IRA) and their semen from brucellosis-affected countries is assessed as very low.

21.2.2 Exposure assessment

Transmission routes include direct and indirect contact with infected animals, via ingestion, inhalation, through skin abrasions and through mucous membranes with fluids and tissues (fetal membranes, lochia, and post-parturient discharges), fomites and contaminated feed and water. Vertical transmission occurs in utero and through the shedding of *Brucella* in colostrum and milk.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

Imported infected female animals are likely to act as a source of infection after giving birth or aborting. The bacteria can be found in high volumes in parturient material. If not detected and removed fast enough, this tissue could be a source of infection to other in contact susceptible animals and humans.

Susceptible captive wild ruminants within zoos are likely to contract brucellosis if they are in the same enclosures with infected animals. Other susceptible animals (not in direct contact) may also become infected via flies or fomites if the same zookeepers manage the enclosures or the same equipment is used. If domestic ruminants are kept at zoos in the same enclosures, they too could be susceptible to infection.

As demonstrated, brucellosis is likely to cause clinical signs in captive wild ruminants after a stressful situation. Abortions or ill animals are likely to be rapidly investigated limiting exposure to in contact animals. However, if animals are not pregnant, there may be no obvious signs, thus leading to chronic disease and constant exposure to in contact animals. As mortality is rare, brucellosis could establish in the captive wild and domestic ruminants within the zoo.

Brucella has the ability to survive in the environment for up to 60 days at low temperature and in high organic matter. If abortion and parturient material are not cleaned and the area is not disinfected, it could be a source of infection to other susceptible animals in the enclosure.

Brucella spp. can be transmitted via aerosols. However, the cases that have been described are often in confined spaces with high volumes of aerosolised bacteria. It is unlikely that a handful of infected animals in the zoo would lead to an infective dose of *Brucella* that would result in aerosol infection of susceptible domestic or wildlife ruminants outside the zoo. It is also unlikely that scavenger species would carry the bacteria from captive wild animals to animals outside the zoo.

However, if domestic ruminants become infected and are released onto New Zealand farms, they could transmit *B. abortus* and *B. melitensis* to other domestic animals. Brucellosis could spread and establish in the domestic animal population.

As mentioned, the likelihood of entry of *B. abortus* and *B. melitensis* in semen is very low. However, if the semen is contaminated with *B. abortus* and *B. melitensis,* the likelihood of infection resulting from artificial insemination is moderate as *Brucella* transmission via AI has been demonstrated in domestic ruminants.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of *B. abortus* and *B. melitensis* exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as low, the likelihood of exposure and establishment outside the zoo is assessed as very low, and the likelihood of exposure and establishment via contaminated semen of captive wild Bovidae, Giraffidae and Tragulidae is assessed as low.

21.2.3 Consequence assessment

Brucellosis (infection with *B. abortus, B. melitensis* and *B. suis*) is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of brucellosis would be due to infected imported animals that may succumb to clinical disease during pregnancy, after stressful conditions or chronic disease. Mortality is rare, and therefore, there would be no loss of valuable animals. However, brucellosis is likely to cause abortions and other reproductive disorders in both male and female animals. This would negatively impact the breeding programmes in zoos.

Since brucellosis rarely causes mortality and could lead to subclinical, latent and chronic infections, the disease could establish in the domestic and captive wild ruminants within the zoo. Establishment in these populations could lead to a continuous source of infection to other animals within the zoo.

The likelihood of brucellosis exposure and establishment in domestic and wild ruminants outside the zoo is assessed as very low. *Brucella abortus* has a wide host range and can result in infection of domestic ruminants (cattle, sheep, goats, alpacas), dogs, horses and pigs, while *B. melitensis* affects sheep and goats. Affected animals are likely to suffer reproductive disorders. Abortion rates in cattle vary from 30% to 80% in infected herds. New Zealand's cattle and sheep population could be severely affected.

Brucellosis is a zoonotic disease. Humans could become infected through direct contact with contaminated animal products such as abortion material. Zoo and veterinary staff could be exposed to infected animals and contaminated animal products. The public would not come into contact with infectious material, and therefore, the likelihood of exposure to the public would be negligible. The likelihood that humans outside the zoo would be infected is negligible.

Indirect consequences would entail the costs for control and surveillance within the affected zoos.

There would be additional costs to New Zealand for brucellosis control in the event of an incursion in domestic ruminants.

Any trade impacts as a result of a *B. abortus* or *B. melitensis* incursion at the zoo is likely to be minimal if New Zealand can prove that the incursion was contained and there was no spread to animals outside the zoo. However, this may be difficult to prove if domestic animals are released from the zoo.

Therefore, the overall consequences as a result of a brucellosis incursion are assessed as moderate.

21.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for brucellosis is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

Therefore, risk management measures can be justified.

21.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Brucellosis (infection with *B. abortus, B. melitensis* and *B. suis*) is an OIE-listed disease affecting multiple species.
- Bovine brucellosis (*B. abortus*) was last detected in New Zealand in 1989.
- Caprine and ovine brucellosis (*B. melitensis*) has never occurred in New Zealand.
- Brucellosis (caused by *B. abortus* and *B. melitensis*) is a zoonotic disease.
- *Brucella abortus* and *B. melitensis* infections have been reported in domestic and wildlife ruminants.
- Brucellosis (*B. abortus* and *B. melitensis*) has never occurred in Singapore and has been eradicated from the UK and Australia.
- The incubation period may be 2 weeks to 1 year or longer.
- Wildlife reservoirs of brucellosis include bison, elks, African buffaloes and Alpine ibexes.
- Clinical signs vary and include subclinical, latent and chronic infections or reproductive disorders. Mortality is rare.
- Transmission is via direct and indirect contact (ingestion, inhalation, through skin abrasions and through mucous membranes) with fluids and tissues and vertically in utero and through milk.
- Diagnosis includes bacterial culture and typing, PCR, BST, RBT, CFT, BPAT, indirect ELISA and bulk milk tests.
- *Brucella abortus* and *B. melitensis* has been detected in the semen of domestic and wildlife ruminants. Artificial insemination with *B. abortus*-contaminated semen has resulted in the transmission of infection to naïve dams.

21.3.1 Options

One or a combination of the following options may be used:

Option 1

1. Country freedom for brucellosis (*B. abortus* and *B. melitensis*); AND
2. the animal(s)/donor male(s) were resident in brucellosis (*B. abortus* and *B. melitensis*) free countries since birth or for at least 2 years immediately prior to export or semen collection; AND
3. the animal(s)/donor male(s) showed no clinical signs of brucellosis on the day of export or semen collection.

Option 2

Animals:

1. The animal(s) were resident since birth or for 12 months immediately prior to export in a facility where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* and *B. melitensis*) has occurred in any species during the previous 2 years and the disease is compulsorily notifiable in the country; AND
2. the animal(s) showed no clinical signs of brucellosis on the day of export; AND
3. the animal(s) was tested with an MPI approved test for brucellosis (*B. abortus* and *B. melitensis*) within 30 days of export. In the case of post-parturient females, the test was carried out at least 30 days after giving birth. The test result was negative.

Semen:

1. The donor male(s) were resident since birth or for 12 months immediately prior to semen collection, in a facility where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* and *B. melitensis*) has occurred in any species during the previous 2 years and the disease is compulsorily notifiable; AND
2. the donor male(s) showed no clinical signs of brucellosis on the day of semen collection; AND

3. the donor male(s) were subjected to an MPI-approved test for brucellosis (*B. abortus* and *B. melitensis*) within the 30 days immediately after collection. The test result was negative; AND/OR
4. A straw from each semen sample was tested for brucellosis (*B. abortus* and *B. melitensis*) with an MPI-approved test with negative results.

22 Bovine tuberculosis

22.1 Technical review

22.1.1 Aetiological agent

Family: *Mycobacteriaceae*
Genus: *Mycobacterium*
Species: *Mycobacterium bovis*

The Mycobacterium tuberculosis complex (MTC) includes *M. tuberculosis, Mycobacterium bovis, Mycobacterium bovis* BCG (vaccine strain), *M. africanum*, *M. microti*, *M. caprae* and *M. pinnipedii* (Govender, 2013), also known as 'tubercle bacilli' (Cousins et al., 2004)

Of the MTC members, *Mycobacterium bovis* will be assessed, as there is evidence of association with the commodity.

Mycobacterium bovis is the causative agent of bovine tuberculosis (BTB).

22.1.2 OIE list

Infection with MTC is an OIE-listed infection affecting multiple species (OIE, 2020e).

22.1.3 New Zealand status

Mycobacterium bovis is present in New Zealand. The point prevalence of infected cattle and deer herds at 30 June 2017 was 0.08%. The 12-month period prevalence for 2016 to 2017 was 0.11% (Livingstone & Edge, 2019). Bovine tuberculosis is subject to an eradication programme by TBfree New Zealand, which is a programme under OSPRI. It is responsible for implementing the national pest management plan under the Biosecurity Act 1993. Bovine tuberculosis is not a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

22.1.4 Zoonotic potential

Bovine tuberculosis is a zoonotic disease. Humans become infected after close contact with infected animals or consumption of unpasteurised contaminated dairy products (Lan et al., 2016; OIE Terrestrial Manual, 2018g).

22.1.5 Epidemiology

Host range

Almost all higher animals (mammals, birds, reptiles and fish) are susceptible to the tubercle bacillus (Henning, 1995).

Mycobacterium bovis is the primary agent causing disease in cattle, but can also affect other domestic animals and wildlife (OIE Terrestrial Manual, 2018g).

Mycobacterium bovis has been isolated in elks, deer (Cervidae), giraffes (Giraffidae), buffaloes, impalas (Böhm et al., 2007; Brook & McLachlan, 2006; Govender, 2013; Krajewska-Wędzina et al., 2018), captive bison, kudus, duikers, and Arabian oryxes (Bovidae) (de Lisle et al., 2001; Flamand et al., 1994; Himes et al., 1976; OIE Terrestrial Manual, 2018g).

Captive wild ruminants

Cases of BTB have been described in various captive wild Bovidae and Giraffidae at zoos in approved countries (Amanfu, 2006; de Lisle et al., 2001; Flamand et al., 1994; Govender, 2013; Himes et al., 1976; OIE Terrestrial Manual, 2018g).

Geographical distribution

Bovine tuberculosis was last reported in Australia in 2002 and in Singapore in 2016. The disease has never been reported in the UAE. Bovine tuberculosis is present in the UK, the USA, Canada, Europe and the RSA (WAHIS, 2019d).

Pathogenesis

The incubation period of BTB is variable and depends on the age of the animal, route of infection, strain of organism and dose of bacteria.

The outcome of a mycobacterial infection is also dependent on whether the animal has been previously sensitised or not (Cousins et al., 2004). In domestic ruminants like cattle, lesions may remain dormant, progress or regress (Neill et al., 2001). In unsensitised animals, bacteraemia may develop as soon as 20 days post infection (Cousins et al., 2004).

In a BTB case in captive kudus, infected animals showed no significant signs of illness until the disease had progressed to lesions in the lungs. The duration of infection was unknown, and therefore, the animals could have been incubating the disease for months or years (Himes et al., 1976).

Tuberculosis develops and spreads in 2 stages, the primary complex and post-primary dissemination. The primary complex consists of the lesion at the point of entry and the local lymph node. A visible primary focus develops within 8 days of entry. Calcification of the lesion occurs 2 weeks later but not in all cases. The necrotic foci are soon surrounded by granulation tissue and lymphocytes, and the pathognomonic “tubercle” is established. Post-primary dissemination occurs when bacteria are transmitted from the primary focus either in the respiratory tract to the regional lymph node and lower respiratory tract and cause a similar lesion. This may also occur via transmission from the alimentary tract (pharyngeal or mesenteric lymph nodes) to the liver (Henning, 1995). Haematogenous spread may result in miliary tuberculosis which is caused by acute bacteraemia (Cousins et al., 2004).

Bison and Cape buffaloes are maintenance hosts of BTB (de Lisle et al., 2001). These species are, however, not within the scope of this IRA. Greater kudus in the KNP in the RSA are reported to have maintenance host potential (Michel et al., 2006). Further evidence to prove this theory is required.

Clinical signs

Tuberculosis is generally a chronic disease; clinical signs in the early stages are usually inapparent. Clinical signs may vary depending on the sites of localisation of the infection. For example, differences may be seen with involvement of the pleura, bowel and mesenteric glands, liver, peritoneum and reproductive organs, and therefore, generalisations cannot be made (Cousins et al., 2004; Drewe et al., 2009; Henning, 1995). In most species, tuberculosis is a chronic progressive disease, with infection usually lasting for many weeks and disease lasting for several months or years (Drewe et al., 2009).

In pulmonary tuberculosis of domestic ruminants, clinical signs start out as a short, dry, vigorous cough when the animal rises or drinks cold water or in cold weather. Within a few months, the coughing becomes more frequent and appears more painful. The breath has a foul odour, and there may be periodic nasal and buccal mucopurulent discharges. Eventually hyperpnoea and dyspnoea are accompanied by grunting and groaning. The animals suffer from anorexia and become emaciated and anaemic (Henning, 1995).

Signs of the alimentary form are diarrhoea and constipation, and they are also caused by pressure of the enlarged lymph nodes. Enlarged retropharyngeal lymph nodes cause dysphagia and irregular breathing sounds.

Tuberculous mastitis is of major importance because of the danger to public health and the spread of the disease to calves. A characteristic marked induration and hypertrophy of the udder occurs (Blood et al., 1979).

Haematogenous spread is characterised by discrete nodular lesions in various organs or chronic organ tuberculosis (Cousins et al., 2004).

Greater kudus show distinct clinical signs of BTB characterised by uni- or bilateral abscessation of parotid lymph nodes, which are usually accompanied by formation of draining fistulae (Michel et al., 2006). Arabian oryxes infected with BTB showed signs of emaciation, coughing, rales, conjunctivitis and general weakness, while some showed no signs at all (Flamand et al., 1994). In cervids, tubercles are often found in the lymph nodes of the head and thorax. In some species, discharging sinus tracts occur in cranial lymph nodes (Zanella et al., 2008).

Transmission

It is suggested that the respiratory tract is the most common portal of entry of the organism especially in animals that are in close contact (Liebana et al., 2008). Infection occurs via inhalation of aerosol and infected droplets from a coughing or sneezing animal or dust particles (Cousins et al., 2004).

Mycobacteria are excreted in exhaled air, in sputum, faeces, milk, urine, vaginal and uterine discharges and discharges from fistulated peripheral lymph nodes (Thoen et al., 1977). The mycobacteria can spread through direct contact between infected and susceptible animals, through airborne exposure, or through shared foods, milk, urine and faeces (Brook & McLachlan, 2006). The drinking of infected milk by young animals and aerosol transmission are the most common methods by which tuberculosis is spread in young animals, both wild and domestic.

Venereal transmission is rare but has been reported in domestic ruminants (Thoen et al., 1977).

Diagnosis

The detection of BTB in wildlife individuals and populations depends on bacteriological investigation, the type of tests used, the use of valid testing methods for these species and epidemiological evaluation of information.

Various tests and testing methods are available for BTB diagnosis (Cousins & Florisson, 2005; Govender, 2013; OIE Terrestrial Manual, 2018g).

In order to identify the agent, the following tests may be used:

- Microscopic examination
- Culture (gold standard method for routine confirmation of infection)
- PCR

Delayed hypersensitivity tests:

- Tuberculin test (prescribed test for international trade)

Blood-based laboratory tests:

- Gamma-interferon assay
- Lymphocytic proliferation assay
- ELISA
- Fluorescence polarisation assay
- Chembio's rapid card serological test
- Multi-antigen print immunoassay.

Treatment, control and prevention

The treatment of BTB is not usually attempted and is prohibited in domestic ruminants in some countries. Antibiotic treatment may be attempted in rare cases to salvage important genotypes and valuable animals, such as in the case of captive wild animals (Bourne, 2020).

In countries where BTB is present, the control of BTB in wildlife is aimed at preventing the spread of BTB from wildlife to domestic animals and humans and from livestock to wildlife, as well as the preservation of protected wildlife. Where wildlife is not protected, levels of culling of these species may form part of the control strategy.

Vaccination of wildlife may be another means of protecting wildlife and preventing the spread of BTB from these species; however, this is still under research (de Lisle et al., 2001; OIE Terrestrial Manual, 2018g).

The options for the control of BTB in wildlife in Africa include a laissez faire approach, minimal interference, limited intervention and major intervention.

Minimal interference includes active intense research and monitoring of the disease but no active control efforts. This is the option of choice in the KNP in the RSA, while limited intervention is being carried out in the Hluhluwe-iMfolozi Park in the RSA (de Lisle et al., 2001).

Limited intervention involves focal depopulation of maintenance hosts to create a host-free zone, depopulation of high prevalence herds to reduce spillover, or depopulation of finite infected foci and mass capture of maintenance hosts, followed by testing and removal of positive animals.

Major intervention involves total depopulation of infected herds in the conservation area with the intention of repopulating the area after a period of time with BTB-free animals (de Lisle et al., 2001).

The tuberculin testing and culling strategy in some countries has led to the elimination of BTB in domestic ruminant populations.

Semen

Infectious domestic ruminants may contain tubercle bacilli in their semen (Cousins, 2001).

Mycobacterium spp. have been listed as organisms that are known to be present in large ruminant semen and are known to transmit disease via artificial insemination (Hare, 1985).

In cases where haematogenous spread of tubercle bacilli has led to lesions in the prostate, seminal vesicles and testes, semen is almost always contaminated due to shedding from these lesions. *Mycobacterium bovis* DNA has also been detected via PCR in naturally contaminated semen of a known/suspected BTB-positive bull (Ahmed et al., 1999).

There is no published evidence demonstrating the presence of *Mycobacterium bovis* in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with *Mycobacterium bovis* contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

22.1.6 Hazard identification conclusion

Infection with the MTC is an OIE-listed infection affecting multiple species.

Mycobacterium bovis DNA has been detected in the semen of domestic ruminants. It is suggested that *Mycobacterium* spp. can be transmitted via AI with contaminated semen. Reports on the presence of *Mycobacterium* spp. in semen are rare, implying occurrence is not common. There is no evidence demonstrating the presence of *Mycobacterium bovis* in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with *Mycobacterium bovis*-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

Mycobacterium bovis is identified as a hazard in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

22.2 Risk assessment

22.2.1 Entry assessment

According to the WAHIS interface, BTB (*Mycobacterium bovis*) was last reported in Singapore in 2016, in Australia in 2002 and has never occurred in the UAE. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, these imports are also likely to be infrequent. Therefore the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

There are numerous reports of BTB in wildlife ruminants in approved countries. The incubation period and clinical manifestation of BTB in wildlife ruminants is variable. In some animals, the incubation period may be protracted if the mycobacteria are contained in necrotic foci. These animals may remain subclinical and not show any obvious signs of disease for months or years. In some captive collections, clinical signs were noted in some animals only after exposure to stressful conditions. It is plausible that subclinically infected animals, not exhibiting signs of BTB, could be passed as clinically sound for export or as donors for semen collection.

Mycobacterium bovis DNA has been detected in the semen of domestic ruminants. It is suggested that *Mycobacterium* spp. can be transmitted via AI with contaminated semen. There is currently no published evidence of *Mycobacterium bovis* in semen of wildlife ruminants or the transmission of disease to naïve dams if inseminated with *Mycobacterium bovis* contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

Therefore the likelihood of entry of *Mycobacterium bovis* via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from approved countries is assessed as low.

The likelihood of entry of *Mycobacterium bovis* via semen of captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from approved countries is assessed as very low.

22.2.2 Exposure assessment

Transmission of BTB occurs via direct and indirect contact with infected animals and contaminated animal products through inhalation and ingestion. Mycobacteria are excreted in exhaled air, in sputum, faeces, milk, urine, vaginal and uterine discharges and discharges from open peripheral lymph nodes.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

The progression of disease in infected captive wild ruminants is variable. If subclinically infected animals are imported, there is a high likelihood that exposure to stressful conditions of translocation would result in progression of infection and the manifestation of clinical signs. If these animals suffer open tuberculosis or miliary tuberculosis, they can shed mycobacteria, whereas some other animals may have lesions that could remain latent or regress.

Since greater kudus appear to be the only animals within this scope that show distinct clinical signs of BTB, other ill or infected species may go unnoticed, excreting mycobacteria, contaminating the environment and acting as a source of infection for other in-contact susceptible species. Susceptible captive wild animals within zoos are likely to contract BTB if they are in direct contact in the same enclosures with infected animals. As mentioned, all higher animals are susceptible to infection with BTB. Other susceptible animals (not in direct contact) may also become infected via fomites if the same zookeepers manage the enclosures or the same equipment is used.

Cattle, bison, Cape buffaloes and potentially greater kudus are maintenance hosts of BTB.
If these animals are present in zoos and become infected, they could maintain the disease and become a continuous source of infection. Animals that do become infected are likely to eventually succumb to clinical disease and die. However, due to the variability of disease progression, this may take months or years. If BTB is not suspected or diagnosed, transmission to many animals could occur prior to the death of infected animals. Bovine tuberculosis could therefore establish in the captive wild animal population prior to the disease being identified.

The most common route of transmission of BTB is via inhalation of aerosol droplets. Transmission has been reported in animals in close contact or sharing the same enclosures. If domestic animals at the zoo are kept in close contact or in the same enclosure, they could become infected. Should these domestic animals be released onto New Zealand farms, they could transmit *Mycobacterium bovis* to other domestic animals. Bovine tuberculosis is present in New Zealand, and therefore, establishment is evident.

Infected animals may also be a source of infection to zoo personnel. The likelihood of exposure to the public is negligible unless there are animal encounters (i.e. direct contact) with infected species.

There is uncertainty as to whether *Mycobacterium bovis*-infected wildlife ruminants are likely to produce *Mycobacterium bovis*-contaminated semen. It is also not known whether naïve wildlife ruminants will become infected with *Mycobacterium bovis* if contaminated semen is used for AI. There is a single report of transmission of mycobacterium via AI in domestic ruminants. By extrapolating the outcomes from domestic ruminants to wildlife ruminants, it is assumed that infection may occur.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore, the likelihood of *Mycobacterium bovis* exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate, the likelihood of exposure and establishment outside the zoo is assessed as very low, and likelihood of exposure and establishment via contaminated semen of captive wild Bovidae, Giraffidae and Tragulidae is assessed as very low.

22.2.3 Consequence assessment

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of BTB would be due to infected imported animals that may succumb to clinical manifestation of BTB after stressful conditions, chronic disease and eventual mortality. This will result in loss of valuable species and negatively impact the breeding programmes in zoos.

Bovine tuberculosis could result in subclinical, latent and chronic infections. Therefore, the disease could establish in the captive wild animal population. Establishment in these populations could lead to a continuous source of infection to other animals within the zoo and cause further losses.

The likelihood of BTB exposure and establishment in animals outside the zoo is assessed as very low. It is noteworthy that BTB is present in New Zealand cattle and deer herds. In June 2017, the point prevalence of infected cattle and deer herds was 0.08%, and OSPRI are currently working towards eradication. Bovine tuberculosis is also present in brushtail possums, which are maintenance hosts in New Zealand. The release of probably infected domestic animals from the zoo could result in an additional source of *Mycobacterium bovis*, which may thwart current eradication efforts.

Bovine tuberculosis is a zoonotic disease. Humans could become infected through close contact with infected animals. Zoo personnel could be exposed to infected animals. However, this has been documented to be uncommon. Human infections can be treated with antibiotics.

The public would not come into contact with infected animals (with the exception of animal encounters) and are not likely to drink milk from animals at the zoo, and therefore, the likelihood of exposure to the public would be very low.

Bovine tuberculosis is present in New Zealand at a low incidence rate. Humans and animals are currently exposed to the disease, but this is being managed by a control scheme.

Indirect consequences would entail the costs for control and surveillance within the affected zoos. This could amount to an expensive exercise.

Although infection with MTC is an OIE-listed infection affecting multiple species, there would be no negative trade impacts due to New Zealand's current BTB status.

Therefore, the overall consequences as a result of a BTB incursion are assessed as very low.

22.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for BTB is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae, Tragulidae and their semen.

Therefore, risk management measures can be justified.

22.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Infection with MTC is an OIE-listed infection affecting multiple species.
- *Mycobacterium bovis* is present in New Zealand and is subject to an eradication programme.
- Bovine tuberculosis is a zoonotic disease.
- Almost all higher animals (mammals, birds, reptiles and fish) are susceptible to the tubercle bacillus.
- The incubation period of BTB is variable and may range from days to years.
- Transmission of BTB occurs via direct and indirect contact with infected animals and contaminated animal products through inhalation and ingestion.
- Mycobacteria are excreted in exhaled air, in sputum, faeces, milk, urine, vaginal and uterine discharges and discharges from fistulated peripheral lymph nodes.
- Diagnosis includes microscopic examination, culture, PCR, tuberculin test, gamma-interferon assay, lymphocytic proliferation assay, ELISA, FPA, Chembio's rapid card serological test and multi-antigen print immunoassay.
- *Mycobacterium* spp. have been listed as organisms that are known to be present in large ruminant semen and are known to transmit disease via artificial insemination.

22.3.1 Options

One or a combination of the following options may be used:

Option 1

1. Country/zone freedom for BTB; AND
2. the Animal(s)/donor male(s) were resident in BTB-free countries or zones since birth; AND
3. the Animal(s)/donor male(s) showed no clinical signs of BTB on the day of export.

Option 2

Animals:

1. The animal(s) showed no clinical signs of infection with BTB on the day of export; AND
2. the animal(s) were resident, since birth, at a facility that is free from infection with BTB, in a country or zone free from infection with BTB; OR
3. the animal(s) were resident, since birth, at a facility that is free from infection with BTB, and are tested with an MPI-approved test, within 30 days prior to export, with negative results; OR
4. the animal(s) have been isolated for at least 6 months prior to export, including protection from contact with any reservoir of BTB, and all isolated animals showed negative results to at least 2 consecutive MPI-approved tests carried out at a 6-month interval, with the second test performed within 30 days prior to export.

This option is similar to the recommendations of the OIE Code for the importation of bovids or cervids for breeding or rearing.

Semen:

1. The donor male(s) showed no clinical signs of infection with BTB on the day of semen collection; AND
2. the donor male(s) were resident, since birth, at a facility that is free from infection with BTB, in a country or zone free from infection with BTB; OR
3. the donor male(s) were resident, since birth, at a facility that is free from infection with BTB and were tested for BTB, within 30 days prior to semen collection, with an MPI-approved test, with negative results.

This option is similar to the recommendations of the OIE Code for the importation of semen of bovids.

Option 3

Animals:

1. For 12 months immediately before export, the animal has not resided on any premises where clinical, epidemiological or other evidence of BTB has occurred during the previous 5 years and the disease is compulsorily notifiable; AND
2. the animals for export were each subject to a test for BTB performed between 210 and 72 days immediately before export, with negative results.
    a. The test must be TST or CTST. The test must be read 72 hours post-inoculation, with negative results; OR
    b. the test must be performed on a blood sample taken during this period and tested using either a gamma interferon assay or a serological test approved by MPI, with negative results.

Semen:

1. For 12 months immediately before collection, the animals from which the semen for export was collected did not reside on any premises where clinical, epidemiological or other evidence of BTB has occurred during the previous 5 years and the disease is compulsorily notifiable; AND
2. the animals from which the semen for export was collected were each subject to a test for BTB performed between the 210 and 72 days immediately before semen collection.
    a. The test must be TST or CTST. The test must be read 72 hours post-inoculation, with negative results; OR
    b. the test must be performed on a blood sample taken during this period and tested using either a gamma interferon assay approved by the department or a serological test approved by the department, with negative results; OR
    c. at least 3 straws from each collection batch of semen from each donor animal(s) must be tested via PCR for BTB, with negative results.

Option 4

Animals:

1. For 12 months immediately before export, the animals for export have not resided on any premises where clinical, epidemiological or other evidence of BTB has occurred during the previous 3 years and the disease is compulsorily notifiable; AND
2. For 12 months immediately before export, the animals for export were part of a collection subject to a documented tuberculosis screening program. As part of the screening program:
    a. There must be diagnostic testing of all captive wild bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department (e.g. TST, CTST, approved gamma interferon, approved serological test).
    b. The collection must have been a 'closed herd' during that time.
    c. The collection must contain at least 4 captive wild bovids.
    d. Full post-mortem investigations were conducted on any dead ungulate species to determine the cause of death.

Semen:

1. For 12 months immediately before collection, the animals from which the semen for export was collected have not resided on any premises in the country of export where clinical, epidemiological or other evidence of BTB has occurred during the previous 3 years and the disease is compulsorily notifiable; AND
2. For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. As part of the screening program:
    a. There must be diagnostic testing of all captive wild bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department (e.g. TST, CTST, approved gamma interferon, approved serological test).
    b. The collection must have been a 'closed herd' during that time.
    c. The collection must contain at least 4 captive wild bovids.
    d. Full post-mortem investigations were conducted on any dead ungulate species to determine the cause of death.

OR

3. At least 3 straws from each collection batch of semen from each donor animal(s) were tested via PCR for BTB, with negative results.

23 Mycoplasmosis

23.1 Technical review

23.1.1 Aetiological agent

Family: *Mycoplasmataceae*
Genus: *Mycoplasma*
Species: *Mycoplasma agalactiae*, *Mycoplasma bovis*, *Mycoplasma capricolum* subsp. *capripneumoniae*, *Mycoplasma mycoides* subsp. *mycoides* SC

For the purposes of this IRA, only the above *Mycoplasmas* spp. have been identified as hazards in the preliminary hazard list, and only these species will be assessed.

Mycoplasma agalactiae (Ma), *Mycoplasma bovis*, *Mycoplasma capricolum* subsp. *capripneumoniae* (Mccp) and *Mycoplasma mycoides* subsp. *mycoides* SC (mmSC) are the causative agents of contagious agalactia, *M. bovis* infections, contagious caprine pleuropneumonia (CCPP) and contagious bovine pleuropneumonia (CBPP), respectively.

Mycoplasmas are wall-less bacteria (mollicutes).

23.1.2 OIE list

M. agalactiae
Contagious agalactia is an OIE-listed disease of sheep and goats.

M. bovis
Mycoplasma bovis infection is not an OIE-listed infection.

M. capricolum subsp. *capripneumoniae*
Contagious caprine pleuropneumonia is an OIE-listed disease of sheep and goats.

M. mycoides subsp. *mycoides* SC
Contagious bovine pleuropneumonia is an OIE-listed disease of cattle (OIE, 2020d).

Contagious bovine pleuropneumonia-affected countries are excluded from international trade of live animals (OIE Terrestrial Manual, 2018l).

23.1.3 New Zealand status

M. agalactiae
New Zealand is free from contagious agalactia (WAHIS, 2019d), as reported to the OIE.
Contagious agalactia is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016 primarily affecting sheep and goats.

M. bovis
Mycoplasma bovis was detected in the New Zealand cattle population for the first time in 2017. Currently, a national *M. bovis* eradication programme is in place (Biosecurity New Zealand, 2018a).
Mycoplasma bovis infection is notifiable under the Biosecurity (Notifiable Organisms) Order 2016, reprint as at 10 September 2018.

M. capricolum subsp. *capripneumoniae*
New Zealand is free from CCPP (WAHIS, 2019d), as reported to the OIE.
Contagious caprine pleuropneumonia, primarily affecting sheep and goats, is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

M. mycoides subsp. *mycoides* SC
New Zealand is free from CBPP (WAHIS, 2019d), as reported to the OIE. Contagious bovine pleuropneumonia was last reported in New Zealand in 1864.
Contagious bovine pleuropneumonia is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016 primarily affecting cattle.

23.1.4 Zoonotic potential

M. agalactiae
A review of the literature found no evidence that contagious agalactia is a zoonotic disease.

M. bovis
There are just 2 reported cases in the literature of *M. bovis* isolation in humans who were immunocompromised (Madoff et al., 1979; Pitcher & Nicholas, 2005).

M. capricolum subsp. *capripneumoniae*
A review of the literature found no evidence that contagious bovine pleuropneumonia is a zoonotic disease.

M. mycoides subsp. *mycoides* SC
A review of the literature found no evidence that contagious bovine pleuropneumonia is a zoonotic disease.

23.1.5 Epidemiology

Host range

M. agalactiae
Contagious agalactia is a serious disease of sheep and goats (OIE Terrestrial Manual, 2018k). There have been occasional reports of Ma in apparently healthy cattle (Catania et al., 2016; Pinho et al., 2009).

Mycoplasma agalactiae has been isolated from wild small ruminants such as ibexes, chamois (Tardy et al., 2012; Verbisck et al., 2010) and mountain goats (OIE Terrestrial Manual, 2018k).

Antibodies against Ma have been detected in deer (Cervidae) in Spain (Sumithra et al., 2013).

It is noteworthy that these wild ruminant species are not covered within the scope of this IRA.

There is no published evidence that species covered within the scope of this IRA play an epidemiological role in contagious agalactia.

M. bovis
Mycoplasma bovis is host-specific to cattle. However, there are rare reports of *M. bovis* infections in other domestic ruminants such as sheep and goats (Egwu et al., 2001; Kumar et al., 2012).

Mycoplasma bovis has also been detected infrequently in deer (Cervidae) (Dyer et al., 2004).

There is no published evidence that species covered within the scope of this IRA play an epidemiological role in *M. bovis* infections.

M. capricolum subsp. *capripneumoniae*
Contagious caprine pleuropneumonia mainly affects domestic and wild goats of the subfamily Caprinae. Other species of wild ruminants may also be affected (OIE Terrestrial Manual, 2018m).

In rare occasions, sheep that are in close contact with CCPP-infected goats may also contract the disease (Bölske et al., 1996).

Antibodies to Mccp have been detected in llamas and alpacas in the absence of isolation of the organism (OIE Terrestrial Manual, 2018m).

Contagious caprine pleuropneumonia has been reported in various wildlife ruminants (wild goats, Nubian ibexes, mouflons, gazelles and gerenuks) (Arif et al., 2007).

Mycoplasma capricolum subsp. *capripneumoniae* antibodies have been detected in wild ruminants (buffalo and impala) in Kenya (Paling et al., 1978).

Contagious caprine pleuropneumonia has been detected in wild Tibetan antelope in China, captive Arabian oryx and sand gazelle in UAE (Chaber et al., 2014; Lignereux et al., 2018; Yu et al., 2013).

There is still much uncertainty as to the role of wild ruminants in the epidemiology of CCPP. There is evidence that some wild and captive wild ruminant species are susceptible to infections, but it is unclear whether they act as reservoir hosts or merely dead-end hosts (More et al., 2017b).

M. mycoides subsp. *mycoides* SC
Mycoplasma mycoides subsp. *mycoides* SC only affect ruminants of the *Bos* genus (bovine, zebu cattle and yak).

Mycoplasma mycoides subsp. *mycoides* SC has been isolated in sheep and goats (OIE Terrestrial Manual, 2018l).

There are reports of CBPP infections in water buffaloes (OIE Terrestrial Manual, 2018l).

Wildebeest tested positive on CFT for MmmSC (Paling et al., 1978). Subsequent inoculation of live *Mycoplasma* subcutaneously into the wildebeest did not result in lesions (Shifrine et al., 1970).

Cape buffaloes have been experimentally infected with MmmSC. No clinical signs of disease were observed, but MmmSC could be re-isolated from animals (Shifrine et al., 1970).

Studies conducted in wildlife as part of international efforts to eradicate CBPP (caused by MmmSC) from domestic cattle indicate that wild ruminants are resistant and do not play an important epidemiological role in CBPP (More et al., 2017a; Sumithra et al., 2013; Williams & Barker, 2001).

Captive wild ruminants

M. agalactiae
There is no published evidence of contagious agalactia (caused by Ma) in captive wild animal collections.

M. bovis
There is no published evidence of *M. bovis* infections in captive wild animal collections.

M. capricolum subsp. *capripneumoniae*
There are a handful of cases of CCPP in captive wild ruminant collections that involve species within the scope of this IRA (Arif et al., 2007).

Contagious caprine pleuropneumonia has been detected in captive wild Arabian oryxes and sand gazelles in the UAE (Chaber et al., 2014; Lignereux et al., 2018).

Cases of CCPP have also been reported in captive wild ruminants (wild goats, Nubian ibexes, mouflons and gerenuks) in Qatar (Arif et al., 2007).

M. mycoides subsp. *mycoides* SC
There is no published evidence of CBPP in captive wild animal collections.

Geographical distribution

M. agalactiae
Contagious agalactia occurs in countries farming with domestic small ruminants such as areas in Europe, the Mediterranean regions, Asia, North Africa and sporadically the USA (OIE Terrestrial Manual, 2018k).

Contagious agalactia has been reported in Australia, the UK and the UAE (WAHIS, 2019d).

The disease has never occurred in Canada, Singapore or the RSA (WAHIS, 2019d).

M. bovis
Mycoplasma bovis was identified in cattle in the USA in 1961 and thereafter spread to many countries, achieving worldwide distribution (Nicholas & Ayling, 2003).

Countries affected by *M. bovis* include Australia, most countries in Europe, Asia and South America (Nicholas & Ayling, 2003).

M. capricolum subsp. *capripneumoniae*
Contagious caprine pleuropneumonia occurs in countries of Africa, Asia and the Middle East. Contagious caprine pleuropneumonia has recently been reported in the UAE (OIE Terrestrial Manual, 2018m; WAHIS, 2019d).

The disease has never occurred in Australia, the UK, the USA, Canada, Singapore, Japan or the RSA (WAHIS, 2019d).

M. mycoides subsp. *mycoides* SC
Contagious bovine pleuropneumonia is still problematic in many African countries.

Contagious bovine pleuropneumonia was last reported in Australia in 1967, in the UK in 1898, in the USA in 1892, in Canada in 1876, in the RSA in 1924 and in the UAE in 1990 (WAHIS, 2019d).

The disease has never occurred in Singapore (WAHIS, 2019d).

Pathogenesis

M. agalactiae
The incubation period varies from a few days to a few weeks. In some cases, it may extend up to 2 months depending on the route of entry, infectious dose, virulence of organisms and immune status of the animal.

Depending on the route of entry, the predilection site could be the mucosa of the respiratory tract, small intestine or alveoli of the mammary glands. Pyrexia accompanies initial infection. Following initial multiplication, organisms are disseminated via the circulation to other organ systems.

Inflammation of the connective tissue of the mammary glands leads to catarrhal or parenchymatous mastitis with eventual atrophy and agalactia.

A carrier status has been observed in contagious agalactia especially in females that may harbour the mycoplasmas in their genital tract (Kumar et al., 2014).

M. bovis
The incubation period in domestic ruminants ranges from 2 to 6 days.

The pathogenesis of genital mycoplasmosis is poorly defined. The respiratory tract is the predilection site for *M. bovis* (Irons et al., 2004).

Infection in the male and female genital tracts follow exposure to *M. bovis*. Lesions in the uterus oviducts, peritonitis, infertility, vesiculitis and epididymitis are associated with infection (Irons et al., 2004).

Latency is a feature of *M. bovis* infections (Pfützner & Sachse, 1996).

M. capricolum subsp. *capripneumoniae*
Contagious caprine pleuropneumonia is infectious to goats of all ages.

Mycoplasma capricolum subsp. *capripneumoniae* was known to be strictly host-specific to goats. However, reports have shown that sheep housed together with infected goats can be infected, as well as wild and captive wild ruminants (Arif et al., 2007; Bölske et al., 1996).

The incubation period reported during natural exposure was 10 days in domestic ruminants (Lefevre & Thiaucourt, 2004). In experimental studies, the incubation period ranged from approximately 26 to 41 days (MacOwan & Minette, 1977). The OIE refers to an incubation period of 45 days.

The pathogenesis of CCPP has not been studied in detail, and therefore, generalisations are made from other *Mycoplasma* infections.

The pathogenesis may involve inhalation, attachment, ciliostasis, the alteration and loss of cilia, multiplication and destruction of the mucosal epithelial cells, dissemination, inflammation and oxidative stress.

After entry into the respiratory system, mycoplasmas attach to the superficial cell layers. Colonisation and initiation of inflammation proceeds. This is characterized by ciliostasis of epithelia, serofibrinous pleuropneumonia, vasculitis and fibrinocellular exudation. Mycoplasmal antigens activate the hosts' immune system and stimulate the inflammatory and oxidative cascades. There is widespread serofibrinous inflammation and fluid exudation in the respiratory system organs (Iqbal Yatoo et al., 2019).

Mycoplasma capricolum subsp. *capripneumoniae* can persist in animals that recover from disease. These animals serve as chronic, latent carriers. This results in persistence of CCPP in animal populations.

M. mycoides subsp. *mycoides* SC
The incubation period under natural conditions can range from 3 weeks to 6 months.

The pathogenesis of CBPP is still unclear. A mycoplasmal antigen (galactan) may contribute to the severity of lesions by causing inflammation, having a thrombotic effect on capillaries and stimulating the development of connective tissue in the lungs around necrotic foci surrounded by granulocytes called “sequestra”. Thrombi develop in the lymphatic system causing coagulation of lymph (ter Laak, 1992).

The lungs of chronically infected animals may contain sequestra, resulting in these animals being subclinical (silent) carriers. They can be responsible for persistence of CBPP in herds (OIE Terrestrial Manual, 2018l; Thiaucourt et al., 2004).

Clinical signs

M. agalactiae

Clinical signs in domestic small ruminants are pyrexia and inappetence. Milk production is altered. Ewes may experience a decrease in milk production or failure to produce milk due to interstitial mastitis. Lameness and keratoconjunctivitis may develop in 5% to 10% of animals (Bergonier et al., 1997).

In acute cases, a fever may be accompanied by neurological signs. In enzootic areas, subacute to chronic disease is experienced. Pregnant animals may abort (OIE Terrestrial Manual, 2018k).

M. bovis

Mycoplasma bovis infections in domestic ruminants may result in acute to subacute inflammation of various tissues including the udder and joints (in calves and young cattle). In young and adult cattle, *M. bovis* can affect the respiratory and genital tracts (Pfützner & Sachse, 1996).

Signs of mastitis include altered milk production, spread of infection to other udder quarters, resistance to antibiotics and eventually agalactia.

When *M. bovis* mastitis is enzootic in herds, calves and young cattle often suffer from mycoplasmal arthritis and pneumonia. Affected animals fail to thrive.

Genital disease may only occur in a small percentage of animals. This, however, leads to fertility disorders (Pfützner & Sachse, 1996).

M. capricolum subsp. *capripneumoniae*

The disease is manifested in peracute, acute, subacute and chronic forms.

In the peracute form, animals die within 1 to 3 days in the absence of premonitory signs.

Acute and subacute disease is characterised by unilateral serofibrinous pleuropneumonia with severe pleural effusion.

Clinical disease is characterised by systemic serofibrinous deposition and inflammation of the lower respiratory tract, pleura and pleural cavity and may involve the heart, upper respiratory tract and other organs (Iqbal Yatoo et al., 2019).

Clinical signs in goats include pyrexia, anorexia, dyspnoea, polypnoea, coughing, nasal discharges and mortality rates up to 100% (Arif et al., 2007; Iqbal Yatoo et al., 2019; OIE Terrestrial Manual, 2018m).

A Thomson’s gazelle was experimentally infected with Mccp. The gazelle showed antibody evidence of exposure but no clinical signs of disease.

In the outbreak of CCPP in the UAE, captive wild sand gazelles suffered clinical disease and lesions that were similar to disease in goats (Lignereux et al., 2018).

M. mycoides subsp. *mycoides* SC

Clinical manifestation is characterised by hyperacute, acute, subacute and chronic forms.

In the acute form of disease, signs include pyrexia, anorexia, dyspnoea, polypnoea, coughing and nasal discharges.

In the chronic form, there is long-term persistence of the agent with waning clinical signs. These animals are thus difficult to identify (OIE Terrestrial Manual, 2018l).

Transmission

M. agalactiae

Transmission occurs via direct contact with infected animals through the oral, respiratory and mammary route. The main sources of infection include auricular, ocular and nasal secretions, faeces, milk, urine, semen and excretions from joint lesions (Kumar et al., 2014). Indirect transmission through fomites is also probable.

Vertical transmission via contaminated milk and colostrum has been reported (Kumar et al., 2014).

M. bovis

Transmission is via direct and indirect contact.

Infected animals shed *M. bovis* in milk and discharges of the respiratory and genital tracts. The volume of organisms that are shed increases at the onset of clinical disease.

Mycoplasmas enter through the open teat canals. Thus, important sources of infection are contaminated milking machines, milk cups, wash cloths, etc.

Animals also become infected via the respiratory tracts from droplet infection and contaminated dust particles.

Joints become infected through haematogenous spread.

The genital tract in bulls is colonised through ascending infections from the prepuce.

Transplacental transmission has also been reported (Pfützner & Sachse, 1996).

M. capricolum subsp. *capripneumoniae*

Transmission is via direct contact with infected animals. Inhalation of aerosol droplets is the most common route (Iqbal Yatoo et al., 2019). Airborne transmission over 50 to 80 metres has been suggested (Lignereux et al., 2018).

Indirect contact via contaminated objects, fomites or animal products is uncertain (Iqbal Yatoo et al., 2019).

Mycoplasma capricolum subsp. *capripneumoniae* is not very resistant to environmental factors. It may survive for 3 days in tropical areas and 2 weeks in temperate zones (Iqbal Yatoo et al., 2019).

In the experimental study involving the infection of a Thompson's gazelle with Mccp, the goat kept in the same enclosure as the gazelle did not become infected (Paling et al., 1978).

Domestic and wild goats that were reservoirs of Mccp were suggested as the source of the outbreaks in the wildlife Bovidae cases. However, sand gazelles were suggested as the source of infection for the Arabian oryx (Arif et al., 2007; Chaber et al., 2014; Lignereux et al., 2018).

M. mycoides subsp. *mycoides* SC

Transmission is via direct contact with infected or subclinical carrier animals through droplet infection.

Large volumes of organisms are present in bronchial secretions, nasal discharges and exhaled air. Airborne transmission may be probable over 20 metres or more.

Ingestion of contaminated feed or exposure of susceptible animals to contaminated environments or animal products have not been reported as routes of transmission (Thiaucourt et al., 2004).

Diagnosis

M. agalactiae

The OIE recommended testing methods include agent identification and detection of the immune response. A combination of agent identification methods on the same clinical sample are suggested to increase sensitivity.

Culture and identification of the organism, conventional and real-time PCR is recommended for individual animal freedom from infection and confirmation of clinical cases. Culture can also be used for eradication purposes (OIE Terrestrial Manual, 2018k).

ELISA is recommended for population freedom from infection, confirmation of clinical cases, surveillance and determining the immune status in individual animals or populations post-vaccination.

Complement fixation test and ELISA are also recommended for eradication purposes (OIE Terrestrial Manual, 2018k).

M. bovis
Presently, no tests for *M. bovis* are prescribed for international trade. Current detection methods include culture, molecular and serological detection (Wawegama & Browning, 2017).

Milk, joint fluid, bronchiolar lavages, swabs (from different anatomical sites), serum samples (Calcutt et al., 2018), semen or embryos (Bielanski et al., 2000) may be tested.

However, presently, information relating to the analytical and diagnostic performance of such tests is incomplete, and therefore, the validity may need to be established.

M. capricolum subsp. *capripneumoniae*
A presumptive diagnosis may be based on clinical and post-mortem examinations. This should be confirmed by laboratory diagnostics. Culture and isolation of Mccp often proves difficult, and therefore, molecular techniques are preferable.

The OIE recommended test method for the confirmation of clinical cases is PCR.

In relation to tests that detect an immune response, latex agglutination can be used for the confirmation of clinical cases. Competitive ELISA is recommended for use in surveillance, population freedom from infection and determining the immune status in individual animals or populations post-vaccination (OIE Terrestrial Manual, 2018m).

M. mycoides subsp. *mycoides* SC
Clinical diagnosis of CBPP is unreliable due to the numerous differential diagnoses for severe pneumonia. Pathological, microbiological, molecular or serological diagnostic methods can be used to confirm a diagnosis of CBPP. Pathological lesions in CBPP are pathognomonic and can therefore be used for disease surveillance at abattoirs.

The OIE recommended test for CBPP is in vitro culture and isolation (followed by species identification tests) to confirm clinical cases and determine if a population is free from infection. The complement fixation method is recommended for population freedom from infection, individual animal freedom from infection prior to movement, eradication and surveillance and may be used for the confirmation of clinical cases. Competitive-ELISA is the recommended test method for population freedom from infection, individual animal freedom from infection prior to movement, eradication and surveillance and the confirmation of clinical cases. Immunoblotting is recommended for surveillance (OIE Terrestrial Manual, 2018l).

Treatment, control and prevention

M. agalactiae
Local and systemic treatment with antibiotics following an antibiotic sensitivity test is the current therapy of choice.

Control in contagious agalactia affected countries includes hygienic farm management practises, continuous surveillance and monitoring. Early detection of infected animals and the identification of subclinical and chronic carriers is of major importance. The testing and slaughter method is recommended (Kumar et al., 2014).

In enzootic areas, vaccination is used to protect animals and prevent vertical transmission (Kumar et al., 2014). Vaccination is widely used in the Mediterranean countries of Europe and western Asia (OIE Terrestrial Manual, 2018k).

M. bovis
Mycoplasma bovis may be controlled through strict hygiene and sanitary measures on farms. Culling of all *M. bovis* mastitis cows or animals carrying the organism in the genital tract is another method of reducing the number of shedding animals in the herd.

Trade restrictions and testing of domestic animals and animal products prior to importation will assist in preventing entry of *M. bovis*.

M. capricolum subsp. *capripneumoniae*

Macrolides are the antibiotic of choice in the treatment of CCPP. Oxytetracyclines have also been effective. Other antibiotics have been explored; however, limiting factors include cost, availability, acting period, convenience of use and creation of a carrier state (Iqbal Yatoo et al., 2019).

Vaccination is useful in countries where there is a high prevalence of CCPP. Inactivated and adjuvanted vaccines are available for use. Immunity is likely to protect animals for 1 year (Iqbal Yatoo et al., 2019; OIE Terrestrial Manual, 2018m).

South Africa successfully eradicated CCPP by the slaughter and vaccination method. However, this method has not been evaluated (OIE Terrestrial Manual, 2018m).

In the outbreak of sand gazelles in the UAE, drastic reduction in gazelle density, mass vaccination and antibiotic therapy were used as a control strategy (Chaber et al., 2014).

In countries that are free from CCPP, trade restrictions, testing of animals and animal products prior to importation, strict biosecurity and quarantine measures will assist in preventing entry of Mccp.

M. mycoides subsp. *mycoides* SC
Treatment with corticosteroids and antibiotics are not advised, as it does not eliminate the disease from infected herds.

In most African countries, CBPP is controlled by vaccination. Various types of live vaccines have been used in domestic ruminants.

In European countries, vaccination is prohibited. The disease is controlled by testing, movement restrictions and slaughtering of infected cattle.

Semen

M. agalactiae
The presence of Ma has been reported in the semen of small domestic ruminants (Prats-van der Ham et al., 2016). However, transmission via AI is unlikely (Hare, 1985).

There is no published evidence demonstrating the presence of Ma in the semen of wildlife ruminants.

M. bovis
Haapala et al. (2018) reported that AI with *M. bovis*-contaminated semen was the source of the outbreaks in 2 closed dairy herds in Finland. Semen lots from the donor bull were confirmed positive for *M. bovis* following PCR analysis and culture. This is the first study to demonstrate that processed semen used in AI was the source of *M. bovis* infection on a farm (Haapala et al., 2018).

There is no published evidence demonstrating the presence of *M. bovis* in the semen of wildlife ruminants.

M. capricolum subsp. *capripneumoniae*
The presence of Mccp in the semen of small domestic ruminants is likely. However, transmission via AI is unlikely. There is no evidence demonstrating the presence of Mccp in the semen of domestic ruminants, but an assumption had been made as a result of the findings from CBPP (Hare, 1985).

There is no published evidence demonstrating the presence of Mccp in the semen of wildlife ruminants.

Only the semen of domestic and wild goats are risk commodities according to the OIE Code (OIE Terrestrial Code, 2019a).

M. mycoides subsp. *mycoides* SC
The presence of MmmSC has been reported in the semen of large domestic ruminants. However, transmission via AI is unlikely (Hare, 1985).

There is no published evidence demonstrating the presence of MmmSC in the semen of wildlife ruminants.

23.1.6 Hazard identification conclusion

Of the 4 *Mycoplasma* species that were assessed, only *Mycoplasma capricolum* subsp. *capripneumoniae* had demonstrable evidence of infections in wildlife Bovidae species covered within the scope of this IRA.

Mycoplasma capricolum subsp*. capripneumoniae* is identified as a hazard in captive wild Bovidae and not a hazard in captive wild Giraffidae, Tragulidae and their semen.

There is no evidence demonstrating the presence of Mccp in the semen of domestic ruminants, but an assumption had been made based on findings from CBPP. Thus, the OIE only considers the semen of domestic and wild goats a risk commodity. Since there is no scientific evidence of Mccp in the semen of domestic ruminants, an extrapolation to the semen of wildlife ruminants (within the scope of this IRA) cannot be made.

Mycoplasma capricolum subsp*. capripneumoniae* is not identified as a hazard in captive wild Bovidae semen and will not be assessed further*.*

There was no evidence to demonstrate that wildlife ruminants (species covered in this IRA) play an epidemiological role in contagious agalactia, *M. bovis* infections or CBPP.

Mycoplasma agalactiae, M. bovis and *M. mycoides* subsp. *mycoides* SC are not identified as hazards in captive wild Bovidae, Giraffidae, Tragulidae (within the scope of this IRA) or their semen and will not be assessed further*.*

23.2 Risk assessment

23.2.1 Entry assessment

Contagious caprine pleuropneumonia has never occurred in Australia, the UK, the USA, Canada, Singapore, Japan or the RSA. In terms of European countries, there is no known infected European country. However, Turkey has reported cases of CCPP and borders Bulgaria and Greece. Currently, the only approved country affected by CCPP is the UAE.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

Previously, Mccp has been reported as being host-specific to domestic and wild goats. However, recent reports indicate that various wildlife Bovidae are also susceptible to infection.

Contagious caprine pleuropneumonia has been reported in captive gerenuks in Qatar, a captive Arabian oryx and sand gazelles in the UAE and wild Tibetan antelopes in China. The suggested sources of infection for these cases were domestic or wild goats. However, it was suggested that the Arabian oryx was infected by the sand gazelles. In most cases, the infected animals died of CCPP, while others were treated with antibiotics and vaccinated with varying degrees of effectiveness.

The role that these wildlife Bovidae play in the epidemiology of CCPP is still unclear. It is uncertain whether they could act as reservoir hosts or whether they are merely dead-end hosts.

It is assumed that the pathogenesis in wildlife Bovidae is similar to that of domestic and wild goats. The incubation period may range from 10 days to 41 days. Animals that recover from Mccp infection may become latent carriers.

It is plausible that latently infected animals or those incubating the disease, not exhibiting signs of CCPP, could be passed as clinically sound for export.

Therefore, the likelihood of entry of Mccp via captive wild Bovidae (within the scope of this IRA) from CCPP-affected countries is assessed as very low.

23.2.2 Exposure assessment

Transmission is via direct contact with infected animals most commonly through inhalation of aerosol droplets. Aerosol transmission over 50 to 80 metres in captivity has been reported. Indirect transmission is uncertain as Mccp is not very resistant to environmental factors.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

There is variability regarding the probable exposure from imported captive wild Bovidae to susceptible species within the zoo. In all but 1 case of CCPP in captive wild Bovidae, the Mccp sources were suggested to be from domestic or wild goats. Only one case suggested transmission between captive wild Bovidae. Furthermore, in an experimental study, a single Thompson's gazelle that was infected failed to show clinical signs of disease or transmission to the goat it was kept in contact with.

Domestic and wild goats are known to be susceptible to CCPP. The range of wildlife Bovidae that are susceptible is undetermined. Therefore, it is assumed that all wildlife Bovidae in zoos could be susceptible.

Imported Mccp infected or incubating captive wild Bovidae are likely to succumb to clinical CCPP. Susceptible captive wild Bovidae within zoos may contract CCPP if they are in direct contact in the same enclosures with infected animals. Other susceptible animals (not in direct contact) may also become infected by aerosol transmission if enclosures are within 50 to 80 metres as described in previous cases studies. If domestic and wild goats are kept at zoos, they too could be susceptible to infection.

If captive wild Bovidae become infected, they are likely to die unless they are treated in early stages. Recovered animals may become carriers. However, this has not been proven in wildlife Bovidae species. Therefore, the likelihood of establishment in captive wild Bovidae is very low. If domestic or wild goats are kept at zoos, CCPP may establish in these animals.

Aerosol transmission has only been described over a distance of approximately 50 to 80 metres. It is unlikely that a small number of infected animals in the zoo would lead to an infective dose of Mccp that would result in aerosol infection of susceptible domestic or wild goats outside the zoo.

However, if domestic ruminants are released into New Zealand farms, they could transmit Mccp to other domestic ruminants. Contagious caprine pleuropneumonia could thus spread and establish in the domestic ruminant population.

Therefore, the likelihood of Mccp exposure and establishment within the zoo via infected captive wild Bovidae is assessed as very low, and the likelihood of exposure and establishment outside the zoo is assessed as very low.

23.2.3 Consequence assessment

Contagious caprine pleuropneumonia is an OIE-listed disease of sheep and goats.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences in terms of animal infection, disease or losses would affect the imported captive wild Bovidae and susceptible species at the zoos. If animals are imported during the incubation period, they may succumb to clinical disease and mortality after they arrive at the zoo. If these animals are treated in the early stages of the disease, they may recover. The extent of exposure within the zoo is uncertain, due to the variability in the susceptibility of captive wild Bovidae. The worst-case scenario would be that in-contact captive wild bovids and those in adjacent enclosures (if any) become infected and potentially die. Mortality of valuable animals is likely to impact negatively on the ASMP and conservation efforts of the zoo.

The likelihood of CCPP exposure and establishment in domestic goats, wild goats and sheep outside the zoo is assessed as very low and is only likely to happen if domestic ruminants are released from the zoo onto New Zealand farms. Animals affected may suffer from respiratory conditions, with mortality rates of up to 100% in goats.

The consequences for human health are negligible, as CCPP is not zoonotic.

The indirect consequences would relate to the costs for surveillance of in-contact susceptible animals or those in adjacent enclosures and treatment and/nursing of infected animals.

There may be additional costs to New Zealand for CCPP control in the event of an incursion in domestic ruminants.

Any trade impacts as a result of a CCPP incursion at the zoo are likely to be minimal if New Zealand can prove that the incursion was contained and there was no spread to animals outside the zoo. However, this may be difficult to prove if domestic animals are released from the zoo.

Therefore, the overall consequences as a result of a CCPP incursion are assessed as low.

23.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for CCPP is non-negligible, and it is assessed to be a risk in captive wild Bovidae.

Therefore, risk management measures can be justified.

23.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Contagious caprine pleuropneumonia is an OIE-listed disease of sheep and goats, has never occurred in New Zealand and is a notifiable disease.
- There is no evidence that CCPP is a zoonotic disease.
- Contagious caprine pleuropneumonia has been reported in domestic goats, wild goats, sheep and a small number of wildlife Bovidae species.
- The epidemiological role that wildlife Bovidae play in CCPP is unclear.
- The UAE is the only approved country with a known presence of CCPP in animals.
- The incubation period is set at 45 days by the OIE but can vary from 10 to 41 days.
- Latent and carrier status has been described in domestic goats.
- Transmission is via direct contact through aerosol droplets.
- Diagnosis includes culture and isolation, PCR, latex agglutination and C-ELISA.
- There is no evidence to demonstrate the presence of Mccp in the semen of wildlife ruminant species covered in this IRA or transmission via AI in these species.

23.3.1 Options

One or a combination of the following options may be used:

Mycoplasma agalactiae, M. bovis and *M. mycoides* subsp. *mycoides* SC are not identified as hazards in captive wild Bovidae, Giraffidae, Tragulidae or their semen, and therefore, risk management measures are not warranted.

Mycoplasma capricolum subsp*. capripneumoniae* is not identified as a hazard in semen or captive wild Giraffidae and Tragulidae, and therefore, risk management measures are not warranted.

Option 1

1. Country freedom for CCPP; AND
2. the animal(s) showed no clinical signs of CCPP on the day of export; AND
3. the animal(s) were resident in CCPP free countries since birth; OR
4. and the animal(s) were kept in a pre-export isolation for at least 45 days immediately prior to export.

This option is similar to OIE recommendations for the importation of wild goats from CCPP-free countries.

Option 2

1. The animal(s) showed no clinical signs of CCPP on the day of export; AND
2. the animal(s) were kept for at least 45 days immediately prior to export in a quarantine station where:
    a. no case of CCPP occurred during that period; AND
    b. the quarantine station was not situated in a CCPP-infected zone; AND
3. the animal(s) were not vaccinated for CCPP.

This option is similar to OIE recommendations for the importation of wild goats from countries infected with CCPP.

24 Haemorrhagic septicaemia

24.1 Technical review

24.1.1 Aetiological agent

Family: *Pasteurellaceae*
Genus: *Pasteurella*
Species: *Pasteurella multocida*

Pasteurella multocida (P. multocida) is the causative agent of haemorrhagic septicaemia (HS).

Serotypes B:2(6:B) and E:2(6:E) are the main serotypes that cause HS and are the only serotypes that will be assessed.

24.1.2 OIE list

Haemorrhagic septicaemia is an OIE-listed disease of cattle (OIE, 2020d).

24.1.3 New Zealand status

New Zealand is free from HS (*P. multocida* B:2 and E:2) (WAHIS, 2019d), as reported to the OIE. However, *P. multocida* serotypes A, B, and D are present in New Zealand (Jones, 1988).

Haemorrhagic septicaemia (*P. multocida* B:2 and E:2) is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016 primarily affecting cattle.

24.1.4 Zoonotic potential

Pasteurella multocida (B:2 and E:2) is not known to be zoonotic (OIE Terrestrial Manual, 2018q).

There are no reports of infection in humans with the *P. multocida* B:2 or E:2 serotypes. However, humans are susceptible to infection with other serotypes (Arons et al., 1982).

24.1.5 Epidemiology

Host range

Haemorrhagic septicaemia is a disease of concern in cattle and water buffaloes (Bastianello & Henton, 2004; OIE Terrestrial Manual, 2018q).

The disease has, however, been reported in domestic ruminants (sheep and goats) and other domestic animals (pigs, horses, donkeys and camels).

It is suggested that wildlife ruminants (oryxes) could be reservoirs of *P. multocida* (B:2 and E:2) (Bastianello & Henton, 2004; Voigts et al., 1997).

An outbreak of HS (serotype B) was reported in free-ranging saiga antelopes in Kazakhstan (Jones, 2018).

Captive wild elands and wildebeest were involved in an HS outbreak at a Nigerian zoo (Okoh, 1980).

Reported infections in wildlife ruminants include fallow deer, elks (Cervidae) and American bison (Carrigan et al., 1991; Heddleston & Wessmann, 1973; Malhi et al., 2016). These animals, along with buffalo and domestic ruminants, are not covered within the scope of this IRA.

There is no published evidence of *P. multocida* (B:2 and E:2) in species of the Giraffidae or Tragulidae families. However, due to the wide host range, it is assumed that these species may be susceptible.

Captive wild ruminants

Reports of HS in captive wild ruminants are scarce.

An outbreak of HS (serotype not specified) in a zoo in Nigeria led to the death of 6 species of captive wild animals. Captive wild Bovidae (elands and wildebeest) were included in this outbreak (Okoh, 1980).

Geographical distribution

The disease has been reported in Asia, Africa, Europe and the Middle East (OIE Terrestrial Manual, 2018q).

According to WAHIS, HS has never occurred in Australia, the UK, Japan or Canada. Haemorrhagic septicaemia was last reported in Singapore in 1930 and in the USA in 1969 (WAHIS, 2019d).

Pathogenesis

In natural settings, the bacteria are likely to enter the host via the oral or nasal route, initially multiplying in the tonsils.

Bacteraemia develops within 12 hours of experimental infection. The incubation period is short (30 hours) in experimental infections and 2 to 3 days in natural infections.

Effects of infection are dependent on virulence, infectious dose and host susceptibility. Once the hosts' immune system is overwhelmed, septicaemia and clinical disease manifest (Bastianello & Henton, 2004).

As a result, animals may succumb to disease or develop arrested infection. Animals with arrested infections are latent carriers. Animals could be active carriers that shed bacteria or latent carriers that do not. These animals can serve as constant sources of infection (de Alwis, 1992).

In an experimental study of young buffaloes, a carrier status was demonstrated in the nasopharynx 215 days post exposure and in tonsils 229 days post exposure (de Alwis, 1999).

The extent of an outbreak is dependent on the animal population immunity. In areas where HS is enzootic, adults in the herd are normally immune; therefore, disease affects the young with waning immunity. In naïve populations, high morbidity and mortality rates are expected.

Animals that recover from disease develop long-lasting immunity (Bastianello & Henton, 2004).

Clinical signs

The clinical course of disease may be peracute, acute or subacute.

Peracute disease is characterised by sudden death. Dyspnoea or grunting and prostration may be noticeable 24 hours prior to death (Bastianello & Henton, 2004).

Animals affected peracutely or subacutely exhibit pyrexia, anorexia, depression, hypersalivation and nasal discharge.

In subacute cases, animals may develop subcutaneous oedematous swellings on the ventral aspect of the body and show circling movements and incoordination (Bastianello & Henton, 2004; OIE Terrestrial Manual, 2018q).

Water buffaloes are more susceptible to HS than cattle and exhibit more severe clinical signs.

Deaths in wild or captive wild ruminants have been triggered by stressful conditions such as inclement weather, food shortages, handling or translocation.

In outbreaks, wild and captive wild ruminants have died without premonitory signs (Jones, 2018; Voigts et al., 1997).

Transmission
Transmission occurs directly via contact between susceptible and infected animals or indirectly via fomites and aerosols. *Pasteurella multocida* is excreted in respiratory aerosols, saliva, urine, faeces and milk (Bastianello & Henton, 2004).

Diagnosis
A presumptive diagnosis could be based on clinical signs, gross lesions, morbidity and mortality patterns.

Confirmatory diagnosis requires isolation and characterisation of the pathogen.

Isolation and identification of the organism can be done by culture and biochemical methods, serotyping methods (rapid slide agglutination test, indirect haemagglutination test, AGID, counterimmunoelectrophoresis, agglutination test) and molecular methods (PCR, genotype differentiation).

Serological tests are available but are not usually used for diagnosis (OIE Terrestrial Manual, 2018q).

Treatment, control and prevention
Death usually ensues in 100% of clinical cases if treatment is not implemented (Bastianello & Henton, 2004; OIE Terrestrial Manual, 2018q).

Often the acute nature of the disease limits the effective use of treatment. In outbreak scenarios, antibiotic treatment can be administered to all in-contact and susceptible animals that exhibit a febrile reaction (de Alwis, 1992) as a preventive measure.

In countries where the disease is enzootic, vaccination is the principal means of control. For vaccines to be effective, they must contain the local serotypes. However, some serotypes may have a certain degree of cross-protection (de Alwis, 1992).

Semen
There is no published evidence demonstrating the presence of *P. multocida* (B:2 and E:2) in the semen of domestic or wildlife ruminants or the transmission of the agent to naïve dams if inseminated with *P. multocida* (B:2 and E:2)-contaminated semen.

24.1.6 Hazard identification conclusion
Haemorrhagic septicaemia is an OIE-listed disease of cattle.

There is no published evidence demonstrating the presence of *P. multocida* (B:2 and E:2) in the semen of wildlife ruminants.

Pasteurella multocida (B:2 and E:2) is not identified as a hazard in the semen of captive wild Bovidae, Giraffidae or Tragulidae and will not be assessed further.

Pasteurella multocida (B:2 and E:2) is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

24.2 Risk assessment

24.2.1 Entry assessment
Australia, Japan, the UK and Canada are free of HS (*P. multocida* (B:2 and E:2)), and HS was last reported in Singapore in 1930 and in the USA in 1969. Disease is present in all other approved countries.
There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Therefore, the likelihood that an imported animal will be infected in terms of volume of trade is assessed as very low.

Haemorrhagic septicaemia naturally causes disease in cattle and water buffaloes. However, disease has been reported in a variety of domestic and wild animals. There is a paucity of reports of the disease in captive collections, implying that disease in captive wild ruminants may not be a common occurrence.

The incubation period in animals is short and may range from 1 to 3 days. Once infected, animals may either succumb to disease or become latent or active carriers. From the cases described, the former is more likely, as animals that are carriers have been reported to succumb to disease shortly after or during stressful conditions. Therefore, the stress of translocation is likely to result in clinical manifestations, which would be detected before animals are imported. However, should these animals endure the stress and remain carriers, it is plausible that animals not exhibiting signs of HS could be passed as clinically sound for export.

Therefore, the likelihood of entry of *P. multocida* (B:2 and E:2) via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from HS-affected countries is assessed as very low.

24.2.2 Exposure assessment

Transmission occurs directly via contact between susceptible and infected animals or indirectly via fomites and aerosols. *Pasteurella multocida* is excreted in respiratory aerosols, saliva, urine, faeces and milk.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If active or latent carrier animals are imported, active carriers could be a source of infection to other in contact susceptible animals. As mentioned, a variety of domestic and wildlife ruminants are susceptible to *P. multocida* (B:2 and E:2).

Susceptible captive wild animals within zoos are likely to contract HS if they are in direct contact in the same enclosures with active carriers. Other susceptible animals (not in direct contact) may also become infected via fomites if the same zookeepers manage the enclosures or the same equipment is used.

If zoos keep domestic ruminants such as cattle or water buffaloes, the disease could establish in these animals and they would become a continuous source of infection to other susceptible domestic and captive wild animals.

Disease progression in further infected animals would be dependent on a number of factors. These animals could either succumb to disease or go on to become latent or active carriers. From the pathogenesis described, the introduction of HS into naïve animal populations is likely to result in high mortality and morbidity, but there is much uncertainty surrounding this in wildlife.

If animals succumb to disease and mortality or morbidity, cases are likely to be investigated swiftly, thereby limiting the exposure to other susceptible animals. However, if they become carriers, HS could establish in the captive wild animal population.

Pasteurella multocida is excreted in respiratory aerosols, but there are no reported cases of transmission over long distances (i.e. outside the zoo).

However, if domestic ruminants are kept at the zoo, they could become infected with *P. multocida* (B:2 and E:2). Should these domestic ruminants be released onto New Zealand farms, they could transmit *P. multocida* (B:2 and E:2) to other domestic ruminants. Various *P. multocida* serotypes have become established in New Zealand, and therefore, *P. multocida* (B:2 and E:2) could also spread and establish in the domestic ruminant population.

Pasteurella multocida (B:2 and E:2) is not zoonotic, and therefore, there would be no adverse human health effects.

The likelihood of *P. multocida* (B:2 and E:2) exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate, and the likelihood of exposure and establishment outside the zoo is assessed as very low.

24.2.3 Consequence assessment

Haemorrhagic septicaemia is an OIE-listed disease of cattle.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences of HS would be due to infected imported animals that may succumb to clinical disease. Mortality and morbidity is more likely to occur in naïve animal populations, such as those in New Zealand. There would be a loss of valuable animals. This would negatively impact the breeding programmes in zoos.

Since HS has the ability to produce latent and active carriers, the disease could establish in the animal population within the zoo. Establishment in captive wild ruminants is uncertain. However, there are cases that have suggested that *P. multocida* has been present in wild ruminant populations and have only resulted in clinical outbreaks after stressful conditions. Establishment in the captive wild animals and domestic animals (if any) within the zoo could lead to a continuous source of infection to other animals within the zoo.

The likelihood of HS exposure and establishment in domestic and wild animals outside the zoo is assessed as very low and is only likely to happen if infected domestic ruminants are released from the zoo onto New Zealand farms. *Pasteurella multocida (B:2 and E:2)* could be transmitted to cattle, sheep, goats, deer and other domestic animals (pigs, horses, donkeys). In naïve populations, high morbidity and mortality rates are expected.

Pasteurella multocida (B:2 and E:2) is not zoonotic, and therefore, the consequences for human health are negligible.

Indirect consequences would entail the costs for control and surveillance within the affected zoos.

There may be additional costs to New Zealand for HS control in the event of an incursion in domestic ruminants.

Any trade impacts as a result of an HS incursion at the zoo are likely to be minimal if New Zealand can prove that the incursion was contained and there was no spread to animals outside the zoo. However, this may be difficult to prove if domestic animals are released from the zoo.

Therefore, the overall consequences as a result of a HS incursion are assessed as low.

24.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for HS is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

24.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Haemorrhagic septicaemia is an OIE-listed disease of cattle.
- Haemorrhagic septicaemia (*P. multocida* B:2 and E:2) has never occurred in New Zealand.
- *Pasteurella multocida* (B:2 and E:2) is not known to cause zoonoses.
- Haemorrhagic septicaemia is naturally a disease of concern in cattle and water buffaloes, but has been reported in other domestic and wildlife ruminants.
- Haemorrhagic septicaemia has never occurred in Australia, the UK or .Canada and was last reported in Singapore in 1930 and in the USA in 1969.
- The incubation period is 1 to 3 days.
- Latent and active carriers exist.
- Transmission occurs directly via contact between susceptible and infected animals or indirectly via fomites and aerosols.
- Diagnosis includes culture and biochemical methods, serotyping methods and molecular methods.
- There is no published evidence demonstrating the presence of *P. multocida* (B:2 and E:2) in the semen of domestic or captive wild Bovidae.

24.3.1 Options

One or a combination of the following options may be used:

Pasteurella multocida (B:2 and E:2) is not identified as a hazard in semen, and therefore, risk management measures are not warranted.

Option 1

1. Country freedom for HS (*Pasteurella multocida* B:2 and E:2); AND
2. the animal(s) were resident in HS (*Pasteurella multocida* B:2 and E:2)-free countries since birth or for the previous 180 days immediately prior to export; AND
3. the animal(s) showed no clinical signs of HS on the day of export.

This option is similar to recommendations of the OIE code for the importation of cattle and buffaloes from HS-free countries or zones.

Option 2

1. Country freedom for HS (*Pasteurella multocida* B:2 and E:2); AND
2. the animal(s) were resident in HS (*Pasteurella multocida* B:2 and E:2)-free countries since birth or for at least 12 months immediately prior to export; AND
3. the animal(s) showed no clinical signs of HS on the day of export.

This option takes into consideration extended carrier periods.

Option 3

1. For 12 months immediately prior to export, the animal(s) were part of a captive wild animal collection where no clinical, epidemiological or other evidence of HS (*Pasteurella multocida* B:2 and E:2) occurred during the previous 12 months; AND
2. the animal was vaccinated against HS (*Pasteurella multocida* B:2 and E:2) with an MPI approved vaccine.

This option will ensure the facility of origin had no cases of HS in the last 12 months, and animals from an HS-affected country would be protected via vaccination.

25 Bovine anaplasmosis

25.1 Technical review

25.1.1 Aetiological agent

Family: *Anaplasmataceae*
Genus: *Anaplasma*
Species: *Anaplasma marginale, A. centrale*, *A. caudatum*

Anaplasma centrale was formerly referred to as *Anaplasma marginale* subsp. *centrale* (Hove et al., 2018) but will remain *A. centrale* for the purposes of this IRA.

Anaplasma spp. are intracellular rickettsial parasites.

Anaplasma caudatum has been regarded as appendages associated with the *Anaplasma* body in certain isolates, and there is uncertainty regarding its validity as a species responsible for causing disease in animals (Kreier & Ristic, 1963).

Therefore *A. caudatum* will not be assessed, and for the purposes of this IRA, *A. marginale* and *A. centrale* are the causative agents of bovine anaplasmosis.

25.1.2 OIE list

Bovine anaplasmosis is an OIE-listed disease of cattle (OIE, 2020d).

25.1.3 New Zealand status

New Zealand is free from bovine anaplasmosis (WAHIS, 2019d), as reported to the OIE.

Anaplasmosis (*Anaplasma spp.*) is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

25.1.4 Zoonotic potential

Bovine anaplasmosis is not known to be a zoonotic disease (OIE Terrestrial Manual, 2018d).

25.1.5 Epidemiology

Host range

Cattle are the primary hosts of bovine anaplasmosis. Cattle of all ages are susceptible to infection, but calves are more resistant (Aubry & Geale, 2011).

Anaplasma marginale has also been recovered from sheep and goats (Kuttler, 1984).

Isolates of *A. marginale* have been detected in wildlife ruminants such as water buffaloes, bison, African antelopes (Bovidae) and deer (Cervidae) (Aubry & Geale, 2011; Brandt, 2009; Kocan et al., 2010; Kuttler, 1984; Pfitzer et al., 2011; Woldehiwet, 2010).

Anaplasma centrale has been identified in wildebeest, elands, buffaloes and waterbucks (Khumalo et al., 2016).

Unidentified species of *Anaplasma* have been isolated from giraffes (Giraffidae) (Aubry & Geale, 2011).

There is no published evidence of *Anaplasma* in species of the Tragulidae family.

Captive wild ruminants

There is no published evidence of bovine anaplasmosis in captive wild animals or of bovine anaplasmosis outbreaks in zoos.

Geographical distribution
Bovine anaplasmosis has worldwide distribution (Potgieter & Stoltsz, 2004) in tropical and subtropical regions, including South and Central America, the USA, Europe, Africa, Asia and Australia (Aubry & Geale, 2011).

In Australia, the disease is restricted to parts of the country (WAHIS, 2019a) where the vector, *Rhipicephalus* (formerly *Boophilus*) *microplus*, is present (Bock et al., 2006). In other regions of Australia, which are tick-free areas, bovine anaplasmosis is a notifiable disease (Animal Health Australia, 2018; Bock et al., 2006).

In the USA, bovine anaplasmosis is enzootic in almost every state except Hawaii (Aubry & Geale, 2011; McCallon, 1976).

The disease is enzootic in the RSA and is a cause of severe economic loss in the cattle industry (Mtshali et al., 2007).

According to the WAHIS interface, bovine anaplasmosis has never occurred in Singapore or the UK (WAHIS, 2019d).

Pathogenesis
Bovine anaplasmosis is an arthropod-borne haemolytic disease caused mainly by *A. marginale*.

Anaplasma centrale generally produces mild disease (Potgieter & Stoltsz, 2004).

The incubation period varies from 7 to 60 days, depending on the infectious dose, with an average of 28 days (Kocan et al., 2010). In some species, the incubation period may extend to 100 days (Brandt, 2009; Potgieter & Stoltsz, 2004).

After the initial infection and incubation period, the organism invades erythrocytes and undergoes replication. Erythrocytes are the only site of infection for *Anaplasma* spp. Phagocytosis of the infected erythrocytes results in anaemia and icterus. During acute infection, 70% or more erythrocytes will be affected (Kocan et al., 2010).

Once infected, domestic ruminants remain persistently infected or carriers (latently infected) for life (Aubry & Geale, 2011). Animals that recover from bovine anaplasmosis also become lifelong carriers of the organism (Potgieter & Stoltsz, 2004). Carrier animals have lifelong immunity and are resistant to clinical disease on challenge exposure (Kocan et al., 2003).

Wildlife ruminants may also become persistently infected and remain carriers for extended periods (Aubry & Geale, 2011; Kuttler, 1984).

A prevalence study of wildlife ruminants in the RSA demonstrated that *A. centrale* was the most prevalent *Anaplasma* in these animals. This suggests that wildlife ruminants are reservoirs for *A. centrale* (Khumalo et al., 2016).

Clinical signs
Bovine anaplasmosis is generally characterised by fever, progressive anaemia and icterus (Potgieter & Stoltsz, 2004). Pyrexia may occur in early stages of infection and fever over 40°C persists throughout rickettsial circulation (Kocan et al., 2010).

Domestic ruminants (cattle) of all ages can be affected, but the severity of the disease is age-dependent. Calves are less susceptible to the disease, but cattle over 2 years can experience acute and fatal infection (Aubry & Geale, 2011).

Peracute infections with high mortality rates have been reported and are the fatal form of the disease (Kocan et al., 2010; Potgieter & Stoltsz, 2004).

The acute phase of the disease may often result in death and is characterised by signs such as weight loss, fever, abortion and drop in milk production (Kocan et al., 2004). Mortality rates in acute infection vary from 29% to 49% in domestic ruminants (Aubry & Geale, 2011).

Clinical disease in wildlife ruminants is rare.

Even though deer are highly susceptible to infection with *A. marginale*, clinical signs are minimal (Kuttler, 1984).

Various African antelopes have been reported with either serological evidence, or isolation of *Anaplasma*, in the absence of clinical disease. There were reported cases of animals being ill in the presence of *Anaplasma* or *Anaplasma*-like organisms. However, these cases were never investigated or followed up to confirm infection with *A. marginale*.

Apart from 2 reports of acute disease in giraffes suggested to be caused by *A. marginale*, all other reports of infections in wildlife ruminants were clinically inapparent (Aubry & Geale, 2011).

Transmission

Bovine anaplasmosis is an arthropod-borne disease transmitted by approximately 20 different species of ticks worldwide (Aubry & Geale, 2011; Kocan et al., 2004). The most efficient method of transmission is biologically via tick vectors.

Mechanical and transplacental methods may also occur (Aubry & Geale, 2011).

Rhipicephalus microplus is the primary arthropod vector involved in biological transmission in Australia (Bock et al., 2006). In the USA, *Dermacentor* spp. of ticks are the primary vectors (Aubry & Geale, 2011; McCallon, 1976). In the RSA, ticks capable of transmitting *A. marginale* include *R. decoloratus, R. microplus, R. simus, R. e. evertsi* and *Hyalomma m. rufipes* (Potgieter & Stoltsz, 2004).

None of these tick species are present in New Zealand. *Haemaphysalis longicornis* (cattle tick) is present in New Zealand. However, experimental studies attempting transmission of *Anaplasma* spp. by *H. longicornis* have failed (Connell, 1978; Heath, 2002).

Mechanical transmission can occur through blood-contaminated needles, dehorning equipment, ear-tagging devices and nose tongs (Aubry & Geale, 2011; Kocan et al., 2010).

Biting flies, including stable flies (genus *Stomoxys*) and horse flies (genus *Tabanus*), also have the potential to mechanically transmit the disease (Aubry & Geale, 2011; Kocan et al., 2004; McCallon, 1976). These fly genera are present in New Zealand. However, it is suggested that for transmission via this method to be successful, there must be minimal time lapse between feeding off an infected animal and a susceptible animal. This time lapse should be a few minutes (Roberts & Love, 1977). This may be due to the fact that the *Anaplasma* do not multiply in these vectors.

The deduction from various transmission studies is that some *Anaplasma* are host-specific. Strains found in wildlife ruminants may be less pathogenic than those found in domestic ruminants. Therefore, they are not reported to cause clinical disease when inoculated into domestic ruminants (Kuttler, 1984).

Authors Aubry & Geale (2011) stated that “while there are many experimental (mostly blood transfusion) studies, there are no substantiating field studies that demonstrate the transmission of *A. marginale* between cattle and wild ruminants or that wildlife can be an infection reservoir for cattle”.

However, in contradiction to the above statement, Pfitzer et al. (2011) demonstrated the presence of *A. marginale* in nyala from various farms in Pongola, the RSA. These farms were either previously occupied by cattle or shared a common boundary with cattle-grazing areas.

In another study by Ngeranwa et al. (2008), *A. marginale* was isolated from elands in Kenya and the RSA.

Pfitzer et al. (2011) and Ngeranwa et al. (2008) thus concluded that wildlife ruminants play a significant role in the epidemiology of *Anaplasma* organisms and that wildlife could serve as reservoirs for domestic ruminants.

Diagnosis

Clinical diagnosis of bovine anaplasmosis can be made based on clinical signs and necropsy findings of infected animals.

Confirmation of clinical diagnosis may be achieved through direct microscopic evaluation of stained blood smears, or antigen detection and demonstration of an immune response using serological and molecular methods.

Light microscopy of blood and organ smears from liver, kidneys, heart and lungs can be used to confirm the presence of organisms in erythrocytes (Kocan et al., 2010; OIE Terrestrial Manual, 2018d). This method is not reliable in low levels of circulating rickettsia seen in latent infections (OIE Terrestrial Manual, 2018d).

Polymerase chain reaction can detect a lower level of infection (Aubry & Geale, 2011). It is the OIE recommended test for identifying individual animal infection status and the confirmation of clinical cases (OIE Terrestrial Manual, 2018d).

Serological tests used to detect antibodies to *Anaplasma* spp. include the indirect fluorescent antibody test (IFAT), ELISA, card agglutination test (CAT) and the complement fixation test (CFT).
ELISA is the OIE-recommended test for identifying population freedom from infection, for eradication and surveillance, and for determining immune status post-vaccination (OIE Terrestrial Manual, 2018d).

There is a lack of validation of diagnostic tests for wildlife. Given this and the serological cross-reactivity of *Anaplasma* spp., diagnosis of bovine anaplasmosis in wildlife ruminants may prove challenging.

Treatment, control and prevention

Treatment for bovine anaplasmosis involves the administration of tetracycline drugs, imidocarb and the use of various chemotherapeutic agents (arsenicals, antimalarials, antimony derivatives and dyes).

Control measures include arthropod control, prevention of mechanical transmission, administration of antibiotics as a prophylactic measure and vaccination.

Vector control measures include the use of acaricides, modified housing of animals, and pest management of flies.

Vaccination is an economical and effective means of bovine anaplasmosis control. Live and killed (inactivated) vaccines are available. Attenuated strains of *A. marginale* or *A. centrale* are used in the production of live vaccines (Aubry & Geale, 2011; OIE Terrestrial Manual, 2018d).

In countries that are free from bovine anaplasmosis, preventing the entry of infected or carrier animals is the key preventative strategy.

Semen

There is no published evidence demonstrating the presence of *A. marginale* or *A. centrale* in semen of domestic or wildlife ruminants, or the transmission of the agents to naïve dams if inseminated with *A. marginale*- or *A. centrale*-contaminated semen.

In an experimental study, bulls were inoculated with blood from known *A. marginale* carrier animals. The bulls became infected but there was no evidence of *A. marginale* in their semen (Swift et al., 1979).

"The risk of *A. marginale* transmission in frozen semen is considered to be negligible" (Thompson & Goodrich, 2018).

25.1.6 Hazard identification conclusion

Bovine anaplasmosis is an OIE-listed disease of cattle.

There is no published evidence demonstrating the presence of *A. marginale* or *A. centrale* in the semen of wildlife ruminants.

Anaplasma marginale and *A. centrale* are not identified as hazards in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Unidentified species of *Anaplasma* have been isolated from giraffes (Giraffidae) (Aubry & Geale, 2011). As mentioned, anaplasmas are host-specific, and therefore, this isolate may be a strain specific to giraffes and likely to be apathogenic.

There is no published evidence of *Anaplasma* in species of the Tragulidae family.

Anaplasma marginale and *A. centrale* are not identified as hazards in captive wild Giraffidae and Tragulidae.

Anaplasma marginale and *A. centrale* are identified as hazards in captive wild Bovidae.

25.2 Risk assessment

25.2.1 Entry assessment

According to the WAHIS interface, bovine anaplasmosis has never occurred in Singapore or the UK. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild Bovidae imported into New Zealand, and these imports are also likely to be infrequent. Due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Bovine anaplasmosis causes clinical disease in domestic ruminants. However, in wildlife ruminants, clinical disease is a rare occurrence.

The incubation period for bovine anaplasmosis is variable and may range from 7 to 100 days. In domestic ruminants, animals that become infected or recover from infection remain persistently infected (carriers) for life. Wildlife ruminants have reported to become persistently infected as well. If these animals are carriers, it is plausible that animals not exhibiting clinical signs of bovine anaplasmosis could be passed as clinically sound for export.

Bovine anaplasmosis does occur in domestic and wild Bovidae in a number of approved countries. However, there were no published reports of bovine anaplasmosis in captive wild animals or of bovine anaplasmosis in zoos.

Wildlife ruminants have been reported with serological evidence of *Anaplasma* exposure, or isolation of *Anaplasma*, in the absence of clinical disease. This may account for the lack of reporting of clinical cases of *Anaplasma* in captive ruminant collections.

Therefore, the likelihood of entry of *A. marginale* and *A. centrale* via captive wild Bovidae (within the scope of this IRA) from bovine anaplasmosis-affected countries is assessed as very low.

25.2.2 Exposure assessment

Bovine anaplasmosis is an arthropod-borne disease transmitted by approximately 20 different species of ticks. Tick transmission is the most efficient and common route. Mechanical and transplacental methods may also occur.

Ticks present in New Zealand have not been documented as competent vectors of *A. marginale* and *A. centrale.* Certain genera of flies present in New Zealand have been reported as mechanical vectors of *A. marginale* and *A. centrale.* However, transmission would have to occur within a few minutes to be successful, and therefore, the likelihood of transmission via this route is extremely low.

The small number and occasional importation of probably infected live captive wild Bovidae implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If carrier captive wild Bovidae are imported into New Zealand zoos, they could be a source of infection to other susceptible captive wild ruminants. Bovine anaplasmosis is not transmitted by direct contact. Therefore, the likelihood of exposure to in-contact ruminants would be lower in comparison to a contagious disease.

If imported ruminants carry exotic tick species that are competent vectors, they could transmit *A. marginale* and *A. centrale* to susceptible animals within the zoo. If they do not carry any ticks, flies in New Zealand could potentially transmit the *Anaplasma* to susceptible captive wild and domestic ruminants within the zoo.

Various authors have suggested that some strains of *Anaplasma* in wildlife ruminants are host-specific and are likely to be less pathogenic to domestic ruminants. There is uncertainty regarding the role of wildlife ruminants as reservoirs for infection of domestic ruminants.

Clinical disease has not been reported in wildlife ruminants. If infected, they could, however, become carriers of *Anaplasma* in the absence of clinical disease. As a result, bovine anaplasmosis could silently spread via vectors and establish in the captive wild and potentially domestic ruminant populations within zoos.

It is noteworthy, that subclinically or latently infected animals may experience low levels of circulating rickettsia. It is uncertain whether low levels of *Anaplasma* spp. in these carrier animals would constitute an infectious dose sufficient to infect arthropods vectors such as flies.

Since the ticks in New Zealand are not known to be competent vectors of bovine anaplasmosis and transmission by other arthropod vectors (such as flies) has to occur within a few minutes, the likelihood that these flies would infect susceptible domestic or wild ruminants outside the zoo is assessed as negligible.

However, if domestic ruminants kept at the zoo become infected and then released to New Zealand farms, they may act as a source of infection to other domestic ruminants. The likelihood of this occurring is assessed as very low, but bovine anaplasmosis could establish in the domestic ruminant population.

Anaplasma marginale and *A. centrale* are not zoonotic, and therefore, exposure to humans would be a negligible risk.

Therefore, the likelihood of *A. marginale* and *A. centrale* exposure and establishment within the zoo via infected captive wild Bovidae is assessed as very low, the likelihood of exposure and establishment outside the zoo is assessed as very low.

25.2.3 Consequence assessment

Bovine anaplasmosis is an OIE-listed disease of cattle.

There is likely to be a very small number of live captive wild Bovidae imported into New Zealand. These imports are also likely to be infrequent.

Anaplasma marginale and *A. centrale* are not known to cause clinical disease in wildlife ruminants. *Anaplasma* of wildlife ruminants may be host-specific and less pathogenic to domestic ruminants. If infected, wildlife ruminants are likely to become carriers in the absence of clinical disease. The morbidity or mortality resulting from anaplasmosis of zoo animals is likely to be very low.

Animals infected with or that have recovered from *Anaplasma* become life-long carriers. As a result, bovine anaplasmosis has the ability to establish in the captive wild and domestic ruminant population within zoos.

The likelihood of bovine anaplasmosis exposure and establishment in domestic and wild ruminants outside the zoo is assessed as very low and is only likely to happen if infected domestic ruminants are released from the zoo onto New Zealand farms. *Anaplasma marginale* and *A. centrale* could be transmitted to cattle, sheep and goats. Affected animals could suffer reproductive losses and drop in milk production, with mortality rates ranging from 29% to 49%.

Anaplasma marginale and *A. centrale* are not zoonotic therefore the consequences for human health are negligible.

Indirect consequences would entail the costs for control and surveillance within the affected zoos. Since animals become life-long carriers, all animals may require testing. Carriers may either be

removed from the collection (via euthanasia) or treated with long-acting antibiotics in an attempt to clear the animals of infection.

There would be additional costs to New Zealand for bovine anaplasmosis control in the event of an incursion in domestic ruminants.

Any trade impacts as a result of a bovine anaplasmosis incursion at the zoo are likely to be minimal if New Zealand can prove that the incursion was contained and there was no spread to animals outside the zoo. However, this may be difficult to prove since bovine anaplasmosis is a vector-borne disease with known competent vectors present in the country. If zoos have released domestic ruminants to farms, this would present an added difficulty.

Therefore, the overall consequences as a result of a bovine anaplasmosis incursion are assessed as low.

25.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for bovine anaplasmosis is non-negligible, and it is assessed to be a risk in captive wild Bovidae.

Therefore, risk management measures can be justified.

25.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Bovine anaplasmosis is an OIE-listed disease of cattle.
- Bovine anaplasmosis has never occurred in New Zealand and is a notifiable disease.
- Bovine anaplasmosis is not known to be a zoonotic disease.
- Cattle are the primary hosts of bovine anaplasmosis, but *A. marginale* has been found in sheep and goats and various wild ruminant species.
- Bovine anaplasmosis has never occurred in Singapore or the UK.
- The incubation period varies from 7 to 100 days.
- Domestic and wildlife ruminants may also become persistently infected and remain carriers for extended periods.
- Clinical disease in wildlife ruminants is rare.
- Bovine anaplasmosis is an arthropod-borne disease transmitted by ticks, flies, mechanical and transplacental methods.
- Diagnosis includes microscopy of stained smears, PCR and, to a lesser extent, serology.
- The presence of *A. marginale* and *A. centrale* has not been demonstrated in the semen of domestic or wildlife ruminants.

25.3.1 Options

One or a combination of the following options may be used:

Anaplasma marginale and A. centrale are not identified as hazards in semen and species within the Giraffidae and Tragulidae families, therefore risk management measures are not warranted.

Option 1

1. Country freedom for bovine anaplasmosis (*A. marginale* and *A. centrale*); AND
2. the animal(s) were resident in bovine anaplasmosis free countries since birth; AND
3. the animal(s) showed no clinical signs of bovine anaplasmosis on the day of export.

Option 2

1. The animal(s) showed no clinical sign of bovine anaplasmosis on the day of export; and
2. the animal(s) were resident since birth in a zone known to be free from bovine anaplasmosis for the previous 2 years; AND
3. the animal(s) were treated with an acaricide and, if necessary, a repellent against biting insects prior to export and were completely free of ticks.

This option is similar to the OIE Code recommendations for the importation of cattle from countries infected with bovine anaplasmosis.

Option 3

1. The animal(s) were resident since birth at premises where no clinical, epidemiological or other evidence of bovine anaplasmosis (*A. marginale* and *A. centrale*) has occurred during the 2 years prior to export; AND
2. the animal(s) showed no clinical signs of bovine anaplasmosis on the day of export; AND
3. the animal(s) were subjected to an MPI approved test for bovine anaplasmosis (*A. marginale* and *A. centrale*) within 30 days prior to export, with negative results; AND
4. the animal(s) were treated with an acaricide and, if necessary, a repellent against biting insects prior to export and were completely free of ticks.

26 Heartwater

26.1 Technical review

26.1.1 Aetiological agent

Family: *Rickettsiaceae*
Genus: *Ehrlichia*
Species: *Ehrlichia ruminantium*

Ehrlichia are rickettsial blood parasites.

Ehrlichia ruminantium (*E. ruminantium*) is the causative agent of heartwater.

26.1.2 OIE list

Heartwater is an OIE-listed disease affecting multiple species (OIE, 2020d).

26.1.3 New Zealand status

New Zealand is free from heartwater (WAHIS, 2019d), as reported to the OIE.

Heartwater is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

26.1.4 Zoonotic potential

Ehrlichia ruminantium has been suspected as the cause of rapidly fatal encephalitis in 3 human cases in the RSA in 2005. However, diagnosis was made via molecular methods only (Allsopp et al., 2005).

In the absence of isolation and characterisation of the organism, the zoonotic potential of *E. ruminantium* should be regarded with care. There have been no other reported cases in humans (OIE Terrestrial Manual, 2018r).

26.1.5 Epidemiology

Host range

Ehrlichia ruminantium affects cattle, sheep and goats (OIE Terrestrial Manual, 2018r).

Ehrlichia ruminantium has been detected in small rodents, birds and reptiles (Oberem & Bezuidenhout, 1987).

Reported natural or experimental infections in wild ruminants include numerous African antelopes, Cape buffaloes (Bovidae), giraffes (Giraffidae) and deer (Cervidae) (OIE Terrestrial Manual, 2018r; Peter et al., 1997).

Captive wild ruminants

A review of the literature shows a paucity of reports relating to heartwater in captive wild animal collections.

There is a single report of heartwater in a captive wild sitatunga (Bovidae) at a Nigerian zoo. None of the other captive wild Bovidae showed any evidence of heartwater infection. Heartwater is enzootic in Nigeria (Okoh et al., 1987).

There are publications highlighting the concerns surrounding the movement of animals from heartwater enzootic areas to heartwater-free areas that harbour competent *Amblyomma* vectors. As well as introducing competent *Amblyomma* vectors with the infected animal (Deem, 1998; Fowler & Miller, 2008).

There is no published evidence of heartwater in species of the Tragulidae family. However, due to the wide host range, it is assumed that these species would be susceptible to infection.

Geographical distribution
Heartwater is enzootic in all sub-Saharan African countries where the *Amblyomma* tick is present. The disease is also present in the surrounding islands (Madagascar, Réunion, Mauritius, Zanzibar, the Comoros Islands and São Tomé) and 3 Caribbean islands (Guadeloupe, Marie-Galante and Antigua) (OIE Terrestrial Manual, 2018r).

According to the WAHIS interface, heartwater has never occurred in Australia, the UK, the USA, Canada, Singapore or the UAE (WAHIS, 2019d).

The status of heartwater in all European countries is unclear.

The distribution of the disease is limited to the distribution of the *Amblyomma* tick vector.

Pathogenesis
Heartwater is an acute, fatal, non-contagious tick-borne disease.

The incubation period in domestic ruminants range from 9 to 29 days (average of 18 days) in cattle and from 7 to 35 days (average 14 days) in sheep (Allsopp et al., 2004). In wild ruminants, the incubation period appears to be longer (Oberem & Bezuidenhout, 1987).

Vertebrate hosts are infected with *E. ruminantium* through tick saliva or regurgitated gut content. Replication of the organism occurs in cells of the regional lymph nodes. They are disseminated via the bloodstream to endothelial cells of blood vessels of various organs and tissues undergoing further multiplication. *Ehrlichia ruminantium* usually infects the endothelial cells of the brain.

Infection of blood vessel cells leads to increased vascular permeability with resultant tissue oedema and effusions into various body cavities including the pleural cavity and pericardial sac (Allsopp et al., 2004).

Heartwater is capable of maintaining itself in wildlife ruminants in the absence of domestic ruminants. This has been demonstrated in the RSA where heartwater is enzootic (Young & Basson, 1973). However, the scenario could be uncertain if heartwater is introduced into a naïve population of wild or captive wild ruminants.

Factors affecting disease progression include strain of the organism, breed and age of animal, conglutinin levels in wild animals and previous exposure to *E. ruminantium* (Oberem & Bezuidenhout, 1987).

Carrier status has been demonstrated in blesboks, buffaloes, elands, wildebeest, kudus and giraffes (Peter et al., 1997). Carrier status has been detected in some species for up to 6 months (Peter et al., 2002).

Clinical signs
Clinical signs of heartwater in domestic ruminants are either peracute, acute or subacute.

In the peracute form of disease, mortality occurs within a few hours of the initial rise in temperature, with or without clinical signs of convulsions and respiratory distress.

Acute heartwater is the most common form and often affects cattle between 3 to 18 months of age. It is characterised by pyrexia, which lasts 3 to 6 days, mild mucoid diarrhoea (in some instances) or a profuse haemorrhagic diarrhoea. In later stages, nervous signs range from mild incoordination to severe convulsions. Animals are hypersensitive to sound and light. Animals eventually fall into lateral recumbency and exhibit bouts of leg pedalling or limb rigidity. Death ensues shortly thereafter.

In the subacute form, the febrile reaction may last for 10 days or longer. Clinical signs are similar to the acute form but less severe. The animal may either die suddenly or recover within a few days.

Nervous signs are due to oedema of the brain. Hydropericardium contributes to cardiac dysfunction during the terminal stages. Pulmonary oedema and hydrothorax result in asphyxiation (Allsopp et al., 2004).

Animals that suffer clinically inapparent infections do exist but are difficult to identify as signs are non-specific and mild. These are often calves under 3 weeks of age, animals infected with less virulent strains of *E. ruminantium* or those that possess partial immunity.

Clinical signs in wild ruminants have not been studied in great detail but are reported to be similar to the acute form of disease in domestic ruminants (Gradwell et al., 1976).

Young and Basson (1973) documented clinical signs in a young eland infected with heartwater.

Wild ruminants that have demonstrated clinical heartwater include wildebeest, blesboks, springboks, elands, water buffaloes, nilgai, blackbucks (Bovidae) and deer (Cervidae) (Oberem & Bezuidenhout, 1987).

Subclinical infections have been reported in wildebeest, blesboks, Cape buffaloes, elands (Bovidae) and giraffes (Giraffidae) (Oberem & Bezuidenhout, 1987).

Animals that are refractory to disease are wildebeest, hartebeest, duikers, scimitar-horned oryxes, bushbucks (Bovidae) and giraffes (Giraffidae) (Oberem & Bezuidenhout, 1987).

In an experimental study, impalas, blue wildebeest, buffaloes, kudus and giraffes from a heartwater enzootic area were inoculated with the Ball 3 strain of *E. ruminantium*. Some animals showed a mild rise in temperature, which could be attributed to handling, while no other clinical signs were observed in all other animals (Gradwell et al., 1976).

African antelopes (blesboks and black wildebeest) showed no clinical signs of heartwater infection even though organisms could be isolated from the blood (Neitz, 1935). An eland that was experimentally infected with sheep blood only showed a mild increase in temperature (Gradwell et al., 1976). This confirms subclinical infection in these animals.

As noted, various wild ruminant species have demonstrated varying degrees of disease and subclinical infections which may be explained by the virulence of the *E. ruminantium* strains and host susceptibility differences.

Transmission

Heartwater is a non-contagious tick-borne disease.

Competent vectors include *Amblyomma hebraeum, A. variegatum, A. pomposum, A. lepidum* and other *Amblyomma* spp. (Petney et al., 1987).

Ticks become infected after feeding on acutely ill, subclinically infected or animals that are reinfected (Bezuidenhout, 1987) and can transmit the organism transstadially, intrastadially and transovarially (Allsopp et al., 2004).

New Zealand has 1 *Amblyomma* species, i.e. the tuatara tick (*A. sphenodonti*). This tick has only been identified on reptilian species in New Zealand, and its distribution is limited to the warmer parts of the country. It has not been demonstrated as a competent vector for *E. ruminantium*.

Studies have molecularly identified numerous novel uncultured *Ehrlichia* spp. in naturally infected *H. longicornis* ticks (Su et al., 2021), which are ticks present in New Zealand. *Haemaphysalis longicornis* vector competency of *E. ruminantium* has, however, not been demonstrated.

Vertical transmission of *E. ruminantium* from cows to their calves has been demonstrated. Colostral cells are the suggested mode of transmission (Deem et al., 1996). This method of transmission has not been demonstrated in wildlife ruminants but could occur.

Diagnosis

A tentative diagnosis of heartwater could be based on the presence of the *Amblyomma* vectors, nervous signs and transudates in body cavities.

A definitive diagnosis can be made by diagnostic tests. Identifying organisms in a brain smear is regarded as the gold standard test. This is usually done during the post-mortem examination.

As per the OIE recommendations, clinical cases can be confirmed by PCR, and ELISA can be used for surveillance.

Although PCR is useful in identifying clinical cases, nested and real-time PCR have failed to detect *E. ruminantium* in subclinical carriers (OIE Terrestrial Manual, 2018r).

Treatment, control and prevention

In countries affected with heartwater, control measures include tick control, antibiotic treatment of clinical cases, prophylactic use of antibiotics and vaccination.

Two methods of tick control can be applied – intensive or strategic. The intensive method has disadvantages such as cost, in the case of large areas and acaricide resistance. The main disadvantage is that animals cannot build up natural resistance to heartwater. If there is a breakdown in the tick control regime, heavy losses result.

The strategic method controls tick numbers, thus allowing a high level of immunity to develop among animals and is more economical.

Tetracyclines have been the most effective antibiotic in treating clinical heartwater.

Prophylactic tetracyclines were used to protect new animals that are to be introduced into heartwater enzootic areas.

The only vaccine commercially available is the Ball 3 strain in sheep blood. This is an intravenous vaccine that requires the animal to be monitored for a rise in temperature and treatment with an antibiotic. Live organisms are used in this vaccine, and therefore, it cannot be used in non-enzootic areas (Allsopp et al., 2004).

In heartwater free countries, tick control for imported animals and preventing entry of carrier animals would aid as a preventative strategy.

Semen

There is no published evidence demonstrating the presence of *E. ruminantium* in the semen of domestic or wildlife ruminants, or the transmission of the agent to naïve dams if inseminated with *E. ruminantium*-contaminated semen.

The OIE does not consider germplasm to be a risk commodity.

26.1.6 Hazard identification conclusion

Heartwater is an OIE-listed disease affecting multiple species. Wild ruminants have demonstrated varying degrees of subclinical infections and clinical disease.

There is no published evidence demonstrating the presence of *E. ruminantium* in the semen of wildlife ruminants.

Ehrlichia ruminantium is not identified as a hazard in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Ehrlichia ruminantium is identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

26.2 Risk assessment

26.2.1 Entry assessment

According to the WAHIS interface, heartwater has never occurred in Australia, the UK, the USA, Canada, Singapore or the UAE. The status of heartwater in all European countries is unclear. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Heartwater is enzootic in the RSA. Wild ruminants in enzootic regions have experienced disease while some animals become subclinical carriers of *E. ruminantium*. Cases of heartwater in captive collections are a rare occurrence. In the RSA specifically, this may be due to wild animals' innate immunity or the enzootic stability developed from low-level infection of infected ticks.

The incubation period for heartwater ranges from 7 to 35 days in domestic ruminants but may be longer in wildlife ruminants. Carrier status has been described in numerous wildlife ruminants and can

be as long as 6 months. It is plausible that animals not exhibiting clinical signs of heartwater or exhibiting mild signs such as a rise in temperature could be passed as clinically sound for export.

If these animals are not treated or examined for ticks, they could be exported with exotic ticks carrying *E. ruminantium*.

Therefore, the likelihood of entry of *E. ruminantium* via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from heartwater-affected countries is assessed as low to moderate.

26.2.2 Exposure assessment

Heartwater is a non-contagious tick-borne disease transmitted by *Amblyomma* spp. of ticks. Ticks present in New Zealand have not been documented as competent vectors of *E. ruminantium.* Vertical transmission has been demonstrated in domestic ruminants only.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If carrier captive wild ruminants are imported into New Zealand zoos, they could be a source of infection to tick vectors, which could infect susceptible animals. However, if there are no competent vectors in New Zealand, *E. ruminantium* cannot be transmitted to susceptible animals. Therefore, the carrier animals would either harbour the organisms, thus remaining carrier animals until natural death without spreading it to other animals, or they would succumb to clinical disease and mortality. In this scenario, only the imported carrier animals may be affected without further spread or establishment.

The likelihood of heartwater affecting domestic or wild animals outside the zoo is assessed as negligible in the absence of a competent vector.

There has only been one publication on the zoonotic potential of *E. ruminantium*. This article has been noted but does not fully confirm *E. ruminantium* as a zoonotic agent. Therefore, exposure to humans is assessed as negligible.

In the absence of competent vectors, the likelihood of *E. ruminantium* exposure and establishment within the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as negligible, the likelihood of exposure and establishment outside the zoo is assessed as negligible.

26.2.3 Risk estimation

Since the exposure is assessed as negligible, the risk estimate for *E. ruminantium* is negligible, and it is not a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures are not warranted.

27 Q fever

27.1 Technical review

27.1.1 Aetiological agent

Family: *Coxiellaceae*
Genus: *Coxiella*
Species: *Coxiella burnetii*

Coxiella burnetii (*C. burnetii*) is the causative agent of Q fever.

Coxiella burnetii is an obligate intracellular bacterium (OIE Terrestrial Manual, 2018u).

27.1.2 OIE list

Q fever is an OIE-listed disease affecting multiple species (OIE, 2020d).

27.1.3 New Zealand status

New Zealand is free from Q fever (WAHIS, 2019d), as reported to the OIE.

Q fever is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

27.1.4 Zoonotic potential

Q fever is a zoonotic disease (OIE Terrestrial Manual, 2018u). Humans become infected through airborne transmission from infected animals or contaminated animal products.

The acute form of disease may range from self-limiting flu-like symptoms to more severe pneumonia or granulomatous hepatitis. If left untreated, the disease may become chronic and result in complications and even death (OIE Terrestrial Manual, 2018u).

27.1.5 Epidemiology

Host range

A wide range of domestic and wild animals can be infected with *C. burnetii,* including arthropods (ticks).

Sheep, goats and cattle are susceptible to infection (Ho et al., 1995; Palmer et al., 1983; Roest et al., 2012). Domestic ruminants are the main reservoirs of Q fever.

Companion animals such as cats, dogs, rabbits and birds have been suggested as reservoirs for human disease as well (Marrie et al., 1988; Maurin & Raoult, 1999).

Various species of deer and marsupials may also be susceptible to infection (González-Barrio et al., 2015; Marrie & Raoult, 1997).

Q fever has been reported in wildlife Bovidae, namely waterbucks, sable and roan antelopes, dorcas and dama gazelles and water buffaloes (Clemente et al., 2008; Garcia-Seco et al., 2016; González-Barrio & Ruiz-Fons, 2019; Lloyd et al., 2010; Perugini et al., 2009).

Coxiella burnetii antibodies were identified in Arabian oryxes in Saudi Arabia (Greth et al., 1992).

Seropositivity of saiga antelopes to *C. burnetii* was reported in Kazakhstan (Orynbayev et al., 2016).

There is no published evidence of *C. burnetii* infections in species of the Giraffidae and Tragulidae families.

Captive wild ruminants

There are limited published reports of Q fever in captive wild animals.

Coxiella burnetii antibodies in Arabian oryxes were identified from captive and strictly isolated populations in Saudi Arabia (Greth et al., 1992).

Waterbucks and sable antelopes at Lisbon Zoo in Portugal were reported to be infected with Q fever. The disease caused abortions and stillbirths in these animals. The source of the infection was unknown, although it was suggested that the animals may have acquired it at the zoo, as they were born locally (Clemente et al., 2008).

García-Seco et al. (2016) described natural infection in a population of dorcas gazelles (*Gazella dorcas*) from Madrid Zoo in Spain.

Geographical distribution

Q fever has worldwide distribution with the exception of New Zealand. In most countries, Q fever is not included in the list of nationally notifiable diseases (Maurin & Raoult, 1999).

Of the approved countries, Singapore is the only country where Q fever has never occurred. It is a nationally notifiable disease in Singapore (WAHIS, 2019d).

Pathogenesis

Experimentally, the incubation period of Q fever is approximately 2 to 4 weeks after intranasal inoculation of domestic ruminants.

Following this route of infection, *C. burnetii* DNA was detected in the upper respiratory tract, spleen and thymus (14 days post infection), in the uterus and placenta (28 days post infection) and in the upper and lower respiratory tract, haematopoietic system, liver, urinary and alimentary tract and the heart (42 days post infection) (Roest et al., 2012).

The experiment suggested that *C. burnetii* has a tropism for trophoblasts. After parturition, the amount of *C. burnetii* DNA in tissues gradually decreased to zero, except for mucosa of the nostrils. This was likely due to the removal of trophoblasts from the body at parturition, thus eliminating the bacteria's primary replication site (Roest et al., 2012).

Tropism for trophoblasts suggests that only pregnant animals are susceptible to infection by *C. burnetii* (Roest et al., 2012).

It is likely that undetectable *C. burnetii* may be harboured in other bodily tissues, thus infecting trophoblasts when they become available (Roest et al., 2012). Latent infections have been described in guinea pigs, and therefore, other animals may also experience latency (Maurin & Raoult, 1999).

Subclinically infected animals can shed organisms in secretions and excretions (OIE Terrestrial Manual, 2018u). Shedding patterns in animals are still unclear. However, simultaneous shedding in milk, faeces and vaginal discharges is rare (Guatteo et al., 2007; Rousset et al., 2009). The most frequent period of bacterial shedding has been noted in vaginal discharges on the day of parturition (Arricau-Bouvery et al., 2005). Shedding of the virus may persist for several months (Guatteo et al., 2007).

Clinical signs

Clinical manifestations of *C. burnetii* infections may depend on the species, age and sex, immunological status of the challenged animal and strain of bacteria (Maurin & Raoult, 1999).

Coxiella burnetii-infected animals do not usually experience clinical disease (Maurin & Raoult, 1999). The pathogenesis of the organism may be the likely explanation for this (i.e. the bacteria's tropism for trophoblasts).

Sheep, goats and cattle are usually subclinical carriers (OIE Terrestrial Manual, 2018u).

Disease in animals may also manifest in the acute and chronic forms.

In the acute phase, animals remain subclinically infected, but *C. burnetii* can be detected in blood, lungs, spleen and liver.

In the chronic phase, persistent shedding is noted in faeces and urine. The uterus and mammary glands become the primary sites of replication. Shedding into the environment thus occurs mostly at parturition.

In chronically infected domestic ruminants, clinical signs include late abortions and reproductive disorders (premature birth, dead or weak offspring) (Palmer et al., 1983). Metritis and infertility in cattle have been reported (Ho et al., 1995).

Abortions and stillbirths due to *C. burnetii* have been reported in captive wild waterbucks and sable antelopes. No other clinical signs were noted in these animals (Clemente et al., 2008).

Coxiella burnetii has also been isolated in dorcas gazelles in the absence of clinical signs (Garcia-Seco et al., 2016).

Transmission

Transmission is horizontal via direct and indirect contact with infected animals and contaminated animal products. *Coxiella burnetii* is shed intermittently in milk, vaginal discharges, birth products and faeces of infected animals.

Transmission occurs through inhalation of desiccated aerosol particles. Susceptible animals also become infected after exposure to contaminated reproductive tissue and other animal products such as wool (Maurin & Raoult, 1999).

Vertical and sexual transmission could occur as it has been demonstrated in mice (Tylewska-Wierzbanowska & Kruszewska, 1990). However, the significance of vertical and sexual transmission in other animals is still unclear at this stage (OIE Terrestrial Manual, 2018u).

Ticks may be involved in transmission of *C. burnetii* (Marrie et al., 1988). More than 40 tick species can be infected by *C. burnetii*. These include but are not limited to *Amblyomma* spp., *Dermacentor* spp., *Rhipicephalus* spp., *Haemaphysalis* spp. and *Ixodes* spp. (Marrie & Raoult, 1997; Maurin & Raoult, 1999; Pacheco et al., 2013). They are an important aspect in the transmission cycle of wild animals (Knap et al., 2019).

Coxiella burnetii produces spore-like forms that are highly resistant and have the ability to survive extracellularly as infectious particles (Heinzen et al., 1999). This trait aids persistence in the environment and transmission (Kersh et al., 2010). *Coxiella burnetii* may survive for several weeks in the environment (Maurin & Raoult, 1999).

It has also been suggested that infectious particles containing *C. burnetii* can be transported by the wind (Marrie & Raoult, 1997) over a distance of at least 30 kilometres (Eldin et al., 2017). Therefore, animals or humans may become infected with *C. burnetii* without a history of close contact with infected animals (Maurin & Raoult, 1999).

Based on the study by Jones et al. (2006), it was concluded that the infectious dose of *C. burnetii* is likely 1 rickettsia, and that the probability of a single organism resulting in infection is approximately 0.9. The risk of an animal or human becoming infected is dependent on the infectious dose, the airborne concentration of the agent and the animal or human's exposure duration.

Diagnosis

Currently, there is no gold standard diagnostic test for Q fever. Clinical diagnosis can be made using direct detection and quantification by PCR and serological ELISA (Niemczuk et al., 2014; Sidi-Boumedine et al., 2010).

Polymerase chain reaction is the recommended diagnostic test for population freedom from infection, eradication purposes and the confirmation of clinical cases.

ELISA is recommended for population freedom from infection, eradication and surveillance purposes and determining the immune status in individual animals or populations post-vaccination.

It must be noted that not all recommended tests have undergone formal validation (OIE Terrestrial Manual, 2018u).

Individual animals can only be assessed as free if the herd or flock is free and has no serological or clinical history of Q fever. It is difficult to ensure that the status of the animal has not changed over time because transmission is by air (González-Barrio & Ruiz-Fons, 2019).

A diagnosis of Q fever in wildlife can be extremely difficult for various reasons such as the quality of samples, absence of signs in infected animals and the lack of available specific validated diagnostic tests. Test methods that can be used in wildlife include bacterial culture and isolation, staining of impression smears, histology and immunohistochemistry, PCR and ELISA (González-Barrio & Ruiz-Fons, 2019).

As recommended by the OIE, ELISA is the preferred method for surveillance of wildlife populations, and PCR is the most appropriate for detecting *C. burnetii* in biological wildlife samples (González-Barrio & Ruiz-Fons, 2019).

Treatment, control and prevention

Tetracyclines are the most effective antibiotic for use in animal and human infections (Maurin & Raoult, 1999).

Vaccinations have been developed for the protection of humans and animals. Inactivated *C. burnetii* vaccines that contain phase I antigens are available and are likely to generate an appropriate immune response (Elliott et al., 2015; Zhang et al., 2013).

Vaccination helps reduce shedding and abortions. However, their use has been limited due to the fact that vaccination in mainly livestock is protective and safe if animals are uninfected at the time of vaccination (Maurin & Raoult, 1999). There is currently insufficient data to support the theory that vaccination would be safe and protective in wildlife species (González-Barrio & Ruiz-Fons, 2019).

Control measures in humans could be directed toward at-risk groups such as laboratory personnel, abattoir workers, shearers, veterinary personnel, animal handlers and others. Control measures should include proper safety and hygiene practices, personal protective wear and vaccination (Maurin & Raoult, 1999).

Semen

Viable *C. burnetii* has been isolated from the semen of naturally infected bulls. In the experiment, naturally infected bulls were tested serologically. Their semen was also tested. Of the *C. burnetii* seropositive bulls, approximately 22% demonstrated the presence of *C. burnetii* in their semen (Kruszewska & Tylewska-Wierzbanowska, 1997).

There is experimental evidence that cows could become infected after intravaginal inoculation of *C. burnetii* (Parker et al., 1948).

A Saharawi dorcas gazelle was confirmed positive for *C. burnetii* after semen samples tested positive on PCR for *C. burnetii*. This is the first report of a Saharawi dorcas gazelle infection with *C. burnetii* and the first time that *C. burnetii* has been detected in semen from a captive wild animal, suggesting the possibility of venereal transmission in captive wild species (Garcia-Seco et al., 2016).

27.1.6 Hazard identification conclusion

Q fever is an OIE-listed disease affecting multiple species. There is evidence of Q fever in wildlife ruminants within the scope of this IRA.

Coxiella burnetii has been detected in the semen of both domestic and wildlife ruminants.

There is no published evidence of *C. burnetii* infections in species of the Giraffidae and Tragulidae families.

Coxiella burnetii is not identified as a hazard in captive wild Giraffidae, Tragulidae and their semen.

Coxiella burnetii is identified as a hazard in captive wild Bovidae and their semen.

27.2 Risk assessment

27.2.1 Entry assessment

Q fever has never occurred in Singapore. Disease is present in all other approved countries.

There is likely to be a very small number of live captive wild Bovidae imported into New Zealand, these imports are also likely to be infrequent. Due to the low volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Q fever can infect a wide range of animals, including wild and captive wild Bovidae. Subclinical infections as well as clinical disease has been reported in captive wild Bovidae.

The incubation period of Q fever is approximately 2 to 4 weeks. Due to the bacteria's affinity for trophoblasts, captive wild Bovidae are likely to become infected but remain subclinical until they become pregnant. It has also been noted that not all infected pregnant animals will abort or experience reproductive disorders. This is because clinical manifestation is dependent on the species, age, sex and immunological status of the challenged animal and the strain of bacteria.

Coxiella burnetii also has the ability to cause latent infections. If captive wild Bovidae are incubating the disease, are subclinical carriers or have latent infections, they could be passed as clinically sound for export if they are exported from Q fever affected countries.

Coxiella burnetii is shed in all secretions and excretions. There is evidence of the presence of *C. burnetii* in semen of both domestic and wildlife ruminants. However, shedding is intermittent, as was demonstrated in experimental studies where only a percentage of Q fever positive bulls presented with *C. burnetii* in their semen.

Therefore, the likelihood of entry of *C. burnetii* via captive wild Bovidae (within the scope of this IRA) from Q fever affected countries is assessed as moderate.

The likelihood of entry of *C. burnetii* via semen of captive wild Bovidae (within the scope of this IRA) from Q fever affected countries is assessed as low.

27.2.2 Exposure assessment

Transmission occurs horizontally via direct and indirect contact with infected animals and contaminated animal products. Vertical and venereal transmission may also occur. *Coxiella burnetii* can be found in all secretions and excretions including blood, milk, faeces, urine, vaginal discharges and semen.

Humans and animals become infected via inhalation or ingestion of desiccated infectious particles. The bacteria are resistant to environmental conditions and can survive outside the body for several weeks. Infectious particles can travel distances of up to 30 kilometres with the wind.

Coxiella burnetii has been detected in over 40 species of ticks. Ticks are said to play an important role in the transmission cycle of wild animals. There is uncertainty as to whether New Zealand ticks could be competent vectors of *C. burnetii*. The *Haemaphysalis* and *Ixodes* genera, known to carry *C. burnetii,* are present in New Zealand. Currently, there are no documented competent tick vectors of *C. burnetii* in New Zealand.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If infected subclinical animals are imported into New Zealand zoos, they could act as a source of infection to other animals within the zoo. Subclinically infected animals can shed bacteria in their secretions and excretions. The highest volume of bacteria is found in vaginal discharges and birth products on the day of parturition. Animals in the same enclosure could become infected either from secretions/excretions of subclinically infected animals or from birth products if animals give birth in the enclosure.

Coxiella burnetii can remain infectious as desiccated particles in the environment for several weeks. These particles can also travel a distance of at least 30 kilometres with the wind. One rickettsia is capable of initiating infection with a probability of 0.9. This implies that *C. burnetii* can be highly infectious within a suitable environment. As a result, animals and humans within the zoo and outside the zoo within a 30-kilometre radius of infected animals could become infected. It should, however, be noted that in the cases where infectious particles travelled by wind to potentially infect animals and humans 30 kilometres downwind, a large concentration of bacteria was required to result in that outcome. There is a low likelihood of this occurring if there are low numbers of infected animals in the zoo and a lesser likelihood if they are not giving birth at the same time.

If domestic ruminants are kept at the zoo, they could become infected with *C. burnetii*. Should these domestic ruminants be released onto New Zealand farms, they could transmit *C. burnetii* to other domestic ruminants.

Q fever has the ability to establish in domestic ruminant and marsupial populations. There have also been several reports of Q fever infections in captive wild Bovidae that have led to reproductive failure in these captive populations. It has been noted that months after abortions were observed, numerous animals within the captive population showed seropositivity to *C. burnetii*. The disease does not result in the death of infected animals. Instead, the infected animals usually have subclinical or latent infections. As a result, Q fever could establish in captive wild animal populations or animal populations outside the zoo.

Q fever is a zoonotic disease; and therefore, zoo and veterinary personnel could be exposed to infection. Visitors at the zoo are less likely to be exposed, given their lower exposure duration in comparison to zoo personnel. They would also not be exposed to animals giving birth or aborting. However, there are reports of Q fever infections in humans without a history of close contact with infected animals. This has been attributed to *C. burnetii* being carried by wind.

Coxiella burnetii has been detected in the semen of both domestic and wildlife ruminants. There is experimental evidence demonstrating that heifers could become infected with *C. burnetii* after installation of *C. burnetii* intravaginally. This has not been demonstrated under natural settings; however, many authors have suggested that sexual transmission is a plausible route. There is no evidence that semen of wildlife ruminants could cause infection in a naïve dam after AI. Therefore, extrapolation is made from domestic ruminants to wildlife ruminants and it is assumed that infection may occur.

There are limited numbers of captive wild female ruminants in New Zealand zoos, and thus, the volume of imported semen is likely to be very low. The number of animals that could be exposed to contaminated semen is also going to be very low.

Therefore the likelihood of *C. burnetii* exposure and establishment within the zoo via infected captive wild Bovidae is assessed as moderate, the likelihood of exposure and establishment outside the zoo is assessed as very low and the likelihood of exposure, and establishment via contaminated semen of captive wild Bovidae is assessed as very low.

27.2.3 Consequence assessment

Q fever is an OIE-listed disease affecting multiple species.

There is likely to be a very small number of live captive wild Bovidae or semen imported into New Zealand. These imports are also likely to be infrequent.

Direct consequences would relate to the infected imported animals and those that become infected. Q fever does not cause severe life-threatening disease in animals. However, it does result in abortions, stillbirths and other reproductive disorders. If animals are imported into zoos for breeding purposes, this would negatively impact the zoos' ASMP and conservation efforts.

As mentioned, there is a very low likelihood that Q fever would establish in animal populations outside the zoo. However, if this does happen, Q fever could have a severe negative economic impact on the cattle, sheep, goat and deer populations in New Zealand. Q fever affects a wide range of animals, including companion animals, wildlife and birds.

Q fever is a zoonotic disease. Zoo and veterinary personnel could become infected. If Q fever establishes in animals outside the zoo, infected animals could then become reservoirs and sources of infection for a larger number of humans. Infections in humans manifest as mild flu-like symptoms or, if left untreated, could result in complications and death.

Ticks have been implicated in the transmission of *C. burnetii*, especially in wildlife. If New Zealand tick species become competent vectors of *C. burnetii*, this would further hamper efforts to control and eradicate the disease.

Indirect consequences would entail the costs for control and surveillance within the affected zoos and the cost for national control, surveillance and potentially eradication should Q fever spread to animal populations outside the zoo.

Since New Zealand is among the few countries that have never experienced an incursion of Q fever, there are likely to be negative trade impacts. These may be minimal if New Zealand can prove that the incursion was contained and there was no spread to animals outside the zoo. However, this may be difficult to prove due to the probability of transmission of infectious particles via wind and domestic ruminants being released from the zoo.

Therefore, the overall consequences as a result of a Q fever incursion are assessed as moderate to high.

27.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for Q fever is non-negligible, and it is assessed to be a risk in captive wild Bovidae and their semen.

Therefore, risk management measures can be justified.

27.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Q fever is an OIE-listed disease affecting multiple species.
- Q fever has never occurred in New Zealand and is nationally notifiable.
- Q fever is a zoonotic disease.
- A wide range of domestic and wild animals can be infected with *C. burnetii,* including arthropods (ticks).
- Singapore is the only approved country where Q fever has never occurred.
- Q fever has been reported in captive wild Bovidae.
- The incubation period of Q fever is approximately 2 to 4 weeks.
- Most animals experience subclinical infections .
- Subclinically infected animals can shed organisms in secretions and excretions
- Latent infection is a feature of Q fever.
- Transmission occurs horizontally via direct and indirect contact with infected animals and contaminated animal products. Vertical and venereal transmission may also occur.
- *Coxiella burnetii* can survive for several weeks outside the body, and infectious particles can be transported over long distances by wind.
- Diagnostic tests available include PCR, ELISA, culture and isolation, stained impression smears, histology and immunohistochemistry.
- *Coxiella burnetii* has been detected in the semen of both domestic and wildlife ruminants.
- Experimental studies have demonstrated that heifers became infected after intravaginal inoculation with *C. burnetii.*

27.3.1 Options

One or a combination of the following options may be used:

Coxiella burnetii is not identified as a hazard in species within the Giraffidae and Tragulidae families and their semen, therefore risk management measures are not warranted.

Option 1

1. Country freedom for Q fever; AND
2. the animal(s)/male donor(s) were resident in Q-fever-free countries since birth prior to export; AND
3. the animal(s)/male donor(s) showed no clinical signs of Q fever on the day of export.

Option 2

1. For 12 months immediately prior to export the animal(s)/donor male(s) was continuously resident at the premises where no clinical, epidemiological or other evidence of Q fever has occurred in any animal species during the previous 5 years; AND
2. the animal(s)/donor male(s) was tested for Q fever by PCR and/or ELISA prior to entering this premises and was negative; AND
3. the animal(s)/male donor(s) showed no clinical signs of Q fever on the day of export or semen collection; AND

4. the animal(s) were treated with an acaricide prior to export and were completely free of external parasites.

Option 3

For 12 months immediately before export, the animal(s)/donor male(s) were continuously resident at the exporting premises, and no clinical, epidemiological or other evidence of Q fever occurred at the premises in any animal species during the previous 2 years; AND

Animal:

1. Within 30 days prior to export, the animal(s) were tested for *C. burnetii* with an MPI-approved test (PCR and ELISA) with negative results; AND
2. the animal(s) were treated with an acaricide prior to export and were completely free of external parasites.

Semen:

1. Within 30 days following semen collection, donor male(s) were tested for *C. burnetii* with an MPI-approved test (PCR and ELISA) with negative results; OR
2. A straw of semen from each collection batch was tested for *C. burnetii* with an MPI-approved test (microagglutination test or indirect immunofluorescence assay or PCR) and was negative.

28 Besnoitia

28.1 Technical review

28.1.1 Aetiological agent

Family: *Sarcocystidae*
Genus: *Besnoitia*
Species: *Besnoitia besnoiti*

There are a number of species classified under the *Besnoitia* genus. For the purposes of this IRA, only *Besnoitia besnoiti* was a relevant species affecting animals within the scope of this IRA, and only *B. besnoiti* will be assessed.

Besnoitia besnoiti (*B. besnoiti*) is the causative agent of besnoitiosis (Worthington & Bigalke, 2001).

The *Besnoitia besnoiti* strains identified in cattle and antelope are closely related but have some differences.

28.1.2 OIE list

Besnoitiosis is not an OIE-listed disease.

28.1.3 New Zealand status

Following review of the literature, no known cases of besnoitiosis have been reported in New Zealand.

Besnoitiosis is not a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

28.1.4 Zoonotic potential

Members of the genus *Besnoitia* are not known to be zoonotic (Joint Pathology Center, 2018).

28.1.5 Epidemiology

Host range

Examples of intermediate hosts of *B. besnoiti* include cattle, kudus, blue wildebeest and impalas (Bovidae) (Joint Pathology Center, 2018).

The definitive hosts are members of the Felidae family.

Cattle are the only domestic ruminants known to be affected with clinical besnoitiosis (Bigalke & Prozesky, 2004).

In a survey for besnoitiosis in wild ruminants, antibodies against *B. besnoiti* were detected in wildebeest in Namibia. Springboks were included in the survey but showed no serological evidence of exposure (Seltmann et al., 2020).

Clinically inapparent infections have been reported in blue wildebeest, kudus and impalas (Bovidae) (Bigalke et al., 1967; McCully et al., 1966).

There is no published evidence of besnoitiosis in species of the Giraffidae or Tragulidae families.

Captive wild ruminants

There are reports of besnoitiosis in captive wild ruminants caused by a number of different *Besnoitia* spp. None of them include *B. besnoiti* (Ayroud et al., 1995; Foley et al., 1990; Glover et al., 1990).

Geographical distribution

Besnoitiosis is widespread over the African continent and is economically important as a cattle disease in parts of the RSA.

Outside of Africa the disease has been reported in parts of Asia, Europe and the Middle East (Bigalke & Prozesky, 2004).

Other *Besnoitia* spp. have been reported in animals in the USA and Canada; however, *B. besnoiti* has not been reported in North America (Gutiérrez-Expósito et al., 2012).

There are no known reports of *B. besnoiti* in Australia or Singapore.

Pathogenesis

The incubation period in experimental studies is variable depending on the routes of infection. An incubation period of 4 days was reported after oral infection in cattle. The incubation period after mechanical transmission by biting flies was an average of 13 days (Bigalke & Prozesky, 2004).

Besnoitia besnoiti is a protozoan parasite with a life cycle involving an intermediate and definitive host. The cycle in the definitive host has not been described in detail.

Cattle and other susceptible ruminant species are intermediate hosts. They contract *B. besnoiti* by ingestion of oocysts that are excreted in the faeces of members of the Felidae family. The sporozoites enter the circulation and multiply in endothelial cells of the skin, fasciae, upper respiratory tract and testicles. Tachyzoites invade adjacent or distant endothelial cells to produce more endozoites. The developmental cycle is accompanied by the anasarca stage of disease and followed by cyst formation.

Cyst formation begins a week after the proliferation cycle. These cysts are activated hypertrophic cystozoite-containing histiocytic cells. The bradyzoites multiply in enlarged host cells. After 6 weeks, the characteristic thick-walled cysts are formed. Cysts are associated with the chronic scleroderma stage of disease.

Hosts of the Felidae family become infected after ingesting muscle or tissue contaminated with cysts. Other carnivorous animals have been suggested as definitive hosts; however, this has yet to be proven (Bigalke & Prozesky, 2004).

The *B. besnoiti* strains identified in blue wildebeest, impalas and kudus are antigenically closely related to the cattle strains but are non-pathogenic. In experimental studies of the antelope strain in cattle, sheep and rabbits, the strain was demonstrated to be viscerotropic rather than dermatotropic (McCully et al., 1966).

Clinical signs

Most animals (either domestic or wild) that are naturally infected experience clinically inapparent disease (Bigalke & Prozesky, 2004).

Cattle infected with the antelope strains showed only a mild febrile reaction that lasts a few days with no other reactions noted (Bigalke et al., 1967).

Clinical disease in cattle with the cattle strain is characterised by acute anasarca and chronic scleroderma stages.

Signs begin with pyrexia, listlessness and inappetence followed by anorexia. Hyperaemia of the muzzle and hairless areas are seen in light-coloured animals. Anasarca develops at this stage in various parts of the body.

The scleroderma stage is evident by thickening, hardening and prominent folding and puckering of the skin that is generalised or focal. This normally occurs 3 to 4 weeks after the initial rise in temperature.

Movement becomes slow and restricted due to pain. This is accompanied by progressive hair loss, hyperkeratosis and dermatitis crustosa. Fissures that develop may become infected or infested with bacteria or maggots.

Most animals survive; however, recovery is slow, and scleroderma and alopecia may be permanent.

In a survey to investigate besnoitiosis in antelope in KNP, kudus (8), impalas (74) and blue wildebeest (21) that were clinically healthy were euthanased and examined. Of the animals surveyed, 90% of blue wildebeest, 12.5% of kudus and 45% of impalas were positive for *B. besnoiti* cysts in the cardiovascular system (McCully et al., 1966).

Transmission

Horizontal direct and indirect transmission has been reported.

There is experimental evidence that chronically infected cattle with large volumes of cysts can serve as a source of infection to other cattle (Bigalke, 1968; Bigalke & Prozesky, 2004; Shkap et al., 1994).

Direct contact via wounds, or mucous membranes could result in transmission to in contact animals (Basso et al., 2013).

There is evidence that bloodsucking insects such as tabanids are capable of transmitting *B. besnoiti* from chronically infected cattle to susceptible cattle (Bigalke, 1968; Shkap et al., 1994). This form of transmission plays a minor role in the epidemiology of the disease.

The most significant transmission route requires the presence of a Felidae host. Ruminants contract *B. besnoiti* after the ingestion of Felidae faeces contaminated with cysts.

Diagnosis

Endozoites of *B. besnoiti* may be identified in blood smears during the febrile stage but can be difficult.

Histopathology of the skin in the scleroderma stage can be used to make a clinical diagnosis.

From 6 to 7 weeks post infection, cysts are visible to the naked eye in the scleral conjunctivae of infected cattle. These cysts can be identified microscopically from tissue specimens.

The IFAT and ELISA may be used for surveillance purposes and the detection of an immune response post vaccination (Bigalke & Prozesky, 2004).

Treatment, control & prevention

There is currently no effective treatment to cure besnoitiosis. Supportive therapy can be administered to manage the signs. Palliative treatment includes tetracyclines for secondary bacterial infections, wound dressing, controlling infestations, protection from inclement weather and provision of food and water.

A live vaccine for cattle has been developed in the RSA using a blue wildebeest strain. The vaccine prevents clinical disease but not subclinical infections. Cattle require annual boosters to remain protected (Bigalke & Prozesky, 2004).

In enzootic areas, identifying infected animals and removing them from the herd would help reduce new infections (Bigalke & Prozesky, 2004).

Semen

In an experimental study of 40 naturally infected cattle, 40 bulls (seronegative, seropositive subclinical and seropositive clinical infections) had their semen tested via PCR. No *B. besnoiti* DNA was detected in any of the semen samples tested. It was concluded that transmission of *B. besnoiti* via semen is unlikely (Esteban-Gil et al., 2014).

In the study by Gazzonis et al. (2017), natural mating was a risk factor in the transmission of besnoitiosis from infected bulls to naïve cows. This was likely due to the contact of mucous membranes. Artificial insemination was a safer option (Gazzonis et al., 2017).

There is no published evidence demonstrating the presence of *B. besnoiti* in semen of wildlife ruminants or the transmission of the agent to naïve dams if inseminated with *B. besnoiti*-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

28.1.6 Hazard identification conclusion

Besnoitiosis is not an OIE-listed disease.

Besnoitiosis (caused by *B. besnoiti*) has been reported in wild ruminants of the Bovidae family.

Besnoitia besnoiti has not been demonstrated in the semen of domestic ruminants. There is no literature evidence of *B. besnoiti* in semen of wildlife ruminant species covered under this IRA. The transmission of *B. besnoiti* via AI is unlikely.

Besnoitia besnoiti is not identified as a hazard in semen of captive wild Bovidae, Giraffidae and Tragulidae, and will not be assessed further.

There is no evidence to suggest that *B. besnoiti* can infect or be carried by species within the Giraffidae and Tragulidae families.

Besnoitia besnoiti is not identified as a hazard in captive wild Giraffidae and Tragulidae species and will not be assessed further.

Besnoitia besnoiti is identified as a hazard in captive wild Bovidae.

28.2 Risk assessment

28.2.1 Entry assessment

Besnoitiosis (*B. besnoiti*) has not been reported in Australia, Singapore, the USA or Canada. The disease is not notifiable in these countries. Other species of *Besnoitia* have been identified in the USA and Canada. Since the disease is not OIE-listed or notifiable in these countries, assurance that these countries are free of disease may be difficult to establish.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, these imports are also likely to be infrequent. Due to the volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Besnoitiosis (*B. besnoiti*) has been reported in wild ruminants. Species known to carry the organism include blue wildebeest, impalas and kudus.

Studies have shown that wild ruminants are capable of harbouring *B. besnoiti* in the absence of clinical disease. If captive wild ruminants are silent carriers, they may go unnoticed and be passed as clinically sound for export if exported from besnoitiosis-affected countries.

Besnoitiosis has only been reported in wild Bovidae. There are no known reports of besnoitiosis (*B. besnoiti*) in captive wild Bovidae.

Therefore, the likelihood of entry of *B. besnoiti* via captive wild Bovidae (within the scope of this IRA) from besnoitiosis-affected countries is assessed as very low.

28.2.2 Exposure assessment

The modes of transmission of besnoitiosis are direct and indirect contact. Severely infected cattle can act as a source of contamination for susceptible cattle by transmitting cysts from wounds and mucous membranes to animals in contact with them. Haematophagous insects have also been implicated in transmission from chronically infected cattle. These play a minor role in the epidemiology of besnoitiosis. The most significant route of transmission is likely the ingestion of cysts in faeces of definitive hosts (Felidae).

Transmission in wildlife ruminants has not been documented. However, we could extrapolate the transmission methods from cattle.

It should be noted that there are strain differences that relate to the pathogenicity of the strains isolated from cattle and antelope. Studies have demonstrated that *B. besnoiti* strains isolated from antelope are non-pathogenic to cattle and antelope. The antelope strains are viscerotropic instead of dermatotropic, and therefore, wild ruminants are only identified as infected when examined by post-mortem. These infections are often found incidentally. Since wild ruminants are clinically healthy and cysts do not appear in the skin, it is unlikely that biting flies will be able to pick up cysts and transmit these to other susceptible animals.

Additionally, since clinical disease does not occur in wild ruminants, the likelihood of transmission via direct contact of wounds or mucous membranes is also negligible. This therefore suggests that an infected captive wild ruminant cannot transmit the organism to susceptible animals. If the infected captive wild ruminants die, according to zoo procedures, the animal must be examined via post-mortem. The carcass will not be fed to carnivorous animals in the zoo or outside the zoo and will be disposed of accordingly. Therefore, the definitive host cannot become infected or pass cysts in their faeces. There will be no completion of the life cycle of *B. besnoiti*.

Besnoitia besnoiti is not zoonotic, and therefore, humans are not at risk of infection.

Since there is no exposure pathway to susceptible animals, there can be no establishment in these animals.

Therefore, the likelihood of *B. besnoiti* exposure and establishment within the zoo is assessed as negligible, and the likelihood of *B. besnoiti* exposure and establishment outside the zoo is assessed as negligible.

28.2.3 Risk estimation

Since the exposure is assessed as negligible, the risk estimate for besnoitiosis is negligible, and it is not a risk in captive wild Bovidae.

Therefore, risk management measures are not warranted.

29 Bovine babesiosis

29.1 Technical review

29.1.1 Aetiological agent

Family: *Babesiidae*
Genus: *Babesia*
Species: *Babesia bigemina; Babesia bovis; Babesia divergens*

Babesia are intra-erythrocytic protozoan parasites.

Babesia bigemina (B. bigemina), Babesia bovis (B. bovis) and *Babesia divergens (B. divergens)* are the causative agents of bovine babesiosis (OIE Terrestrial Manual, 2018e).

29.1.2 OIE list

Bovine babesiosis is an OIE-listed disease of cattle (OIE, 2020d).

29.1.3 New Zealand status

New Zealand is free from bovine babesiosis (WAHIS, 2019d), as reported to the OIE.

Babesiosis (*Babesia* spp. [exotic]) is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

29.1.4 Zoonotic potential

There are no known reports of disease in humans caused by *B. bigemina*, except for serological evidence of exposure.

Disease in humans caused by *B. bovis* is not common but may occur (Parija et al., 2015).

Babesia divergens is zoonotic (Zintl et al., 2003). In Europe, the disease in humans can lead to a fatal outcome in immunocompromised patients (Kukina et al., 2018).

29.1.5 Epidemiology

Host range

Cattle are the primary hosts of bovine babesiosis and reservoirs of *B. bigemina, B. bovis* and *B. divergens.*

Babesia bigemina and *B. bovis* have a high degree of host specificity. European, Sanga and zebu cattle are susceptible to infection (de Vos et al., 2004).

Evidence of *B. bigemina* and *B. bovis* DNA has been demonstrated in free-ranging nilgai (Bovidae) along the Texas-Mexico border. The organism itself was not identified in these animals (Cardenas-Canales et al., 2011).

Molecular and serological evidence suggest that white-tailed deer (Cervidae) in the USA may harbour *B. bigemina* and *B. bovis* (Holman et al., 2011).

Red deer and roe deer (Cervidae) in Europe have demonstrated PCR evidence of *B. bigemina* (Zanet et al., 2014).

Experimental *B. divergens* infections have been established in splenectomised deer (Cervidae), mouflon and domestic sheep (Caprinae) (Malandrin et al., 2010).

Babesia bovis have been recovered from roe, red and white-tailed deer (Cervidae) and American bison (Bovidae) (Penzhorn, 2006).

Domestic Bovidae, Caprinae, Cervidae and bison are not included within the scope of this IRA.

Captive wild ruminants

A review of the literature shows few reports relating to bovine babesiosis in captive wild animal collections.

Babesia bigemina, B. bovis and *B. divergens* have been identified by molecular methods in captive cervids in the UK, Germany, Mexico and Brazil (Pastor & Milnes, 2019).

Geographical distribution

Babesia bigemina and *B. bovis* are of major importance in Africa, Asia, Australia, Central America, South America and the southern USA (Holman et al., 2011; OIE Terrestrial Manual, 2018e).

Bovine babesiosis is restricted to certain zones/regions in Australia and is a notifiable disease (WAHIS, 2019a).

Babesia divergens is economically important in parts of Europe (OIE Terrestrial Manual, 2018e).

According to the WAHIS interface, bovine babesiosis has never occurred in Canada or Singapore (WAHIS, 2019d).

The disease is present in the UK, Europe, the UAE and SA (WAHIS, 2019d).

Pathogenesis

In experimental conditions, the incubation period is 4 to 5 days in *B. bigemina* and 10 to 12 days in *B. bovis*. Incubation periods under natural conditions range from 8 to 15 days (de Vos et al., 2004; Zaugg & Kuttler, 1987).

Following a tick bite, *Babesia* are injected into the blood vessels and enter the erythrocytes of the animal. The organisms then multiply and infect more erythrocytes.

In *B. bovis* infections, there is an accumulation of parasitised erythrocytes in the peripheral circulation accompanied by vascular stasis. Activation of the coagulation and complement cascade release vasoactive compounds resulting in vasodilation and circulatory stasis. Organ damage occurs due to anoxia and toxic products from organisms and damaged tissues. The parasites cause the release of numerous harmful pharmacological mediators, further intensifying the circulatory stasis and hypotension. The end result is hypotensive shock in acute disease and haemolytic anaemia in protracted cases (de Vos et al., 2004).

European breeds of cattle retain *B. bovis* infection for life. However, they remain infective to ticks for up to 2 years. Cattle with zebu genetics lose the infection within 2 years (de Vos et al., 2004).

The pathogenesis of *B. bigemina* is almost completely related to rapid and large-scale intravascular haemolysis.

Cattle infected with *B. bigemina* only remain infective to ticks for 4 to 7 weeks. Infections rarely persist for more than 1 year.

European, Sanga and zebu cattle are susceptible to infection and develop latent infections after recovery that may persist for various lengths of time (de Vos et al., 2004).

Cape and water buffalo can develop latent infections (de Vos et al., 2004).

The experimental study by Benitez et al. (2018) demonstrated that water buffaloes raised in *B. bovis* enzootic areas do not succumb to clinical babesiosis, unlike domestic bovines that were infected with the same *Babesia* isolates. The parasitaemia in the buffaloes was much lower than in domestic bovids and could only be detected by molecular methods and serology. The author concluded that these animals (buffalo) possess an innate immune mechanism that is able to reduce or eliminate the *B. bovis* parasite from its circulation (Benitez et al., 2018).

There have been serological and transitory infections in animals other than domestic bovids. However, there is little evidence to justify other animals as important reservoirs of *B. bigemina* and *B. bovis* (de Vos et al., 2004).

Clinical signs

The clinical signs observed in both *B. bigemina* and *B. bovis* are similar, but the course and outcome may differ (de Vos et al., 2004).

Signs in domestic ruminants usually appear 2 to 3 weeks after a bite from an infected tick.

In acute disease animals experience a pyrexia for several days which is followed by inappetence, lethargy, depression and weakness. Haemoglobinuria and diarrhoea are present. Anaemia and icterus develop in protracted cases. Cows may abort. Muscle wasting, tremors and recumbency occur, followed by coma in the terminal stages. Affected animals may die or take several weeks to recover.

Cerebral babesiosis develop in some *B. bovis* infections and this is accompanied by neurological signs.

In subacute disease, clinical signs are less severe and often difficult to identify.

In *B. bigemina* infections, the clinical course is more rapid with sudden anaemia, icterus and death. Recovery is often rapid (de Vos et al., 2004).

Transmission

Bovine babesiosis is a non-contagious tick-borne disease.

Competent vectors of *B. bigemina* are *Rhipicephalus decoloratus, R. microplus, R. e. evertsi*, *R. bursa* and *R. annulatus*.

Vectors of *B. bovis* are *R. microplus,* and vectors of *B. divergens* are *Ixodes ricinus* (de Vos et al., 2004; Zintl et al., 2003).

Most competent tick species can transmit the parasite transovarially, and a few may be able to transmit transstadially as well (de Vos et al., 2004).

New Zealand has various ticks of the genus *Ixodes*, none of which have been demonstrated to be competent vectors of the causative agents of bovine babesiosis.

The cattle tick *Haemaphysalis longicornis* which is present in New Zealand, is a competent vector of other *Babesia* spp. (*B. ovata* and *B. major*) and suspected to be a vector of *B. bigemina* (Heath, 2002).

Transplacental transmission of *B. bigemina* and *B. bovis* has been demonstrated in domestic ruminants (cattle) (Costa et al., 2016).

Diagnosis

The OIE-recommended tests for agent identification are microscopic examination and PCR. Microscopic examination is preferred for the confirmation of clinical cases, and PCR is preferred for individual animal freedom from infection prior to movement and confirmation of clinical cases.

Enzyme linked immunosorbent assay is recommended for population freedom from infection, eradication and surveillance, and determining the immune status of animals' post vaccination. Immunofluorescent antibody tests can also be used to determine the immune status of animals (OIE Terrestrial Manual, 2018e).

Treatment, control and prevention

There are various drugs that have been used to treat clinical cases of bovine babesiosis. These include diamidine, quinoline and acridine derivatives. Diamidines have been reported as being safe and effective. However, they produce residues, and this is a concern for export markets. Infected animals require treatment in the early stage of disease, or else supportive therapy is required if animals are to survive.

Short-term control and prevention of bovine babesiosis may be obtained by the use of babesiacides (imidocarb and diminazene) (de Vos et al., 2004).

In countries affected with bovine babesiosis, measures include tick control, vaccination, treatment of clinical cases, chemoprophylaxis and genetic resistance.

Using a single method of control is often not effective or cost efficient. Each method has its own advantages and disadvantages. As a result, a combination of the strategic use of acaricides to manage tick numbers, vaccination of animals in enzootically unstable environments and encouraging the use of tick-resistant cattle breeds should be implemented.

Two methods of tick control can be applied – intensive or strategic.

Cattle develop strong immunity after a single infection with *B. bigemina* and *B. bovis*. As a result, live attenuated vaccines have been used to immunise cattle.

In bovine babesiosis-free countries, tick control of imported animals and preventing entry of *Babesia* infected animals is a preventative strategy.

Semen

There is no published evidence demonstrating the presence of *Babesia* spp. in the semen of domestic or wildlife ruminants, and there is no published evidence demonstrating the transmission of the agent to naïve dams if inseminated with *Babesia* spp.-contaminated semen.

Semen contaminated with blood may contain *Babesia* in erythrocytes. However, transmission through AI has not been demonstrated (Hare, 1985).

The OIE Code does not consider germplasm a risk commodity.

29.1.6 Hazard identification conclusion

Bovine babesiosis is an OIE-listed disease of cattle.

Babesia bigemina and *B. bovis* have a high degree of host specificity to domestic cattle. Molecular and serological evidence of *B. bigemina* and *B. bovis* has been reported in Cervidae species. *Babesia bovis* has also been recovered from Cervidae and bison. These species are not covered within the scope of this IRA.

There is a single report of DNA evidence of *B. bigemina* and *B. bovis* in free-ranging nilgai. However, the organism itself was not identified.

An experimental study in wild Bovidae concluded that these animals do not succumb to infection or harbour detectable parasitaemia, as a result of their efficient innate immune mechanisms that can clear or eliminate parasites. This may be true for other wildlife ruminants.

There are no known reported cases of bovine babesiosis in zoos or other captive animal collections in species within the scope of this IRA.

Following literature review, there is insufficient evidence to suggest that captive wild Bovidae, Giraffidae or Tragulidae play a significant epidemiological role in bovine babesiosis or that these animals are capable of carrying these organisms for extended periods.

There is no published evidence demonstrating the presence *B. bigemina, B. bovis or B. divergens* in semen of wildlife ruminants.

Babesia bigemina, B. bovis and *B. divergens* are not identified as hazards in captive wild Bovidae, Giraffidae, Tragulidae and their semen and will not be assessed further.

These animals may, however, carry Babesia *spp.-infected ticks, and therefore, risk management measures relating to external parasites should be enforced.*

30 Theileriosis

30.1 Technical review

30.1.1 Aetiological agent

Family: *Theileriidae*
Genus: *Theileria*
Species: *Theileriosis annulata, Theileriosis parva*

Theileria spp. are obligate intracellular protozoan parasites (OIE Terrestrial Manual, 2018x). For the purposes of this IRA, only *T. annulata* and *T. parva* have been identified as hazards in the preliminary hazards list, and only these species will be assessed.

Theileriosis annulata (T. annulata), Theileriosis parva (T. parva) cattle-derived and *T. parva* buffalo-derived are the causative agents of Mediterranean theileriosis, East Coast fever (ECF) and corridor disease, respectively.

The epidemiology of ECF and corridor disease are similar, and therefore, only ECF will be discussed in detail.

30.1.2 OIE list

Theileriosis is an OIE-listed disease of cattle (OIE, 2020d).

30.1.3 New Zealand status

New Zealand is free from theileriosis (caused by *T. annulata* and *T. parva*) (WAHIS, 2019d), as reported to the OIE.

Theileria orientalis is present in New Zealand cattle.

Mediterranean theileriosis and ECF are notifiable diseases under the Biosecurity (Notifiable Organisms) Order 2016 primarily affecting cattle.

30.1.4 Zoonotic potential

There is no published evidence that *T. annulata* or *T. parva* are zoonotic.

30.1.5 Epidemiology

Host range

Most *Theileria* species exhibit some form of host or vector specificity (Pienaar et al., 2019). However, various species of *Theileria* can cause disease in a range of domestic and wild ruminants (Mans et al., 2015).

T. annulata
Theileria annulata can infect cattle and yaks.

Wild ruminants affected by *T. annulata* include water buffaloes (OIE Terrestrial Manual, 2018x). These animals are the natural hosts of this parasite.

T. parva
Theileria parva is an original parasite of Cape buffalo that has adapted to cattle.

A prevalence survey of buffalo, bushbuck, eland and impala (Bovidae) in a national park in Uganda, revealed that *T. parva* was not detected in the bushbucks, elands and impalas, but was found in 85% of the buffaloes sampled. Other species of *Theileria* were identified in the antelopes (Oura et al., 2011).

Another study of the prevalence of *T. parva* in waterbucks revealed that none of the 26 waterbucks sampled in an ECF enzootic area in Kenya were positive for *T. parva* (Githaka et al., 2014).

Historically, there have been numerous reports of theileriosis in antelope species. However, many of these reports classified cytauxzoonosis as theileriosis. These reports therefore cannot be used as evidence of theileriosis in antelope. Furthermore, there is an equal number of reports of theileriosis in wildlife ruminants, but the *Theileria* have not been identified to species level (Clift et al., 2020; Nijhof et al., 2005).

There are no known reports of *T. annulata* and *T. parva* in species of the Giraffidae and Tragulidae families.

Captive wild ruminants

There is no published evidence of *T. annulata* or *T. parva* infections in captive wild animal collections.

Experimental infection of captive-bred *Theileria*-free waterbucks with *T. parva* resulted in parasitosis (Stagg et al., 1994).

Geographical distribution

T. annulata

Theileria annulata occurs in southern Europe, North Africa and Asia (OIE Terrestrial Manual, 2018x).

Theileria annulata is absent from Australia.

T. parva

Theileria parva occurs in 13 countries in sub-Saharan Africa including SA (OIE Terrestrial Manual, 2018x).

Theileria parva is absent from Australia.

Theileriosis has a seasonal occurrence that is dependent on the tick vectors (Lawrence et al., 2004).

Pathogenesis

The life cycle of *T. annulata* and *T. parva* are similar.

Piroplasms of *T. parva* in the erythrocytes of animals are ingested with the blood meal of ticks. Development of the sexual stages occurs within the gut of the tick. When the tick moults, the zygotes migrate into the salivary glands and develop into sporozoites during the next feeding stage of the tick. The sporozoites are inoculated with tick saliva into the animals' skin and enter lymphocytes. Further development occurs in the lymphocytes, forming schizonts. Schizonts stimulate the development of the lymphocytes into lymphoblasts and divide with host cells. Initially, schizonts are called macroschizonts, then microschizonts, from which merozoites are liberated. Merozoites invade erythrocytes and are called piroplasms, thus completing the life cycle.

In *T. annulata* and *T. parva* infections, sporozoites invade cells at the site of inoculation or migrate to the draining lymph nodes. Sporozoites infect both T and B lymphocytes and stimulate the proliferation of infected and non-infected cells. Large numbers of hyperplastic, infected and non-infected lymphoblasts enter the peripheral circulation. They establish in lymph nodes, lymph tissue (thymus, spleen) and parenchymatous organs (liver, kidneys, lungs and adrenals), bone marrow and others. Lymphocyte proliferation is followed by lymphocyte destruction. Destruction leads to a fall in serum immunoglobulin levels and immunosuppression.

In non-fatal cases, a protective immune response terminates schizont proliferation. Recovery is dependent on the survival of the number of effector cells over the first 14 days and their ability to achieve a protective response (Lawrence et al., 2004; Pipano & Shkap, 2004).

Some *T. parva* strains have the ability to produce a carrier state in recovered cattle.

A carrier state in waterbuck was demonstrated after experimental infection with *T. parva* (Stagg et al., 1994).

Water buffaloes may act as reservoir hosts of *T. annulata* infections for ticks.

Clinical signs

The incubation period under natural conditions in Mediterranean theileriosis and ECF ranges from 8 to 25 days with an average of 15 days.

T. annulata

The peracute form occurs in highly susceptible breeds and lasts 3 to 5 days ending in death.

The acute form is most common, lasting 1 to 2 weeks with prominent clinical signs. Mortality is high.

The subacute form is similar to the acute form with less severe clinical signs.

The chronic form is quite rare. Cattle become recumbent, emaciated and exhibit intermittent fever. Death ensues within 2 to 3 weeks (Pipano & Shkap, 2004).

Initial signs of Mediterranean theileriosis in domestic ruminants include fever, swelling of superficial lymph nodes and increase in heart and respiratory rates. This is followed by anorexia, reduced rumen movement, dehydration and constipation. Diarrhoea may occur in young animals. Urine is dark brown from the presence of bilirubin. There is a drop in milk production in dairy cattle.

Cattle that are infected with virulent strains but have strong resistance do not exhibit clinical signs or detectible parasites. This is called an immediate latent infection.

Mortality is 20% to 90% depending on the resistance of cattle and virulence of the parasite (Pipano & Shkap, 2004).

Water buffaloes show no apparent signs when infected with *T. annulata.*

T. parva
The classical form of ECF is characterised by pyrexia, enlargement of superficial lymph nodes, pulmonary oedema and emaciation. Death is usually the end result. Other clinical signs are similar to those of Mediterranean theileriosis.

The severity of ECF is dependent on virulence of the *Theileria* strain, sporozoite infection rates in ticks and host susceptibility.

Mortality of cattle in ECF may reach 90% if uncontrolled.

In water buffaloes, clinical signs are similar to domestic ruminants, while signs are subclinical or mild in African buffaloes (Lawrence et al., 2004).

Experimental infection of captive-bred *Theileria*-free waterbucks resulted in mild infections with sporadic schizont parasitosis and sporadic piroplasm parasitosis. These animals were inoculated with doses of sporozoites that were lethal to cattle (Stagg et al., 1994).

Transmission
Mediterranean theileriosis and ECF are non-contagious tick-borne diseases.

T. annulata
Mediterranean theileriosis is transmitted by ticks of the genus *Hyalomma* (OIE Terrestrial Manual, 2018x).

T. parva
East Coast fever is transmitted principally by *Rhipicephalus appendiculatus*. However, other species of the genus *Rhipicephalus* and *Hyalomma* have the ability to transmit *T. parva* (Lawrence et al., 2004).

Only transstadial transmission has been described for these tick species (Lawrence et al., 2004; Pipano & Shkap, 2004).

Ticks of the genus *Rhipicephalus* and *Hyalomma* are not present in New Zealand. None of the ticks that are present in New Zealand have been described as competent vectors of *T. annulata* or *T. parva*. However, *Haemaphysalis longicornis* is a competent tick vector of *T. orientalis* (McFadden et al., 2013). *Haemaphysalis longicornis* has not been associated with the transmission of *T. annulata* or *T. parva* (Heath, 2002).

Thus, currently, there are no known competent tick vectors of Mediterranean theileriosis or ECF in New Zealand.

Diagnosis
In acute cases of theileriosis, diagnosis is based on clinical signs, knowledge of the disease, vector distribution and examination of stained blood, lymph node and tissue impression smears (OIE Terrestrial Manual, 2018x).

The OIE recommended tests for agent identification are microscopic examination and PCR. Microscopic examination is preferred for individual animal freedom from infection prior to movement and confirmation of clinical cases, and PCR is used for the confirmation of clinical cases.

Enzyme linked immunosorbent assay is recommended for surveillance. Immunofluorescent antibody titre test can be used for individual animal freedom from infection prior to movement and surveillance.

A combination of ELISA and PCR may enhance the ability to identify infected animals (OIE Terrestrial Manual, 2018x).

Treatment, control & prevention

In the 1950s, tetracyclines were moderately effective if administered in the incubatory period but were not completely reliable. In the 1970s, it was discovered that parvaquone was highly effective but did not cure the animal of parasites. Some animals remained intermittent carriers and took a protracted time to return to normal health. As a result, no infected animals may be treated chemotherapeutically. These and other drugs used to treat theileriosis are expensive, thus limiting field use.

In countries affected with theileriosis, measures include strategic tick control and vaccination.

Susceptible cattle can be kept in an infected area as long as they are kept free of *R. appendiculatus* ticks. Various acaricides have been used with short dipping intervals as well as local application of acaricides to tick predilection sites.

Vaccination is in the form of the “infection and treatment” method. Protective immunity persists for at least 5 years after natural infection with *T. parva*. As a result, the infection and treatment method was derived. Cattle are inoculated with *T. parva* sporozoites derived from ticks or *T. annulata* schizont-infected cells. Treatment with antibiotics or buparvaquone needs to be implemented during the incubation period or at the time of stabilate inoculation. Immunity is usually lifelong, and immunised animals may become carriers (Lawrence et al., 2004; OIE Terrestrial Manual, 2018x).

Another less effective method is isolation of susceptible animals from pastures and animals that may be carrying infected ticks. This method is prone to breakdown and should be used in conjunction with other control strategies (Lawrence et al., 2004).

In theileriosis-free countries, tick control of imported animals and preventing entry of *Theileria*-infected animals can be a preventative strategy.

Semen

There is no published evidence demonstrating the presence of *Theileria* spp. in semen of domestic or wildlife ruminants, and there is no published evidence demonstrating the transmission of the agent to naïve dams if inseminated with *Theileria* spp.-contaminated semen.

Semen contaminated with blood may contain *Theileria*. However, transmission through AI has not been demonstrated (Hare, 1985).

The OIE does not consider germplasm a risk commodity.

30.1.6 Hazard identification conclusion

Theileriosis is an OIE-listed disease of cattle.

Theileria spp. have a degree of host and vector specificity. *Theileria annulata* has only been reported in cattle, yaks and water buffaloes. *Theileria parva* has been reported in cattle, Cape and water buffaloes and experimental infections of waterbucks.

Although *Theileria* parasites were sporadically detected in the blood of experimentally infected waterbucks and could produce carrier status, these animals do not play an important role in the epidemiology of *T. parva*. With the exception of waterbucks, none of the above species fall within the scope of this IRA. There has been no evidence of natural *T. annulata* or *T. parva* infections in species covered in this IRA.

There have been no known reported cases of theileriosis caused by *T. annulata* or *T. parva* in zoos or other captive animal collections involving species within the scope of this IRA.

A review of the literature found no strong evidence to suggest that captive wild Bovidae, Giraffidae or Tragulidae play a significant epidemiological role in Mediterranean theileriosis or ECF.

There is no published evidence demonstrating the presence of *T. annulata* or *T. parva* in the semen of wildlife ruminants.

Theileria annulata and *T. parva* are not identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae and their semen and will not be assessed further.

These animals may, however, carry Theileria-*infected ticks, and therefore, risk management measures relating to external parasites should be enforced.*

31 Trypanosomosis (surra and tsetse fly associated trypanosomosis)

31.1 Technical review

31.1.1 Aetiological agent

Family: *Trypanosomatidae*
Genus: *Trypanosoma*
Species: *Trypanosoma evansi*, *T. brucei, T. brucei gambiense, T. brucei rhodesiense, T. congolense, T. vivax*

Trypanosomes are flagellate extracellular protozoan parasites (OIE Terrestrial Manual, 2018y).

Trypanosoma evansi is the causative agent of surra (OIE Terrestrial Manual, 2018y).

Trypanosoma congolense, T. simiae, T. vivax, T. uniforme, T. b. brucei, T. b. gambiense, T. b. rhodesiense, T. evansi and *T. equiperdum* are the causative agents of tsetse fly associated trypanosomosis (OIE Terrestrial Manual, 2018a).

For the purposes of this IRA, only trypanosomes *T. evansi, T. b. brucei, T. b. gambiense, T. b. rhodesiense, T. congolense* and *T. vivax* will be assessed, as they are associated with wildlife ruminant species covered within the scope of this IRA.

31.1.2 OIE list

Surra
Surra is an OIE-listed disease affecting multiple species.

Tsetse fly associated trypanosomosis
Tsetse fly associated trypanosomosis is an OIE-listed disease affecting cattle (OIE, 2020d).

31.1.3 New Zealand status

New Zealand is free from surra and tsetse fly associated trypanosomosis (WAHIS, 2019d), as reported to the OIE.

Trypanosomosis (caused by *Trypanosoma* spp.) is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

31.1.4 Zoonotic potential

Surra
Trypanosoma evansi is not known to be zoonotic (OIE Terrestrial Manual, 2018y). However, there was a single reported case in a man in India. The man had a rare genetic mutation that caused him to be susceptible to *T. evansi* infection (Lun et al., 2009; Powar et al., 2006).

Tsetse fly associated trypanosomosis
Of the species that cause tsetse fly associated trypanosomosis, *T. b. gambiense* and *T. b. rhodesiense* are zoonotic (OIE Terrestrial Manual, 2018a). They cause chronic or acute “sleeping sickness” in humans, respectively (World Health Organization, 2020).

31.1.5 Epidemiology

Host range

Surra
Trypanosoma evansi has the widest host range among the sylvarian trypanosomes. Experimentally, almost all mammals are receptive to *T. evansi* infections. However, only some animals develop clinical disease (Desquesnes et al., 2013a).

Host species vary geographically. Domestic species affected include but are not limited to camels, horses, pigs, dogs, llamas and cattle (OIE Terrestrial Manual, 2018y).

Wildlife ruminants susceptible to surra include water buffaloes.

Trypanosoma evansi has been isolated in Cape buffaloes, antelopes (Bovidae) and various species of deer (Cervidae) (Desquesnes et al., 2013a; Tarello, 2003).

There have been cases in captive wild animals such as bears (Ursidae), rhinoceroses (Rhinocerotidae) and felines (Felidae) in South Asia, Asia and India, respectively (Moudgil, 2015; Muhammad et al., 2007; van Strien & Talukdar, 2007).

It is noteworthy that buffaloes and Cervidae are not covered within the scope of this IRA.

Tsetse fly associated trypanosomosis
Tsetse fly associated trypanosomosis can affect a wide range of wild and domestic mammals. However, the disease is of significant economic concern in cattle.

Susceptible hosts include horses, donkeys, camels, pigs, dogs, goats, sheep, cattle, humans and various wild animals (OIE Terrestrial Manual, 2018a).

Due to the wide host range of *Trypanosoma* spp., it is assumed that species within the Giraffidae and Tragulidae families would be susceptible to infection.

Captive wild ruminants

Surra
Cases of surra in captive wild ruminants (gazelles) have been reported in the UAE (Tarello, 2003).

Tsetse fly associated trypanosomosis
Studies have suggested that captive wild animals are more likely to succumb to clinical trypanosomosis than their wild counterparts due to the added levels of stress experienced in captivity (Mbaya et al., 2009). This scenario is more likely to occur in cases where wild animals are brought to captivity, rather than in captive wild animals born and bred in captivity.

Trypanosoma brucei, T. congolense and *T. vivax* have been identified in captive wild Bovidae (duikers, kobs, gazelles, sitatunga and red-fronted gazelles) at Nigerian zoos (Mbaya et al., 2008).

Geographical distribution

Surra
Surra has been reported in Africa, Asia, Central America, South America, parts of Europe and the UAE.

Surra is absent from Australia and has never occurred in the UK, the USA, Canada, Singapore, Japan or the RSA (WAHIS, 2019d).

Tsetse fly associated trypanosomosis
Tsetse fly associated trypanosomosis occurs in Africa and parts of the Arabian Peninsula, South America and Central America.

According to the WAHIS interface, tsetse fly associated trypanosomosis has never occurred in Australia, the UK, the USA, Canada, Singapore or the UAE.

Pathogenesis

Surra
There is a large degree of variation in pathogenicity of different strains of *T. evansi* as well as variation in host susceptibility (OIE Terrestrial Manual, 2018y).

The incubation period is 5 to 60 days. However, longer periods of over 3 months have been reported.

Morbidity rates can reach 50–70% in affected countries with similar mortality rates. Mortality is low in enzootic areas but higher when animals are moved from non-enzootic to enzootic areas.

Infected haematophagous flies feed on healthy susceptible animals, thus injecting the trypanosomes into the peripheral blood. Multiplication of the trypanosomes in the blood and extracellular fluid results in parasitaemia accompanied by anaemia.

The cause of anaemia is not completely elucidated. The first phase of anaemia is, however, haemolytic in nature resulting from erythrophagocytosis. The second phase may be due to impaired erythropoiesis and erythrophagocytosis.

Leukopenia has been reported, and this is likely due to reduced myelopoiesis (Silva et al., 1995).

The parasite also has the ability to cause immunosuppression in affected animals. This allows the parasite to replicate while evading the host's immune system. This also causes the animal to become susceptible to other infections (Desquesnes et al., 2013a). Trypanosomes have the ability to evade the host's immune systems by changing their surface coat (Horn, 2014).

A carrier status has been alluded to in that animals affected mildly or subclinically have the ability to harbour the parasite in the absence of clinical disease.

Wildlife reservoirs include deer, wild pigs, capybaras (Desquesnes et al., 2013a) and vampire bats (Hoare, 1965).

Tsetse fly associated trypanosomosis
The pathogenesis in tsetse fly associated trypanosomosis is similar to surra.

The *Trypanosoma* spp. causing significant disease in domestic ruminants are *T. congolense* (most pathogenic), *T. vivax* (most prevalent) and *T. b. brucei* (Bengaly et al., 2002).

The incubation period ranges from 4 to 60 days (Dagnachew et al., 2015; Wellde et al., 1989).

Morbidity and mortality are influenced by the health status of the animal, strain of trypanosome and infectious dose.

A carrier status has been described in various animals especially in *T. vivax* infections (Rodrigues et al., 2015). Animals may develop subclinical infections or become chronic carriers.

Clinical signs

Surra
Surra is characterised by acute or chronic forms of the disease. Chronic disease may persist for months or even years.

Clinical disease is most pronounced in horses, camels and dogs while disease is mild to subclinical in sheep, goats and pigs. The severity of the disease in deer is variable (Desquesnes et al., 2013a).

Clinical disease in domestic ruminants is characterised by pyrexia accompanying parasitaemia. This is followed by progressive anaemia, loss of body condition and lethargy.

Some animals may experience oedema of the ventral aspects of the body, urticarial plaques and petechial haemorrhages of the serous membranes.

In advanced cases, parasites may enter the central nervous system, resulting in neurological signs (OIE Terrestrial Manual, 2018y).

Buffaloes and cattle usually develop mild or subclinical infections, but the disease may prove fatal in these species as well. These animals may develop parasitaemia in the absence of anaemia (Dargantes et al., 2005).

Trypanosoma evansi is non-pathogenic in Cape buffaloes due to a trypanocidal factor in the serum. Eland serum showed the same characteristic (Reduth et al., 1994).

Water buffaloes in the Philippines have been reported to suffer from either wasting sickness that lasts weeks to months or acute disease that results in death within hours (Dargantes et al., 2009).

Surra may also be the cause of late gestation abortion and stillbirth in water buffaloes (Desquesnes et al., 2013a; Löhr et al., 1986).

Tsetse fly associated trypanosomosis
The disease is most often chronic. However, acute disease resulting in mortality within a few weeks may also occur.

Clinical signs are similar to those seen in surra and include intermittent fever, anaemia, oedema, abortion, decreased fertility and emaciation. Anaemia is often followed by loss of body condition, reduced productivity and often mortality.

Trypanosomes cause immunosuppression in animals (OIE Terrestrial Manual, 2018a).

A haemorrhagic syndrome has been described in domestic ruminants infected with *T. vivax*. Animals develop widespread haemorrhages and severe anaemia that often leads to death (Gardiner et al., 1989).

Captive wild Bovidae at the Maiduguri Zoological Garden in Nigeria showed no clinical signs of trypanosomosis although the survey revealed the presence of trypanosomes in these animals (Mbaya et al., 2008).

However, clinical trypanosomosis was reported in red-fronted gazelles at 2 Nigerian zoos. Disease was attributed to the stress of captivity (Mbaya et al., 2008).

Transmission

Surra
Surra is an arthropod-borne disease. However, vertical, horizontal, iatrogenic and oral transmission has been reported.

The most significant route of transmission is mechanical, via biting insects. This includes haematophagous flies (tabanids and *Stomoxys* spp.).

Experimental transmission of *T. evansi* by *Aedes* spp. and *Anopheles* spp. has been successful. However, the significance of these vectors is yet to be determined (Desquesnes et al., 2013b; OIE Terrestrial Manual, 2018y).

Trypansoma evansi can be transmitted orally, especially if there are lesions in the mucous membranes of the oral cavity. This is more applicable to carnivorous animals (Desquesnes et al., 2013b).

In Brazil, vampire bats are reported to be biological vectors. Bats have the ability to become infected with *T. evansi*: after feeding off infected hosts, the bat becomes infected, and the parasite multiplies in the bat. Bats can either become ill and die or experience subclinical or chronic infection and become a reservoir of *T. evansi*. They can then transmit infection to other susceptible hosts including other bats (Desquesnes, 2004; Hoare, 1965).

New Zealand has 2 native species of bat, but they are not vampire bats. These bats feed on insects, fruit and flowers.

Tsetse fly associated trypanosomosis
Tsetse fly associated trypanosomosis is transmitted cyclically by flies of the genus *Glossina* (tsetse flies). These flies are biological vectors that allow trypanosomes to develop for 1 to 2 weeks into the infective stage.

Mechanical transmission by haematophagous flies (tabanids and *Stomoxys* spp.), surgical instruments, needles and syringes has been reported in the transmission of *T. vivax*. This is the primary route of transmission in South America. *Trypanosoma vivax* has a short life cycle, which can be completed in the fly's mouth parts, thus allowing mechanical transmission.

Transplacental transmission has been reported (Silva et al., 2013).

Tsetse flies are responsible for the transmission of *T. b. gambiense* and *T. b. rhodesiense* from infected animals or humans to susceptible humans, causing “sleeping sickness” (World Health Organization, 2020).

Regarding trypanosome transmission in general, the following should be noted:
The survival of *T. vivax* in tabanids was reported to be 30 minutes and shorter in *Stomoxys* spp. during immediate transmission (Desquesnes et al., 2013b). Experimental evidence shows that transmission via biting flies is most efficient if there is a short time lapse between feeding on animals. Therefore, transmission is more likely to occur between animals in the same enclosure (in zoos) or those that are grazing together (Mihok et al., 1995).

In delayed transmission, trypanosomes can survive in tabanid and *Stomoxys* spp. stomachs for longer periods. In some cases, successful transmission was reported after 72 hours. However, given the persistent feeding behaviour of tabanids, they are more likely to seek hosts immediately to complete the blood meal. Once satisfied they will only feed again after 5 to 7 days. Trypanosomes will not survive in the gut for this period. Therefore, the likelihood of delayed transmission in tabanids is very low.

On the other hand, due to their feeding behaviour, *Stomoxys* spp. flies have the ability to transmit trypanosomes 4 to 48 hours after feeding, suggesting that infection can be transmitted between herds/animals in the absence of close contact (Desquesnes et al., 2013b).

The incidence of transmission is related to the level of parasitaemia of the host, the density of vectors feeding on the animals and the size of the vector (Desquesnes et al., 2013b).

Sucking flies (*Musca* spp.) have been shown to transmit trypanosomes by contamination of wounds.

Other means of transmission may include contamination of wounds and iatrogenic transmission by non-sterile surgical equipment or needles (Desquesnes et al., 2013b).

Sexual transmission or grooming may result in direct horizontal transmission. More research is required to prove these theories.

Vertical transmission in the form of transplacental infections has been described (Desquesnes et al., 2013b; Ogwu & Nuru, 1981; Silva et al., 2013). This route of transmission may result in persistence of the disease within the population.

Diagnosis

Surra

Confirmation of surra requires laboratory diagnosis as clinical signs are not pathognomonic.

In acute disease when there is high parasitaemia, wet blood films, stained blood smears or lymph node material can be used to identify the trypanosomes. In chronic cases during low parasitaemia, thick blood smears, concentration methods (haematocrit centrifugation technique (HCT)) and animal inoculation may be used.

Polymerase chain reaction assays are available. These assays are more sensitive than parasitological examination but may produce false negatives during periods of low parasitaemia. Therefore, serological methods are more appropriate to identify animals that are carriers or chronically infected.

There are a number of serological tests available for use. ELISA may be used for surveillance. Card agglutination tests (CATT/*T. evansi*) can be used to target individual animals that are infected and require treatment.

Where a definitive confirmation of an animal’s status is required, mouse inoculation is the most appropriate test. However, animal testing must be justified.

If individual animals require disease-free status declarations, serial testing using CATT and ELISA should be used. Testing can be performed with an interval of 40 days. It is recommended that any suspicious samples be retested and confirmed by PCR (OIE Terrestrial Manual, 2018y).

Tsetse fly associated trypanosomosis

There are numerous OIE recommended tests for the diagnosis of tsetse fly associated trypanosomosis. It is recommended that a combination of agent identification methods be applied to the same clinical samples to increase sensitivity.

Thin stained blood smears, PCR and HCT can be used for the confirmation of clinical cases. Polymerase chain reaction and HCT are also recommended for population and individual animal freedom from infection, surveillance and eradication, and determining the immune status of the population and individual animals post vaccination.

In terms of serological tests, ELISA is recommended for population and individual animal freedom from infection, surveillance and eradication (OIE Terrestrial Manual, 2018a).

Treatment, control and prevention

Two strategies are used for controlling vector-borne diseases: pathogen control and vector control (Desquesnes et al., 2013b).

The following applies to both surra and tsetse fly associated trypanosomosis.

Pathogen control can be achieved by chemical control of parasites.

Curative and chemoprophylactic drugs or trypanocides (diminazene aceturate, isometamidium chloride, cymelarsan, suramin and quinapyramine) are used to eliminate parasites from a sick animal. These drugs have proven to be successful in case studies (Muhammad et al., 2007; Reid & Copeman, 2002; Tarello, 2003).

Several factors limit the use of these drugs. This includes overuse (resulting in resistance), availability, cost effectiveness, practicality of administering the drugs, lack of regulated drug usage and lack of production of high-quality drugs.

Vector control is part of the preventative strategy. In tsetse fly associated trypanosomosis in Africa, control is implemented by reducing the tsetse fly pressure. This can be achieved by using insecticide impregnated traps and insect sterilisation techniques.

Control of mechanical vectors like tabanids and *Stomoxys* spp. is more difficult to achieve. However, certain traps, spraying animals with insecticide and using smoke and nets could help.

Vector control can be expensive if used on a large scale and may not be effective or sustainable (Giordani et al., 2016).

No vaccines are currently available.

In countries that are free from trypanosomes, restricting importation and movement of infected animals from trypanosomosis areas remains the most effective means of prevention.

Semen

Surra

In an experimental study with sheep, rams were intravenously infected with *T. evansi* and semen samples tested for trypanosomes. Rams were mated to ewes to determine whether sexual transmission occurred. The study revealed that ewes became pregnant with no evidence of infection and the semen showed no evidence of trypanosomes (Da Silva et al., 2016).

Tsetse fly associated trypanosomosis

Venereal transmission has been suggested after the detection of nucleic acid of *T. vivax* in the semen of goats that were experimentally infected (Bezerra et al., 2018). Presence of the parasite in semen is yet to be proven.

Numerous studies describe the adverse effects of trypanosome infections on the male reproductive organs and semen quality in various domestic and wild ruminants (Adamu et al., 2007; Mbaya et al., 2011; Raheem et al., 2009). None of these studies demonstrate the presence of trypanosomes in semen or show unequivocal evidence of venereal transmission.

There is no published evidence demonstrating the presence of trypanosomes in the semen of domestic or wildlife ruminants, and there is no published evidence demonstrating the transmission of the agent to naïve dams if inseminated with trypanosome-contaminated semen. In the absence of such evidence, extrapolation is made from domestic to wildlife ruminants.

31.1.6 Hazard identification conclusion

Surra and tsetse fly associated trypanosomosis are OIE-listed diseases.

There is no published evidence demonstrating the presence of *Trypanosoma* spp. (under assessment) in semen of domestic or wildlife ruminants, and there is no published evidence demonstrating the transmission of the agent to naïve dams if inseminated with semen contaminated with *Trypanosoma* spp. (under assessment).

Trypanosoma spp. (causing surra and tsetse fly associated trypanosomosis) are not identified as hazards in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Trypanosoma spp. (causing surra and tsetse fly associated trypanosomosis) are identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae.

31.2 Risk assessment

Since surra and tsetse fly associated trypanosomosis are caused by protozoa of the same genus, with multiple similarities relating to epidemiology, the entry, exposure and consequence assessments will be combined where necessary.

31.2.1 Entry assessment

Surra is absent from Australia and has never occurred in the UK, the USA, Canada, Singapore, Japan or the RSA. Tsetse fly associated trypanosomosis has never occurred in Australia, the UK, the USA, Canada, Singapore or the UAE. Disease may be present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Due to the low volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Surra and tsetse fly associated trypanosomosis has been reported in domestic and wild ruminants in some approved countries (UAE, the RSA and countries in Europe).

There are limited reports of these diseases in captive wild ruminants. The lack of cases in ruminants could be either due to a lack of reporting or because captive wild ruminants are subclinically infected. It has been mentioned that animals in captivity are more likely to succumb to clinical disease as a result of stressful conditions. This may relate only to wild animals brought to captivity.

Both surra and tsetse fly associated trypanosomosis can have long incubation periods (2 to 3 months). If captive wild ruminants within the scope of this IRA are incubating the disease or are subclinical, latent or chronic carriers not showing signs of clinical disease (surra or tsetse fly associated trypanosomosis), they may go unnoticed and be passed as clinically sound for export if they are exported from surra or tsetse fly associated trypanosomosis affected countries.

Therefore, the likelihood of entry of *Trypanosoma* spp. (causing surra and tsetse fly associated trypanosomosis) via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from surra or tsetse fly associated trypanosomosis affected countries is assessed as low.

31.2.2 Exposure assessment

Surra and tsetse fly associated trypanosomosis are arthropod-borne diseases. However, vertical, horizontal, iatrogenic and oral transmission has been reported.

The most significant route of transmission is via haematophagous flies. Both *T. evansi* and other trypanosomes can be transmitted by tabanids and *Stomoxys* spp. Experimental transmission of

T. evansi has also been demonstrated by *Aedes* spp. and *Anopheles* spp. These arthropod vectors are all present in New Zealand.

Tsetse fly associated trypanosomosis is also transmitted by tsetse flies.
Tsetse flies are also responsible for the transmission of *T. b. gambiense* and *T. b. rhodesiense* from animals and humans to susceptible humans, causing "sleeping sickness". These flies are not present in New Zealand. Therefore, the likelihood of exposure to humans is assessed as negligible.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If subclinically infected or carrier animals are imported into zoos, they would serve as a source of infection for other susceptible animals.

Effective transmission of trypanosomes from an infected animal is dependent on numerous factors, which include the level of parasitaemia of the host, the density of vectors feeding on the host, the size of the vector and the time lapse between feeding.

The level of parasitaemia is likely to be low in subclinical or chronic carriers (imported ruminants). This may change if the animal becomes stressed and succumbs to clinical disease. The density and size of vectors feeding on infected hosts would be variable. The time lapse between feeding has to be less than 30 minutes in tabanids to allow effective transmission. Therefore, only animals that are kept in the same enclosure or those in adjacent enclosures are likely to become infected.

Due to the feeding behaviour of *Stomoxys* spp*., Stomoxys* flies could transmit infection up to 48 hours after feeding. *Stomoxys* spp. and potentially *Aedes* spp. and *Anopheles* spp. are most likely the only means of transmission of trypanosomes to susceptible animals outside the zoo. However, the likelihood of this occurring is extremely low.

However, if domestic ruminants are kept at the zoo, they could become infected with *Trypanosoma* spp. Should these domestic ruminants be released onto New Zealand farms, they could be a source of infection to other domestic ruminants. Trypanosomosis could therefore establish in animals outside the zoo.

Other means of transmission such as contamination of wounds and sexual transmission would only expose those animals in direct contact with the infected animal. Iatrogenic transmission should not occur, as zoo staff usually follow aseptic techniques during procedures.

As mentioned above, both surra and tsetse fly associated trypanosomosis can affect a wide range of animals. Thus, almost all animals within the zoo are likely to be susceptible. However, only a limited number may be exposed. Since all animals are susceptible, the disease may manifest as acute or chronic disease, with the potential for subclinical, chronic or latent carriers. The disease could therefore establish in animals within the zoo.

Therefore, the likelihood of *Trypanosoma* spp. (causing surra and tsetse fly associated trypanosomosis) exposure and establishment within the zoo is assessed as low, and the likelihood of exposure and establishment outside the zoo is assessed as very low.

31.2.3 Consequence assessment

Surra and tsetse fly associated trypanosomosis are OIE-listed diseases.

There is likely to be a very small number of live captive wild ruminants or semen imported into New Zealand. These imports are also likely to be infrequent.

There may be direct consequences for the imported animals that were subclinical, chronic or latent carriers. Infections in wild ruminants are usually mild to subclinical. However, authors have reported that animals in captivity are more likely to suffer clinical disease.

If captive wild ruminants become immunocompromised due to stressful conditions either during translocation or quarantine or after importation to the zoos in New Zealand, they could succumb to clinical disease. If these animals are not treated in time, they could die.

Trypanosomes cause immunosuppression, and this could result in animals becoming more susceptible to other types of diseases or conditions. Infection has also resulted in abortions and stillbirths of some wild ruminants. Animals in New Zealand are naïve to trypanosomosis; therefore, the mortality and morbidity rates are likely to be higher for animals in New Zealand than for animals in enzootic areas. The losses experienced would adversely affect zoos' ASMP, breeding and conservation efforts.

Stomoxys spp. and potentially *Aedes* spp. and *Anopheles* spp. are the only vectors that could result in spread of trypanosomes to susceptible animals outside the zoo. Transmission would have to occur within 48 hours of feeding off an infected host to be successful. Even though this could occur, the likelihood is extremely low. Domestic ruminants kept at the zoo that may become infected and then be released to New Zealand farms could also serve as a source of infection to other domestic ruminants.

The consequences of animals outside the zoo becoming infected are variable. The pathogenicity in various animal species depends upon the strain of trypanosome and the host susceptibility. Some animals may develop mild to subclinical infections, while others may develop severe to chronic infections. High mortality and morbidity could be the result in naïve populations.

Tsetse flies are not present in New Zealand. There would be no transmission of zoonotic *Trypanosoma* spp. to humans. Therefore, the consequences for human health would be negligible.

Indirect consequences would entail costs for control and surveillance within the affected zoos. Since these are vector-borne diseases that affect a wide range of animals, testing of almost all animals within the zoo may be required. Infected animals may be either treated or euthanised.

There would be additional costs to New Zealand for surra or tsetse fly associated trypanosomosis control in the event of an incursion in domestic ruminants.

Since surra and tsetse fly associated trypanosomosis are OIE-listed diseases, an outbreak of either could have negative trade impacts. Proving that the disease was restricted to the zoo may be difficult due to vector transmission and the presence of competent vectors in New Zealand.

Therefore, the overall consequences as a result of surra or tsetse fly associated trypanosomosis incursions are assessed as moderate.

31.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for surra or tsetse fly associated trypanosomosis is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

31.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Surra and tsetse fly associated trypanosomosis are OIE-listed diseases that have never occurred in New Zealand and are nationally notifiable.
- *Trypanosoma evansi* is not known to be zoonotic.
- *T. b. gambiense* and *T. b. rhodesiense* are zoonotic but are transmitted by tsetse flies, which are absent from New Zealand.
- Surra and tsetse fly associated trypanosomosis have a wide host range.
- Surra is absent from Australia and has never occurred in the UK, the USA, Canada, Singapore or the RSA.
- Tsetse fly associated trypanosomosis has never occurred in Australia, the UK, the USA, Canada, Singapore or the UAE.
- The incubation period in surra and tsetse fly associated trypanosomosis is 4 to 60 days.
- Carrier status has been described in surra and tsetse fly associated trypanosomosis.
- Surra and tsetse fly associated trypanosomosis are arthropod-borne diseases. However, vertical, horizontal, iatrogenic and oral transmission has also been reported.

- Diagnostic tests for surra and tsetse fly associated trypanosomosis include stained blood smears, HCT, PCR and ELISA. Further tests that can be used for surra are mouse inoculation and CATT/*T. evansi*.
- Control strategies include pathogen control and vector control.
- There is no evidence to demonstrate the presence of trypanosomes in semen or the transmission of infection via AI.

31.3.1 Options

Trypanosoma spp. (causing surra and tsetse fly associated trypanosomosis) are not identified as hazards in semen, and therefore, risk management measures are not warranted.

One or a combination of the following options may be used for animals.

Option 1

1. Country freedom for surra and tsetse fly associated trypanosomosis; AND
2. the animal(s) were resident in surra and tsetse fly associated trypanosomosis free countries since birth; AND
3. the animal(s) showed no clinical signs of disease on the day of export.

Option 2

1. The exporting country had no clinical, epidemiological or other evidence of surra or tsetse fly associated trypanosomosis in any animal species in the previous 2 years; AND
2. surra and tsetse fly associated trypanosomosis are compulsorily notifiable in the country; AND
3. the animal(s) was resident in the exporting country for at least 6 months; AND
4. During the pre-export isolation, a blood sample was drawn from a peripheral vein of the animal(s) and tested using the haematocrit centrifuge technique, and a CATT was performed. The test was negative for *Trypanosoma* spp.

32 Screw-worm fly

32.1 Technical review

The technical review will refer to both fly species except where indicated.

32.1.1 Aetiological agent

New world screw-worm fly (NWS)
Family: *Calliphoridae*
Genus: *Cochliomyia*
Species: *Cochliomyia hominivorax*

Old world screw-worm fly (OWS)
Family: *Calliphoridae*
Genus: *Chrysomya*
Species: *Chrysomya bezziana*

Both fly species are obligate parasites of mammals during their larval stages. Larval stages are responsible for causing traumatic myiasis in hosts (OIE Terrestrial Manual, 2019a).

32.1.2 OIE list

Infestations of new world screw-worm fly (NWS) (*C. hominivorax*) and old world screw-worm fly (OWS) (*C. bezziana*) are OIE-listed infestations affecting multiple species.

32.1.3 New Zealand status

New Zealand is free from NWS (*C. hominivorax*) and OWS (*C. bezziana*) myiasis (WAHIS, 2019d), as reported to the OIE.

New world screw-worm fly (*Cochliomyia* spp.) and old world screw-worm fly (*Chrysomya* spp.) are notifiable organisms under the Biosecurity (Notifiable Organisms) Order 2016.

32.1.4 Zoonotic potential

Larval stages of NWS and OWS can feed on humans (OIE Terrestrial Manual, 2019a).

Young, old and debilitated individuals are more at risk of infestations, which, when severe enough, can lead to death (Aggarwal et al., 2014; Olea et al., 2014; Spradbery, 1994).

32.1.5 Epidemiology

Host range

Larval stages of both flies can infest all mammals and humans. They are rarely found on birds (OIE Terrestrial Manual, 2019a).

Wildlife ruminants also experience infestations (News Desk, 2018; Obanda et al., 2013; USDA, 2016b).

Old world screw-worm fly infestations were reported in free-ranging elands in a wildlife conservancy in Kenya (Obanda et al., 2013).

Captive wild ruminants

New world screw-worm fly (NWS)
In an outbreak of NWS in Florida, affected animals were mostly wildlife, more especially deer (Cervidae) at a deer refuge (USDA, 2016b).

Old world screw-worm fly (OWS)
Old world screw-worm fly infestations were reported in sambar deer (Cervidae) in a Singapore zoo (News Desk, 2018).

Geographical distribution

New world screw-worm fly (NWS)
New world screw-worm fly is present over most of South America, parts of central Asia and West Africa (WAHIS, 2019d).

In 1988, NWS was detected in Libya (North Africa) but was eradicated in 1991 by an intensive sterile insect technique (SIT) programme.

The use of the SIT in major programmes has resulted in eradication of NWS from the USA, Mexico, Curaçao, Puerto Rico, the Virgin Islands, Guatemala, Belize, El Salvador, Honduras, Nicaragua, Costa Rica and Panama (OIE Terrestrial Manual, 2019a).

Imported cases of NWS have been reported in Mexico, the USA and the UK. New world screw-worm fly was last reported in the USA in 2017 (OIE Terrestrial Manual, 2019a).

Myiasis caused by NWS has never occurred in Australia, the UK, Europe, Canada, Singapore, the UAE or the RSA (WAHIS, 2019d).

Old world screw-worm fly (OWS)
The OWS is distributed over most of Africa including the RSA, the Middle East Gulf region, the Indian subcontinent, Southeast Asia, the Malay Peninsula, the Indonesian and Philippine islands, Papua New Guinea (James, 1947; Sutherst et al., 1989) and Singapore.

Old world screw-worm fly was last reported in the UAE in 2000 and in the RSA in 2018. The status of Europe is unclear.

Myiasis caused by OWS has never occurred in Australia, the UK, the USA or Canada (WAHIS, 2019d).

New world screw-worm fly and OWS prefer tropical and subtropical climates (Hosni et al., 2020). Currently New Zealand lies far south of the southernmost limits of NWS and OWS distribution (Heath et al., 2016). New Zealand has a largely temperate climate, with the Far North experiencing subtropical temperatures during summer (Discover New Zealand, 2017).

Taking global warming and climate change into consideration, rising global temperatures could be expected. The future global climatic modelling by Hosni et al. (2020) predicts that the northern parts of New Zealand may allow the establishment of OWS *(C. bezziana).* The climatic condition of both screw-worm flies are similar, and their potential geographical distributions could overlap (Sutherst et al., 1989). This implies that both NWS and OWS could establish in parts of New Zealand following further global warming.

Pathogenesis
The life cycle of the NWS and OWS are similar.

The period of time between eggs being laid and disease being expressed due to burrowing larvae could be as short as 1 to 2 days.

The adult female flies lay their eggs on living animals, either around wound edges or orifices. An average first batch of eggs may contain 175 eggs for OWS and 343 for NWS (Spradbery, 1994). Natural wounds that are most often infested are navels of newborn animals and the vulval and perineal regions of their mothers, especially if traumatised. Orifices that can be infested are nostrils and associated sinuses, the eye orbits, mouth, ears and genitalia (OIE Terrestrial Manual, 2019a).

Within 12 to 24 hours, larvae emerge from eggs and burrow into underlying tissues and begin feeding. As they feed, the wound is enlarged and deepened, causing extensive tissue damage. The infested wounds produce a distinctive odour, which attracts more females, which lay more eggs (Hall, 1995).

Larvae pass through 3 larval instars to reach maturity 5 to 7 days after eggs hatch. The larvae drop off the wound to the ground, where they burrow and pupate. The pupa develop in the puparium, which is a protective structure that becomes hard and dark. Once development is complete, the adult fly emerges and works its way through the soil to the surface.

Adult males can mate within 24 hours. Females are receptive to males from 3 days old but require 6 to 7 days before the ovaries mature. Adult flies live an average of 2 to 3 weeks (OIE Terrestrial Manual, 2019a).

The rate of development of immature stages depends on environmental and wound temperature. Development is slower at lower temperatures. The complete life cycle of NWS may take 2 to 3 months in cold weather, 24 days at an average air temperature of 22°C (James, 1947) and 18 days in tropical conditions of approximately 29°C (Thomas & Mangan, 1989).

The distance adult flies can travel varies between 10 to 20 kilometres in warm humid atmospheres and up to 300 kilometres in arid environments (OIE Technical Disease Cards, 2013e).

Clinical signs

Infested animals develop large wounds at the site of larval infestation. The wounds are socket-like and circular and may become infected by secondary bacteria. Wounds may manifest draining, serosanguinous discharge or suppuration (Obanda et al., 2013; OIE Technical Disease Cards, 2013e).

On closer observation, numerous cream-coloured eggs may be seen around the wound (OIE Technical Disease Cards, 2013e).

The wounds release a characteristic odour (Obanda et al., 2013).

Animals with screw-worm fly infestations appear to be in pain or discomfort, unthrifty and depressed (Obanda et al., 2013). They may exhibit anorexia or a drop in milk production (OIE Technical Disease Cards, 2013e).

Morbidity is variable but can reach 100% in enzootic areas.

Infestations that are left untreated could result in mortality of the animal in 1 to 2 weeks (OIE Technical Disease Cards, 2013e).

Transmission

Screw-worm fly myiasis is not easily transmitted between hosts (OIE Technical Disease Cards, 2013e). The most likely route for an animal to become infested would be for an adult fly to lay eggs on the animal.

Diagnosis

Diagnostic test methods available include morphological identification, hydrocarbon analysis, mitochondrial DNA analysis and serology. However, morphology is the OIE recommended method for population freedom from infestation, individual animal freedom from infestation prior to movement, surveillance and eradication, and the confirmation of clinical cases (OIE Terrestrial Manual, 2019a).

Treatment, control and prevention

Organophosphorus (coumaphos, dichlofenthion or fenchlorphos) has been used to treat infested wounds (Spradbery, 1994).

Avermectins administered subcutaneously have been reported to kill screw-worm fly larvae (Spradbery et al., 1985).

Prevention of screw-worm fly infestations can be achieved by spraying and dipping animals. Careful handling of animals during certain procedures (sheering, tail docking, etc.) would minimise wounding. Removal of sharp objects in and around animal enclosures would prevent injury to animals. Prevention of other wound-causing parasites like ticks with ectoparasiticides decreases the likelihood of infestations (OIE Terrestrial Manual, 2019a).

The SIT has been used successfully (USDA, 2016b). The technique involves the release of a large number of sterilised male flies in the environment. The sterilised males mate with females, which produce infertile eggs. This technique should result in initial reduction of the population and eventually eradication (Alphey, 2016).

There are no vaccines for NWS or OWS.

Semen

There is no published evidence demonstrating the presence of *C. hominivorax* or *C. bezziana* in the semen of domestic or wildlife ruminants.

32.1.6 Hazard identification conclusion

New world screw-worm fly (*C. hominivorax*) and old world screw-worm fly (*C. bezziana*) are OIE-listed infestations affecting multiple species.

There is no published evidence demonstrating the presence of *C. hominivorax* or *C. bezziana* in the semen of wildlife ruminants.

New world screw-worm fly *(C. hominivorax)* and old world screw-worm fly *(C. bezziana)* are not identified as hazards in semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

New world screw-worm fly *(C. hominivorax)* and old world screw-worm fly *(C. bezziana)* are identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae.

32.2 Risk assessment

Since new world screw-worm and old world screw-worm flies have similar life cycles and epidemiology, the entry, exposure and consequence assessments will be combined.

32.2.1 Entry assessment

Myiasis caused by NWS has never occurred in Australia, the UK, Europe, Canada, Singapore, the UAE or the RSA. Myiasis caused by OWS has never occurred in Australia, the UK, the USA or Canada. Myiasis caused by NWS and OWS may be present in all other approved countries.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Due to the low volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Both new world and old world screw-worm fly have been reported in some approved countries. Larval stages can infest all mammals. There are reports of screw-worm fly infestations in captive wild ruminants.

Adult flies only lay eggs on live animals, either around wound edges or orifices. Larvae that emerge burrow into the underlying tissue and may not be clearly visible to the naked eye. Captive wild ruminants that have eggs that are hidden around orifices or larvae in the underlying tissues may go unnoticed and be passed as clinically sound for export if they are exported from NWS- and OWS-affected countries.

Larval stages complete their development in 5 to 7 days and drop off the animal. If transportation of the exported ruminants occurs in less than 5 to 7 days, animals could still be harbouring larvae.

Therefore, the likelihood of entry of NWS *(C. hominivorax)* and OWS *(C. bezziana)* via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from NWS- and OWS-affected countries is assessed as moderate.

32.2.2 Exposure assessment

Screw-worm fly myiasis is not easily transmitted between hosts. The most likely route for an animal to become infested would be for an adult fly to lay eggs on the animal.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If infested animals carrying eggs or larvae enter zoos in New Zealand and enable the completion of the NWS or OWS fly life cycles, adult flies could mate and deposit eggs on other animal species within the zoo.

Flies can live for 2 to 3 weeks and can travel distances of up to 300 kilometres in dry arid climates. Some regions in New Zealand are dry during summer. This implies that flies may be able to travel to animals outside the zoo.

Flies may also lay eggs on young, old or debilitated humans who may be visiting the zoos. The zoo staff do not fit the demographic of humans that are usually affected by NWS or OWS myiasis.

Exposure of animals and humans to NWS and OWS may therefore be probable. However, establishment of the NWS and OWS flies in New Zealand is questionable at present time. It has been mentioned that this IRA does not take into consideration future events or trends such as global warming that may result in a change of climatic conditions.

Both NWS and OWS prefer tropical and subtropical climates. These flies are sensitive to cold dry conditions such as those experienced in New Zealand. Currently, New Zealand lies far south of the southernmost limits of NWS and OWS distribution. Furthermore, there are no zoos or wildlife parks located in the Far North region of New Zealand. Therefore, at present, NWS *(C. hominivorax)* and OWS *(C. bezziana)* are unlikely to establish in New Zealand due to the current climatic conditions.

Although the likelihood of NWS *(C. hominivorax)* and OWS *(C. bezziana)* exposure within the zoo is assessed as very low and the likelihood of exposure outside the zoo is assessed as very low, the establishment of NWS *(C. hominivorax)* and OWS *(C. bezziana)* in New Zealand is assessed as negligible due to unsuitable climatic conditions.

32.2.3 Risk estimation

Since the exposure is assessed as negligible, the risk estimate for NWS *(C. hominivorax)* and OWS *(C. bezziana)* is negligible, and it is not a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures are not warranted.

33 Transmissible spongiform encephalopathy

33.1 Technical review

33.1.1 Aetiological agent

Transmissible spongiform encephalopathies (TSEs) are a family of rare progressive neurodegenerative disorders affecting both humans and animals.

The causative agents of TSEs are prions (Centers for Disease Control and Prevention, 2018).

Of the TSEs that affect animals, bovine spongiform encephalopathy (BSE) affects bovines, chronic wasting disease affects Cervidae, scrapie affects ovines and caprines (USDA, 2019), transmissible mink encephalopathy affects ranch-reared mink (*Neovison* and *Mustela* genera) (Liberski et al., 2009), feline spongiform encephalopathy affects Felidae (Gruffydd-Jones et al., 1991) and ungulate spongiform encephalopathy affects exotic zoo ruminants of the family Bovidae (Imran & Mahmood, 2011).

It is hypothesised that ungulate spongiform encephalopathy was caused by infection with host-encoded prion protein BSE (Imran & Mahmood, 2011).

Therefore, for the purposes of this IRA, only BSE will be assessed, as it is the only relevant prion disease that could affect species within the scope of this IRA.

33.1.2 OIE list

Bovine spongiform encephalopathy is an OIE-listed disease of cattle (OIE, 2020d).

33.1.3 New Zealand status

New Zealand has a negligible BSE-risk status, as recognised by the OIE (OIE, 2020a).

Bovine spongiform encephalopathy is a notifiable disease under the Biosecurity (Notifiable Organisms) Order 2016.

33.1.4 Zoonotic potential

There is a causal link between the ingestion of classical (C-type) BSE and the variant Creutzfeldt-Jakob disease in humans (Bruce et al., 1997).

33.1.5 Epidemiology

Host range

Bovine spongiform encephalopathy primarily affects adult cattle.

Sheep, deer and various other animals are susceptible to experimental infections with BSE prions (Dagleish et al., 2008; Foster et al., 2001).

Bovine spongiform encephalopathy has also been diagnosed in captive Bovidae species (Pattison, 1998; Williams & Miller, 2003).

A TSE indistinguishable from BSE was detected in pumas, cheetahs, ocelots and a tiger in zoos in the UK between 1992 and 1995 (Animal and Plant Health Agency, 2019; Pattison, 1998).

Natural transmission of BSE has not been reported in species of the Giraffidae or Tragulidae families.

Captive wild ruminants

Spongiform encephalopathy was noted in a nyala from a wildlife park in England. A year after this case, a similar case was reported in a gemsbok. There was no association between these 2 animals (Jeffrey & Wells, 1988).

Bovine spongiform encephalopathy has since been diagnosed in Arabian oryxes, scimitar-horned oryxes, elands, greater kudus and North American bison (Bovidae) (Pattison, 1998; Williams & Miller,

2003) at UK zoos. The relationship between TSE noted in wildlife ruminants and BSE was confirmed through strain typing studies (Bruce et al., 1994).

Geographical distribution

Classical BSE was initially identified in the UK in 1986. It was likely present in the country's cattle population since the 1970s or earlier. Since then, it has been diagnosed in 25 other countries in Europe, Asia, the Middle East and North America (OIE, 2020a).

Bovine spongiform encephalopathy has never occurred in Australia, Singapore, the UAE or the RSA (WAHIS, 2019d).

The OIE has established official recognition of sanitary BSE-risk status in countries in their entirety or in defined zones and compartments. This applies only to C-type BSE. The OIE officially recognises 2 categories "negligible risk" and "controlled risk"; however, countries/zones that do not fall into these categories, fall into the third "undetermined" BSE risk category.

Of the approved countries within this IRA, the OIE recognises Australia, the USA, Singapore, Japan, and a zone in the UK (Northern Ireland) as having "negligible BSE-risk". Canada and some zones in the UK (England, Wales and Scotland) are recognised as having "controlled BSE-risk" (OIE, 2020a).

Pathogenesis

Studies have suggested that the incubation period is at least 2 years but may be as long as 10 years. This is likely dependent on the infectious dose (Konold, Arnold et al., 2012).

In the UK, clinical C-type BSE was reported in cattle aged 20 months to 22 years. In the epizootic experienced during the 1980s and 1990s (Bradley, 1998), most cases were seen in dairy cattle between 4 to 6 years of age (OIE Terrestrial Manual, 2018f).

The disease is caused by the accumulation of abnormal prion protein (PrP^{Sc}, PrP^{d} or PrP^{res}) in the central nervous system (CNS). This protein is a partially protease-resistant isoform of a host-encoded protein (PrP^{C}).

After oral exposure, prion proteins are transcytosed from the lumen of the gut to the epithelium, likely by M-cells. Accumulation of PrP^{Sc} occurs in the lymphoid tissue of the gut, within 8 months post exposure. During infection of the gut, the PrP^{Sc} comes into contact with nerve fibres of the enteric nervous system. It then travels via the sympathetic nervous system to the brainstem and the parasympathetic nervous system to the brain (Costassa et al., 2016).

Strain variations have been reported (Biacabe et al., 2008; Casalone et al., 2004).

Molecular profiles of atypical BSE differ to those of C-type BSE, and it is suggested that they are biologically distinct (Béringue et al., 2006; Lombardi et al., 2008). The 2 forms of atypical BSE are defined as L-type or H-type, based on the lower or higher mass, respectively, of the unglycosylated PrP^{Sc} fragment in Western immunoblots compared to C-type BSE (Casalone et al., 2004; Jacobs et al., 2007).

The origin of atypical BSE remains unclear. It is hypothesised that it represents a spontaneous or sporadic TSE in cattle. In cases that have been identified there has been no record of clinical signs or history of exposure to prion contaminated meat or bone meal (Costassa et al., 2016).

Clinical signs

Bovine spongiform encephalopathy is a fatal disease of cattle. The disease course is usually subacute to chronic.

The disease usually has a gradual onset and slow progression (OIE Terrestrial Manual, 2018f). Acute cases with rapid deterioration have also been reported.

Clinically, BSE in cattle manifests as a neurological disease. Animals show apprehension, change in behaviour, hyperreactivity and ataxia. Repeated, startled responses to external stimuli often supported suspicion of BSE (Konold et al., 2004).

Subtle clinical signs can be exacerbated by stressful conditions such as transportation (OIE Terrestrial Manual, 2018f).

In advanced cases, locomotive abnormalities, generalised weakness, reduction in milk yield, falling and recumbency may be noted (Konold et al., 2004).

Clinical signs of naturally occurring atypical BSE have not been recorded due to the cases being identified by active surveillance of fallen stock or apparently healthy animals. All infected cattle were older than 8 years (Konold, Bone et al., 2012).

Clinical signs in captive wild ruminants (nyala) have included hind limb ataxia, abnormal head posture, frequent urination, and persistent licking and biting of rump, causing mutilation (Jeffrey & Wells, 1988).

Transmission

Certain tissues of infected animals – those referred to as specified risk materials (SRMs) – are most likely to contain the BSE prion. These tissues include brain, eyes, spinal cord, skull, vertebral column, tonsils and distal ileum (OIE, 2020a).

Transmission occurs via ingestion of BSE-contaminated meat, bone meal or feedstuff containing meat or bone meal (Wells & Wilesmith, 1995).

There is no evidence of direct (horizontal) transmission (OIE, 2020a).

There is little evidence supporting vertical transmission (OIE, 2020a; Prince et al., 2003).

Cases of BSE reported in captive wild ruminants were presumed to have been due to contaminated feedstuffs (Williams & Miller, 2003).

The infectious prion is not inactivated by commercial procedures such as heating, implying rendering is ineffective in destroying the prions (OIE, 2020a).

Diagnosis

Bovine spongiform encephalopathy may be suspected based on clinical signs. There is currently no diagnostic test to confirm BSE in live animals.

The OIE recommended tests for the confirmation of clinical cases by the detection of the PrP^{Sc} in brain tissue are immunohistochemistry and Western immunoblot. It is recommended that a combination of these tests be applied to the same clinical sample to increase sensitivity (OIE Terrestrial Manual, 2018f).

Rapid screening tests (rapid Western immunoblot, lateral flow assays and ELISA) have been developed to aid with eradication and surveillance. They allow for the processing of large numbers of brain samples (OIE Terrestrial Manual, 2018f).

Treatment, control and prevention

There is no known treatment. Affected animals will eventually die of the disease.

There are no vaccines available.

Control and prevention measures that are implemented in countries affected with BSE are:

- banning the use of meat and bone meal in ruminant feed (ruminant-to-ruminant feed ban, further reinforced by a mammalian-to-ruminant feed ban)
- prohibition of the inclusion of SRMs in animal feeds, thus removing potentially contaminated material from the food chain
- removal of SRMs during slaughter and processing of carcasses
- appropriate disposal of carcasses and all animal products
- humane destruction of all suspected animals exposed to prion-contaminated feed
- targeted surveillance of occurrences of clinical neurological disease
- livestock identification to enable effective surveillance and tracing of suspected livestock
- transparency in reporting findings of BSE.

These measures have shown to be effective in limiting the exposure of susceptible animals to BSE prions (OIE, 2020a).

In an attempt to control potential human infection by BSE-contaminated products, countries have enforced the removal of SRMs from bovine carcasses (OIE, 2020a).

For countries free of BSE, there should be safeguards on the importation of live ruminant species and their products.

New Zealand has in place the Biosecurity (Ruminant Protein) Regulations 1999, which manages the risk of an outbreak of BSE in the country. These regulations achieve this objective by (a) prohibiting the feeding of ruminant protein to ruminant animals, and (b) requiring those manufacturing feed for ruminant animals to have an MPI-registered ruminant protein control programme (RPCP) if using or storing ruminant protein onsite (Biosecurity New Zealand, 2018b).

Semen

There is no published evidence demonstrating the presence of BSE prions in the semen of domestic or wildlife ruminants, and there is no published evidence demonstrating the transmission of the agent to naïve dams if inseminated with semen contaminated with BSE prions.

In the study by Morales et al. (2013), no evidence of sexual transmission of prions could be found in the hamster model (Morales et al., 2013).

According to the OIE Code recommendations, semen is not a risk commodity and does not require conditions for its importation, regardless of the BSE status of the exporting country.

33.1.6 Hazard identification conclusion

Bovine spongiform encephalopathy is an OIE-listed disease affecting cattle.

There is no evidence demonstrating the presence of BSE prions in the semen of wildlife ruminants.

Bovine spongiform encephalopathy prions are not identified as a hazard in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

Bovine spongiform encephalopathy prions are identified as a hazard in captive wild Bovidae, Giraffidae and Tragulidae.

33.2 Risk assessment

33.2.1 Entry assessment

Bovine spongiform encephalopathy has never occurred in Australia, Singapore, the UAE or the RSA. The OIE recognises Australia, the USA, Singapore, Japan and a zone in the UK (Northern Ireland) as having “negligible BSE risk”.

There is likely to be a very small number of live captive wild ruminants imported into New Zealand, and these imports are also likely to be infrequent. Due to the low volume of trade, the likelihood that an imported animal will be infected is assessed as very low.

Bovine spongiform encephalopathy has been reported in domestic and captive wild ruminants in some approved countries. Potential sources of infection in captive wild ruminants were reported to be contaminated feedstuffs.

The incubation period for BSE is long, with an average of 2 years. Bovine spongiform encephalopathy has a gradual onset and slow progression. If captive wild ruminants are incubating the disease or are in the very early stages of disease, clinical signs may be absent or go unnoticed. Animals could be passed as clinically sound for export, if they are exported from BSE-affected countries.

However, since the BSE epizootic in the UK, countries have implemented feed bans (a ruminant-to-ruminant feed ban, further reinforced by a mammalian-to-ruminant feed ban). The use of meat and bone meal in ruminant feed should therefore be eliminated. Therefore, the exposure of captive wild ruminants to BSE prions is assessed as very low.

Therefore, the likelihood of entry of BSE prions via captive wild Bovidae, Giraffidae and Tragulidae (within the scope of this IRA) from BSE-affected countries is assessed as very low.

33.2.2 Exposure assessment

Transmission occurs via ingestion of BSE prion contaminated meat, bone meal or feedstuff containing meat or bone meal. There is no evidence of direct (horizontal) transmission and little evidence supporting vertical transmission.

Tissues regarded as SRM are most likely to contain the BSE prion. Prions are not inactivated by commercial procedures such as rendering.

The small number and occasional importation of probably infected live captive wild ruminants implies that there would be a small number of animals acting as a source of exposure to susceptible species.

If infected imported animals enter New Zealand zoos, they may only begin showing clinical signs of BSE months to years later. However, it has been mentioned that stressful conditions, such as transportation or handling, could exacerbate disease manifestation.

Since transmission can only occur via ingestion of contaminated feedstuffs, animals or humans in contact with infected animals would not contract the BSE prions from these animals. Therefore, exposure to animals and humans is assessed as negligible.

Once animals begin showing neurological signs, they are likely to be examined by veterinary staff. A diagnosis of BSE may only be suspected and cannot be confirmed in live animals. Therefore, the animal may be treated symptomatically for other conditions.

There is no treatment for BSE, and animals infected will eventually die. In zoos, animals that reach advanced stages of BSE with severe neurological signs are likely to be euthanased for animal welfare reasons. All animals in zoological collections that die or are euthanased undergo a post-mortem examination. These animals or carcasses would not be fed to other animals or humans within or outside the zoo.

Since there is no exposure pathway, there would be no establishment of BSE in animals within or outside the zoo.

Therefore, the likelihood of BSE prion exposure and establishment within and outside the zoo via infected captive wild Bovidae, Giraffidae and Tragulidae is assessed as negligible.

33.2.3 Risk estimation

Since the exposure is assessed as negligible, the risk estimate for BSE is negligible, and it is not a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures are not warranted.

34 Internal parasites

34.1 Technical review

34.1.1 Aetiological agent

For the purposes of this IRA, internal parasites are defined as parasites of the classes Cestoda and Trematoda, and the phylum Nematoda.

The internal parasites that have been identified in wildlife ruminant species within the scope of this IRA are too vast to be listed and assessed individually. An attempt is made to cover groups of internal parasites and general principles of epidemiology. The groups of internal parasites include nematodes (roundworms), cestodes (tapeworms) and trematodes (flukes).

Examples of internal parasite species within each group that have been identified in wildlife ruminants are given below. This is not an exhaustive list:

Cestodes:
Moniezia expansa
Stilesia hepatica (Boomker et al., 1986)

Nematodes:
Cooperia neitzi
Dictyocaulus viviparous
Haemonchus contortus
Longistrongylus meyeri
Trichostrongylus axei (Boomker et al., 1986)

Trematodes:
Calicophoron microbothrium
Calicophoron calicophorum
Cotylophoron cotylophorum
Carmyerius bubalis
Bilatorchis papillogenitalis (Pfukenyi & Mukaratirwa, 2018).

34.1.2 OIE list

Internal parasites (affecting mammals) that are OIE-listed include *Echinococcus granulosus, Echinococcus multilocularis, Taenia solium* (cestodes) and *Trichinella* spp. (nematode) (OIE, 2020d).

34.1.3 New Zealand status

Numerous internal parasites have been identified in wild ruminants in New Zealand. Many of these internal parasites have also been reported in domestic ruminants (Andrews, 1969).

The internal parasites recorded include nematodes, cestodes and trematodes. These internal parasites were introduced with a variety of domestic and game animals from Europe, Australia and America (Andrews, 1969; Charleston & McKenna, 2002).

34.1.4 Zoonotic potential

Various internal parasites of ruminants are zoonotic. Some examples include *Trichostrongylus* spp., *Taenia* spp. and *Echinococcus* spp. (Heyneman, 1996; Roeber et al., 2013).

34.1.5 Epidemiology

In the text below, some species are mentioned as examples, but this does not imply that these are the only species that could be found in the commodity.

Many internal parasites of ruminants infest both domestic and wildlife ruminant species. Boomker et al. (1986) reported that the host specificity of the internal parasites that were identified in his study was not marked and there was a fair degree of cross-infection.

There are, however, various internal parasites that are quite host-specific, such as *Monodontella giraffae* (Boomker et al., 1986).

Internal parasites undergo various stages of development known as the “life cycle”. Some stages may be free-living and pre-infective, and other stages require an intermediate host for further development, while some stages are parasitic to animal hosts.

In nematodes, eggs are passed in faeces of infested animals. Eggs then develop in faeces into infective larvae. Larvae remain on grass until ingested by animals. Larvae then develop into adult worms in the gastrointestinal tract of animals. Adult worms in turn lay eggs, and the cycle continues.

Trematodes and cestodes usually require 2 hosts to complete their life cycles: a definitive host and an intermediate host.

In cestodes such as *Moniezia* spp., definitive hosts are ruminants, and the intermediate hosts are oribatid mites (Sinitsin, 1931). Infested ruminants pass eggs in gravid proglottids in faeces. Eggs are ingested by mites. Eggs develop into the cysticercoid stage in mites. The infected mite is then ingested by ruminants. Mature cysticercoids are digested out of the mite and develop into adult tapeworms in the gastrointestinal tract of the ruminant (Constable et al., 2017).

The trematode *Calicophoron* spp. (fluke), also make use of ruminants as their definitive hosts. Eggs are passed in faeces of infested ruminants. When eggs reach water, miracidia hatch and penetrate intermediate hosts, which are snails. Miracidia undergo various developmental stages to become cercariae and escape the snail. Metacercariae encyst on water plants, which are then eaten by ruminants (Iglesias-Piñeiro et al., 2016; Mohammed Samn, 2017).

Cestodes and trematodes have a wide range of intermediate host species including snails, reptiles, fish, crustaceans, insects and mammals.

Internal parasites across the approved countries may differ due to varying climatic and geographical habitats, and host specificity.

Ruminants in New Zealand have been introduced from Europe, Australia and America; therefore, some internal parasites present in New Zealand may be similar to those identified in domestic and wildlife ruminants of those countries. However, internal parasites from other approved countries such as Singapore, UAE and the RSA may be absent from New Zealand.

Studies have revealed that animals kept in captivity often suffer high internal parasite burdens (Mirzapour et al., 2018). Despite extensive quarantine periods, repeated faecal examinations and anthelmintic treatment to prevent their introduction, nematodes are still problematic in captive wild ruminants (Goossens et al., 2005). High stocking densities (Flach & Sewell, 1987) and limited ability to rotate grazing are contributing factors.

Studies and case reports of captive collections report internal parasite prevalences of 65% to 100% (Geraghty et al., 1982; Goossens et al., 2005).

Mortality due to parasitic gastroenteritis ranged from 5% to 17% (Kaneene et al., 1985). Captive roan and sable antelopes, blackbucks and gazelles suffering high worm burdens experienced loss of body condition, diarrhoea and death (Church, 1986; Flach & Sewell, 1987).

Transmission of internal parasites from infested to susceptible animals occurs via indirect contact. Internal parasite eggs are passed out in faeces of animals into the environment. Therefore, animals within enclosures are likely to ingest eggs or infective stages from the environment. Depending on the type of enclosures in captivity, dung may be removed regularly, but in larger spaces, this may be impractical.

Diagnosis of internal parasites can be achieved by various methods. The most practical, non-invasive method is to conduct a faecal flotation or to determine faecal eggs counts. Fresh faeces are collected, and eggs are counted using techniques such as the McMaster method.

Faeces can be incubated to grow larvae that can be identified to either genus or species level.

Animals could be necropsied, and the gastrointestinal contents and lining can be analysed for adult parasite burdens.

Various anthelmintics can be used to treat ruminants with internal parasite burdens (Flach & Sewell, 1987); however, anthelmintic resistance is common.

Anthelmintics that are effective against nematodes are benzimidazoles, imidazothiazoles, tetrahydroprimidine and macrocyclic lactones (Craig, 2003).

The sulfonamide clorsulon and the benzimidazole albendazole are effective against liver flukes and can be administered orally, topically or by injection.

Benzimidazoles and probenzimidazoles have cestocidal activity (Constable et al., 2017; Flach & Sewell, 1987).

Ivermectin has also been used with success (Flach & Sewell, 1987), but may be extra-label in wildlife ruminants (Soll, 1989).

Control of internal parasites could be achieved by:

- strategic and targeted treatment of animals with anthelmintics with special attention regarding refugia
- regular removal of dung from the environment
- ensuring low stocking densities in enclosures
- eliminating intermediate hosts if possible and practical.

In facilities that want to prevent entry of internal parasites, ensuring that imported animals are appropriately treated for internal parasites, quarantining animals and testing faecal samples for internal parasite eggs will aid prevention.

There is no published evidence demonstrating the presence of internal parasites in the semen of domestic or wildlife ruminants.

34.1.6 Hazard identification conclusion

Internal parasites are identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae.

Internal parasites are not identified as hazards in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

34.2 Risk assessment

34.2.1 Entry assessment

Internal parasites have been reported in wild and captive wild ruminants worldwide. Animals in captivity with high stocking densities are likely to experience higher internal parasite burdens, unless this is controlled by a preventative medicine programme.

Only animals with high internal parasite burdens are likely to experience clinical signs of infestation such as loss of body condition and diarrhoea. Those animals with low burdens could easily go unnoticed, as internal parasites are not visible. Animals could be passed as clinically sound for export.

Therefore, the likelihood of entry of internal parasites via captive wild Bovidae, Giraffidae and Tragulidae is assessed as high.

34.2.2 Exposure assessment

Transmission of internal parasites from infested to susceptible animals occurs via indirect contact. Animals within the same enclosure or in contiguous enclosures could become infested after ingesting eggs or infective stages from the environment. In the case of cestodes, arthropod intermediate hosts (mites) could transfer to susceptible animals and be ingested. If enclosures contain water features, they could harbour intermediate hosts (snails) of trematodes and infective stages could attach to vegetation and be ingested by ruminants.

If infested animals are not treated, internal parasites could establish in these animals. However, the parasites are unlikely to spread to animals outside the enclosure. In cestodes and trematodes, intermediate hosts such as snails, reptiles, fish, crustaceans and invertebrates may not be present in enclosures of infested animals. Some species of cestodes and trematodes cannot complete their life cycles in the absence of these intermediate hosts. Therefore, these species are unlikely to establish in animals.

It is unlikely that animals in completely separate enclosures within the zoo could be exposed to eggs or infective stages, unless intermediate hosts travel between enclosures. However, if domestic animals are kept in the same enclosures as imported captive wild ruminants, they could become infested with internal parasites. Should these domestic ruminants be released onto New Zealand farms, they could be a source of infestation to other domestic ruminants. These new internal parasites could thus spread and establish in the domestic ruminant population.

Some internal parasites are known to be zoonotic. Therefore, zoo and veterinary personnel could be exposed to infection with zoonotic parasites. Visitors to the zoo are unlikely to be exposed to internal parasites.

Therefore, the likelihood of internal parasite exposure and establishment within the zoo via infested captive wild Bovidae, Giraffidae and Tragulidae is assessed as low, and the likelihood of exposure and establishment outside the zoo is assessed as very low.

34.2.3 Consequence assessment

Direct consequences would relate to the infested imported animals and those that become infested. In zoological collections with low stocking densities, regular removal of dung and a preventative medicine programme, internal parasite burdens are not likely to cause significant health concerns in captive wild ruminants. However, there are case reports where internal parasite prevalences were high and there was significant morbidity and mortality associated with internal parasites.

Animals in zoological collections are very valuable, and an infestation of internal parasites would negatively impact the zoos' ASMP and conservation efforts.

It is likely that nematode parasites could establish in captive ruminant populations and spread to animals outside the zoo if domestic animals are kept in the same enclosures and then released to New Zealand farms. Cestode and trematode species may only establish in captive wild ruminants if suitable intermediate hosts are present.

Some internal parasites are zoonotic, and therefore, zoo and veterinary personnel could become infected.

Indirect consequences would entail the costs for regular treatment and testing of captive wild animals to determine faecal egg counts.

Numerous species of internal parasites are present in domestic animals in New Zealand. Currently, farmers and owners manage worm burdens by regular treatment with endoparasiticides. Should new internal parasites be introduced, they too could be managed in a similar way. This may amount to minimal costs.

Of the 3 OIE-listed internal parasites, *Trichinella* spp. do not affect ruminants, and ruminants are dead-end hosts of *Echinococcus* spp. Therefore, there are unlikely to be any trade impacts following entry of internal parasites via captive wild ruminants.

Therefore, the overall consequences as a result of internal parasite incursions are assessed as very low.

34.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for internal parasites is non-negligible, and internal parasites are assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

34.3 Risk management

The aim of risk management measures under this chapter is to cover all potential internal parasites that could be harboured by imported captive wild ruminants. Not all internal parasites are absent from New Zealand or are a biosecurity risk. However, the broad spectrum risk management measures that are recommended are likely to mitigate the entry of internal parasites that are assessed as risks in the commodity as well as those that are not.

The following points were taken into account when describing options for managing the risks:

- The internal parasites of wildlife ruminants are too vast to be listed and assessed individually.
- OIE-listed infestations of mammals include *Echinococcus granulosus, Echinococcus multilocularis, Taenia solium* and *Trichinella* spp.
- Various nematodes, cestodes and trematodes are present in New Zealand and have been introduced with domestic and game animals
- Various internal parasites of ruminants are zoonotic, e.g. *Trichostrongylus* spp., *Taenia* spp. and *Echinococcus* spp.
- Host specificity of internal parasites identified in wildlife ruminants is low, and there is a fair degree of cross-infection.
- Internal parasites undergo various stages of development known as the “life cycle” with free-living and parasitic stages.
- Trematodes and cestodes usually require 2 hosts (definitive and intermediate host) to complete their life cycles.
- Animals kept in captivity, with high stocking densities, may suffer high internal parasite burdens if unmanaged.
- Morbidity and mortality rates can be high due to internal parasite burdens.
- Transmission of internal parasites from infested to susceptible animals occurs via indirect contact.
- Diagnostic methods include faecal flotation, faecal egg counts, and necropsy and analysis of gastrointestinal contents and lining.
- Anthelmintics can be used to treat ruminants for internal parasite burdens.
- There is no published evidence of the presence of internal parasites in the semen of domestic or wildlife ruminants.

34.3.1 Options

Internal parasites are not identified as hazards in semen; therefore, risk management measures are not warranted for semen.

One or a combination of the following options may be used for animals.

Options

1. The animal(s) must be treated with an MPI-approved endoparasiticide that covers nematodes, cestodes and trematodes 7 to 10 days prior to entering pre-export isolation; AND
2. the animal(s) must be treated twice with an MPI-approved endoparasiticide that covers nematodes, cestodes and trematodes during pre-export isolation; AND
3. all dung should be removed regularly during pre-export isolation; AND
4. fresh faecal samples from the animal(s) should be tested using an MPI-approved test to determine faecal egg counts, with zero egg counts.

35 External parasites

35.1 Technical review

35.1.1 Aetiological agent

The external parasites that have been identified on wildlife ruminant species within the scope of this IRA are too vast to be listed and assessed individually. An attempt is made to cover groups of external parasites. The groups of external parasites include ticks, mites, fleas, lice and flies.

Examples of external parasite species that have been identified on wildlife ruminants are given below. This is not an exhaustive list.

Ticks:
Amblyomma hebraeum
Rhipicephalus spp. (Horak et al., 1983)

Mites:
Demodex spp. (Bukva et al., 1988)
Psoroptes cuniculi (Wright & Glaze, 1988)

Lice:
Damalinia reduncae
Linognathus fahrenholzi (Horak et al., 1988)

Flies:
Kirkioestrus minutus (Horak et al., 1980)
Gedoelstia spp. (Horak & Boomker, 1998).

35.1.2 OIE list

External parasites that are OIE-listed infestations include new world screw-worm fly (*Cochliomyia hominivorax*) and old world screw-worm fly (*Chrysomya bezziana*) (OIE, 2020d). These parasites have been assessed individually in previous chapters.

35.1.3 New Zealand status

New Zealand has 10 native tick species (Southern Monitoring Services Limited, 2014). The cattle tick (*Haemaphysalis longicornis*) was introduced and has established in New Zealand.

In New Zealand, over 1,200 species (in 540 genera belonging to over 180 families) of mites had been described by the year 2000 (Landcare Research, 2020). *Psoroptes ovis*, *Psorergates ovis*, *Demodex* spp. are examples of mites that infest domestic ruminants in New Zealand (Heath, 1994).

Lice present in New Zealand include *Bovicola ovis.*

Flies present in New Zealand include *Calliphora stygia, Lucilia sericata* and *Chrysomya rufifacie*.

Mosquitoes present in New Zealand include *Aedes* spp. and *Anopheles* spp.

Some external parasites have been introduced with the introduction of domestic and wild animals. The parasites were then able to establish in New Zealand.

External parasites are not a major threat to farming. However, they do pose a major concern to farmers, as they are a drain on resources (Heath, 1994).

35.1.4 Zoonotic potential

Various external parasites of ruminants can affect humans. Examples of these include ophthalmomyiasis caused by flies (Basmaciyan et al., 2018). Various diseases can be transmitted to humans via tick vectors (CDC, 2020).

35.1.5 General considerations

In the text below, some species are mentioned as examples, but this does not mean that these are the only species that might be found on the commodity.

Many external parasites of ruminants infest both domestic and wildlife ruminant species. *Psoroptes* spp., *Sarcoptes* spp. and *Demodex* spp. have been found on antelopes as well as domestic ruminants (Bukva et al., 1988; Wright & Glaze, 1988).

External parasites across the approved countries may differ due to varying climatic and geographical habitats, and host specificity. Ruminants in New Zealand have been historically introduced from Europe, Australia and America. Therefore, some external parasites present in New Zealand may be similar to those identified in domestic and wildlife ruminants of those countries. However, external parasites from other approved countries such as Singapore, the UAE and the RSA may be absent from New Zealand.

In natural ecosystems hosts, disease agents and vectors have coevolved in the environment such that pathogens and parasites may be borne by wild animals without overt signs of clinical disease.

Heartwater is an example of a tick-borne disease where a susceptible mammalian host can be tolerant to the rickettsial parasite carried by the tick. An innate resistance exists in wildlife ruminants that share the same habitat as the *Amblyomma* spp. tick vector (e.g. giraffes, elands, African buffaloes), and the *Ehrlichia ruminantium* organism will complete its life cycle without the mammalian host ever becoming clinically ill.

Wildlife ruminants are also tolerant to indigenous pathogens transmitted by winged arthropods (e.g. *Rift Valley fever virus* and *Bluetongue virus)* and are likely integral to the epidemiology. However, infrequent clinical disease has been reported in some species.

Where natural ecosystems are disturbed, new relationships between host, disease agent and environment are likely. Hence, it is not uncommon for diseases to emerge on foreign continents with exotic pathogens using novel indigenous vectors.

External parasites may not necessarily cause significant health impacts in wildlife ruminants. However, captive wild animals may be kept in confined areas in zoological collections. External parasites such as ticks, mites and fleas could establish on animals and in the environment, and infestations could become problematic in the absence of a dilution effect that is often experienced in extensive environments.

Animals in zoological collections could be exposed to stressful conditions like handling, movement and human exposure. This could cause some animals to become immunocompromised and succumb to infestations that would not normally affect healthy animals or those in the wild. An example of this is demodectic mange in captive wild elands (Bukva et al., 1988).

Another concern relating to external parasites is their vector capability. In previous chapters, various external parasites have been implicated as vectors of infectious diseases. Table 3 lists vectors and the respective agents they could carry.

Table 3. Vector-borne diseases

Disease	Vector
BT	*Culicoides,* sheep keds, cattle lice, ticks
CCHF	Ticks
EHD	*Culicoides*
LSD	Mosquitoes, biting and non-biting flies, ticks
NSD	Ticks
RVF	Mosquitoes

Disease	Vector
Bovine anaplasmosis	Ticks, flies
Heartwater	Ticks
Q fever	Ticks
Besnoitia	Flies
Bovine babesiosis	Ticks
Theileriosis	Ticks
Trypanosomosis	Flies, mosquitoes

Transmission of external parasites from one animal to another usually occurs via close contact and sharing of the environment.

Diagnosis of mites can be made by skin scrapes and smears, skin biopsies (Bukva et al., 1988) and histology. A fair majority of external parasites can be identified visually via morphological identification. Various developmental stages are more appropriate for identification as they have characteristic features.

Craig (2003) discussed various methods of treatment of external parasites in domestic ruminants. Some of these methods may be used on wildlife ruminants.

For flies, animals could be treated with pesticides via sprays, dips, oilers or dust bags. Organophosphate compounds work well in sprays and dips (Craig, 2003).

Persistent insecticides are effective against fleas.

Ivermectin has been used effectively for mites in domestic ruminants (Craig, 2003). Avermectins have been used to treat wildlife ruminants for internal and external parasites however, their use may be "extra label" (Soll, 1989).

A formadine compound, amitraz, has been used with success against organophosphate-resistant ticks, mites and lice.

Pesticides that are effective against external parasites include pyrethrins, organophosphates and macrocyclic lactones (Craig, 2003).

Vector control measures include the use of acaricides, modified housing of animals and pest management of flies (Aubry & Geale, 2011; OIE Terrestrial Manual, 2018d). Tick control can be achieved by the intensive method of treating animals with acaricides until they are free of ticks or by using the strategic method of treating animals to reduce tick numbers. During importation, the intensive method is recommended (Allsopp et al., 2004).

In facilities that want to prevent entry of external parasites, ensuring that imported animals are appropriately treated for external parasites, quarantining animals and physical examination of animals prior to importation and introduction will aid prevention.

There is no published evidence demonstrating the presence of external parasites in the semen of domestic or wildlife ruminants.

35.1.6 Hazard identification conclusion

External parasites are identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae.

External parasites are not identified as hazards in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

35.2 Risk assessment

35.2.1 Entry assessment

External parasites have been identified on wild and captive wild ruminants worldwide.

External parasites such as mites burrow into the skin. Ticks often attach to areas such as the axilla or inside the ear canal and may not be identified during physical examination. Animals with low external parasite numbers are unlikely to exhibit unnatural behaviour or show any clinical signs of infestation. These animals could be passed as clinically sound for export.

Therefore, the likelihood of entry of external parasites via captive wild Bovidae, Giraffidae and Tragulidae is assessed as high.

35.2.2 Exposure assessment

Transmission of external parasites from one animal to another usually occurs via close contact and sharing of the environment.

Animals within the same enclosure or in contiguous enclosures could become infested. Flying parasites have the ability to travel to enclosures a distance from the initial infested host and could infest susceptible animals within the zoo. These parasites may be able to travel outside the zoo and infest domestic and wild animals. Introduction and establishment of various external parasites has been demonstrated. Therefore, new external parasites brought in with imported captive wild ruminants could also establish in animal populations in New Zealand if environmental conditions are suitable.

Some external parasites are known to be zoonotic. Therefore, zoo and veterinary personnel could be exposed to infection with these parasites. Visitors to the zoo are unlikely to be exposed to external parasites, unless they are flying insects. However, should external parasites establish in animal populations outside the zoo, this would lead to a larger number of humans being exposed to external parasites.

Therefore, the likelihood of external parasite exposure and establishment within the zoo via infested captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate, and the likelihood of exposure and establishment outside the zoo is assessed as low.

35.2.3 Consequence assessment

Direct consequences would relate to the infested imported animals and those that become infested. Large numbers of mites and fly larvae can negatively impact the health of captive wild ruminants. In some cases, they could result in death or elective euthanasia due to the severity of infestations. Animals in zoological collections are very valuable, and this would impact negatively on zoos' ASMP and conservation efforts.

External parasites that are vectors of infectious agents could cause the spread of infectious diseases, as discussed in previous chapters.

External parasites have the ability to establish in animal populations within and outside the zoo.

Some external parasites are zoonotic therefore zoo and veterinary personnel could be exposed. Since flying parasites could establish in animals outside the zoo, a larger number of humans could be exposed to external parasites.

Indirect consequences would entail the costs for regular treatment and examination of animals and potentially the control of any infectious diseases that could be transmitted by vectors.

There are unlikely to be any trade impacts following the entry of external parasites covered under this chapter.

Therefore, the overall consequences as a result of external parasite incursions are assessed as moderate.

35.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for external parasites is non-negligible, and it is assessed as a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

35.3 Risk management

The aim of risk management measures under this chapter is to cover all potential external parasites that might be found on imported captive wild ruminants. Not all external parasites are absent from New Zealand or are a biosecurity risk. However, the broad spectrum risk management measures that are recommended are likely to mitigate the entry of external parasites that are assessed as risks in the commodity as well as those that are not. Measures would also prevent the entry of vectors of infectious diseases.

The following points were taken into account when describing options for managing the risks:

- The external parasites of wildlife ruminants are too vast to be listed and assessed individually.
- There are a number of external parasites that infest both domestic and wildlife ruminants in New Zealand.
- External parasites are not a major threat to farming but can be a drain on resources.
- Various external parasites of ruminants can affect humans.
- External parasites can infest captive wild ruminants and cause morbidity or mortality and be vectors of infectious diseases.
- Transmission from one animal to another occurs via close contact and sharing of the environment.
- Ectoparasiticides can be used on captive wild ruminants to eliminate external parasites.
- There is no published evidence of the presence of external parasites in the semen of domestic or wildlife ruminants.

35.3.1 Options

External parasites are not identified as hazards in semen; therefore, risk management measures are not warranted for semen.

One or a combination of the following options may be used for animals.

Options

1. The animal(s) must be treated with an MPI-approved ectoparasiticide 7 to 10 days prior to entering pre-export isolation; AND
2. within 48 hours of entering pre-export isolation, an MPI-approved ectoparasiticide should be applied to the predilection sites of external parasites, e.g. axilla and ear; AND
3. ten days after entering pre-export isolation, the animals must be physically examined for external parasites. If still infested, the treatment must be repeated, and the animals inspected again 10 days later. Treatments must be repeated until the animals are free from evidence of external parasites.

36 Seeds

36.1 Technical review

36.1.1 Aetiological agent

Viable seeds attached to an animal's hair/coat.

36.1.2 New Zealand status

Seeds that are attached to the animal's hair/coat and do not meet the recommended importation standards, could be a risk to New Zealand.

36.1.3 General considerations

Seeds could be found attached to the hair/coat of captive wild ruminants. Large seed heads and pieces of plant material would be easily visible and could be removed before shipment, but small seeds might not be visible.

Some 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 and retain viability better in dry conditions, although some are specifically adapted to remain viable in water.

Seeds may survive passage through an animals' digestive system and be passed out in faeces. For example, many leguminous plants may pass unaltered through the gastrointestinal tract of domestic ruminants (Suckling, 1996).

Suckling (1996) further demonstrated that viable seeds are passed in ruminant faeces a day after feeding on seeds. However, only a percentage of seeds could be recovered or remained viable. Seeds were passed for at least 6 days after feeding on seeds ceased. Seeds were still recovered from the gastrointestinal tract of ruminants slaughtered 6 days after feeding on seeds. This implies that ingested seeds may take a longer timeframe to be voided in faeces.

Germination of the seeds passed in faeces of domestic ruminants was tested, demonstrating that seeds were able to germinate (Suckling, 1996).

Domestic ruminants are herbivores and have the same anatomical gastrointestinal system as wildlife ruminants. Therefore, digestion would be similar. These findings can be extrapolated to captive wild ruminants.

Seeds are not found in the semen of animals.

36.1.4 Hazard identification conclusion

Seeds are identified as hazards in captive wild Bovidae, Giraffidae and Tragulidae.

Seeds are not identified as hazards in the semen of captive wild Bovidae, Giraffidae and Tragulidae and will not be assessed further.

36.2 Risk assessment

36.2.1 Entry assessment

Seeds that remain attached to the hair/coat of animals, especially in areas that are hard to visualise like the axillary region or the ventral aspect of the body, could go unnoticed during physical examination. Seeds that are still in the gastrointestinal tract and are not voided prior to entry could enter New Zealand with the imported animals.

Therefore, the likelihood of entry of seeds via captive wild Bovidae, Giraffidae and Tragulidae is assessed as moderate.

36.2.2 Exposure assessment

It was demonstrated that ruminants may carry seeds in their gastrointestinal tract 6 days after ingestion. Viable seeds can be voided in faeces for longer than 6 days. Germination of seeds passed in faeces may occur.

Seeds could be brought in with imported animals. Seeds could drop from the hair/coat of animals or be passed with faeces into the environment. Only a percentage of seeds that pass through the gastrointestinal tract would be viable and capable of germination. Depending on the environmental conditions, seeds could lie dormant or germinate immediately.

Therefore, the likelihood that seeds could germinate and grow if released into a suitable environment is assessed as low.

36.2.3 Consequence assessment

Seeds could be introduced into New Zealand via imported captive wild ruminants. Plant species from these seeds could become established, with subsequent deleterious effects on the environment, agriculture and other potential areas depending on the species of plant.

There would be no direct public health impacts.

Indirect consequences would entail the cost of controlling or attempting to eradicate these plants.

There are unlikely to be any trade impacts.

Therefore, the overall consequences as a result of entry and establishment of seeds are assessed as very low.

36.2.4 Risk estimation

Since the entry, exposure and consequences are assessed as non-negligible, the risk estimate for seeds is non-negligible, and it is assessed to be a risk in captive wild Bovidae, Giraffidae and Tragulidae.

Therefore, risk management measures can be justified.

36.3 Risk management

The following points were taken into account when describing options for managing the risks:

- Seeds could be attached to the imported animal's hair/coat.
- Seeds attached to animals do not meet import requirements.
- Viable seeds could be carried in the animal's gastrointestinal tract.
- Seeds can survive unfavourable environments by remaining dormant.
- Seeds can survive the digestive tract of ruminants and germinate once passed in faeces into the environment.

36.3.1 Options

Seeds are not identified as hazards in semen; therefore, risk management measures are not warranted for semen.

One or a combination of the following options may be used for animals.

Options

1. Prior to export, ruminants should be fed a diet not contaminated with seeds; AND/OR
2. the pre-export isolation facility should be free of seeds; AND/OR
3. bedding used for animals during pre-export isolation and transport should be free of seeds; AND/OR
4. during pre-export isolation, animals must be physically examined and free of any seeds from hair/coat; AND/OR
5. the faeces and bedding of the imported animals should be collected and treated during the first 10 days of post-arrival quarantine.

Appendix A

Table 4. List of captive wild ruminant species within the scope of the IRA

Family	Subfamily	Genus	Species	Common Name
Bovidae	Aepycerotinae	*Aepyceros*	*A. melampus*	impala
	Alcelaphinae	*Alcelaphus*	*A. buselaphus*	hartebeest
		Beatragus	*B. hunteri*	hirola
		Connochaetes	*C. gnou*	black wildebeest
			C. taurinus	common wildebeest
		Damaliscus	*D. lunatus*	topi
			D. pygargus	blesbok/bontebok
	Antilopinae	*Ammodorcas*	*A. clarkei*	dibatag
		Antidorcas	*A. marsupialis*	springbok
		Antilope	*A. cervicapra*	blackbuck
		Dorcatragus	*D. megalotis*	beira
		Eudorcas	*E. albonotata*	Mongalla gazelle
			E. rufifrons	red-fronted gazelle
			E. thomsonii	Thomson's gazelle
			E. tilonura	Heuglin's gazelle
		Gazella	*G. arabica*	Arabian gazelle
			G. bennettii	chinkara
			G. cuvieri	Cuvier's gazelle
			G. dorcas	dorcas gazelle
			G. gazella	mountain gazelle
			G. leptoceros	slender-horned gazelle
			G. marica	Arabian sand gazelle
			G. spekei	Speke's gazelle
			G. subgutturosa	goitered gazelle
		Litocranius	*L. walleri*	gerenuk
		Madoqua	*M. guentheri*	Guenther's dik-dik
			M. kirkii	Kirk's dik-dik
			M. piacentinii	silver dik-dik
			M. saltiana	Salt's dik-dik
		Nanger	*N. dama*	Dama gazelle
			N. granti	Grant's gazelle
			N. soemmerringii	Soemmerring's gazelle
		Neotragus	*N. batesi*	Bates' pygmy antelope
			N. moschatus	suni
			N. pygmaeus	royal antelope
		Oreotragus	*O. oreotragus*	klipspringer
		Ourebia	*O. ourebi*	oribi
		Procapra	*P. gutturosa*	Mongolian gazelle
			P. picticaudata	Tibetan gazelle
			P. przewalskii	Przewalski's gazelle
		Raphicerus	*R. campestris*	steenbok
			R. melanotis	Cape grysbok

Family	Subfamily	Genus	Species	Common Name
			R. sharpei	Sharpe's grysbok
		Saiga	*S. tatarica*	saiga
	Bovinae	*Boselaphus*	*B. tragocamelus*	nilgai
		Pseudoryx	*P. nghetinhensis*	saola
		Tetracerus	*T. quadricornis*	four-horned antelope
		Tragelaphus	*T. angasii*	nyala
			T. buxtoni	mountain nyala
			T. derbianus	giant eland
			T. eurycerus	bongo
			T. imberbis	lesser kudu
			T. oryx	common eland
			T. scriptus	bushbuck
			T. spekii	sitatunga
			T. strepsiceros	greater kudu
	Cephalophinae	*Cephalophus*	*C. adersi*	Aders' duiker
			C. callipygus	Peters' duiker
			C. dorsalis	Bay duiker
			C. harveyi	Harvey's duiker
			C. jentinki	Jentink's duiker
			C. leucogaster	white-bellied duiker
			C. natalensis	Natal red duiker
			C. niger	black duiker
			C. nigrifrons	black-fronted duiker
			C. ogilbyi	Ogilby's duiker
			C. rufilatus	red-flanked duiker
			C. silvicultor	yellow-backed duiker
			C. spadix	Abbott's duiker
			C. weynsi	Weyns's duiker
			C. zebra	zebra duiker
		Philantomba	*P. maxwellii*	Maxwell's duiker
			P. monticola	blue duiker
		Sylvicapra	*S. grimmia*	common duiker
	Hippotraginae	*Addax*	*A. nasomaculatus*	addax
		Hippotragus	*H. equinus*	roan antelope
			H. niger	sable antelope
		Oryx	*O. beisa*	beisa oryx
			O. dammah	scimitar-horned oryx
			O. gazella	gemsbok
			O. leucoryx	Arabian oryx
	Reduncinae	*Kobus*	*K. ellipsiprymnus*	waterbuck
			K. kob	kob
			K. leche	southern lechwe
			K. megaceros	Nile lechwe
			K. vardonii	puku
		Pelea	*P. capreolus*	grey rhebok
		Redunca	*R. arundinum*	southern reedbuck
			R. fulvorufula	mountain reedbuck

Family	Subfamily	Genus	Species	Common Name
			R. redunca	bohor reedbuck
Giraffidae	Giraffinae	*Giraffa*	*G. camelopardalis*	giraffe
		Okapia	*O. johnstoni*	okapi
Tragulidae		*Hyemoschus*	*H. aqauticus*	water chevrotain
		Moschiola	*M. indica*	Indian chevrotain
			M. kathgyre	yellow-striped chevrotain
			M. meminna	white-spotted chevrotain
		Tragulus	*T. javanicus*	Javan chevrotain
			T. kanchil	lesser oriental chevrotain
			T. napu	greater oriental chevrotain
			T. nigricans	Balabac mouse deer
			T. versicolor	silver-backed chevrotain
			T. williamsoni	Williamson's chevrotain

Appendix B

Table 5. Summary of IRA

Disease	OIE listed	Zoonotic	Hazard in Bovidae	Hazard in Giraffidae	Hazard in Tragulidae	Hazard in semen	Likelihood of entry in live animals	Likelihood of entry in semen	Likelihood of exposure and establishment within the zoo	Likelihood of exposure and establishment outside the zoo	Likelihood of exposure and establishment via semen	Consequences of entry, exposure and establishment	Risk Management	Risk Indicators	
Bluetongue	Y	N	Y	Y	Unknown	Y	Moderate	Very Low	Negligible	Negligible	Negligible	N/A	N	**Risk attributes**	
Malignant catarrhal fever -WA	N	N	Y	N	N	N	High	N/A	Low	Negligible	N/A	Very Low	Y	Negligible	Not worth considering, insignificant
Bovine viral diarrhoea	Y	N	Y	Ab only	Y	Y	Very Low	Very Low	High	Very Low	Low	Low	Y	Non-negligible	Worth considering, significant
Cowpox	N	Y	N	Y	N	N	Negligible	N/A	N/A	N/A	N/A	N/A	N	**Risk descriptors**	
Crimean Congo HF	Y	Y	Y	Y	Unknown	N	Very Low	N/A	Very Low	Very Low	N/A	Mod	Y	Very low	Close to insignificant
Epizootic haemorrhagic disease	Y	N	Ab only	Ab only	Ab only	N	N/A	N/A	N/A	N/A	N/A	N/A	N	Low	Less than average, coming below the normal level
Foot and mouth disease	Y	Y	Y	Y	Y	Y	Low	Low	High	Low	Low	High	Y	Medium	Around the normal or average level
Infectious bovine rhinotracheitis	Y	N	Y	Y	N	Y	Very Low	Very Low	Low	Very Low	Low	Low	Y	High	Extending above the normal or average level
Lumpy skin disease	Y	N	Y	Y	N	Y	Very Low	Very Low	Low	Very Low	Very Low	Low	Y	Very high	Well above the normal or average level
Nairobi sheep disease	Y	Y	Y	N	N	N	Negligible	N/A	N/A	N/A	N/A	N/A	N		
Peste des petits ruminants	Y	N	Y	Y	Y	Unknown	Low	Very Low	Moderate	Very Low	Very Low	Low	Y		
Rabies	Y	Y	Y	Y	Y	N	Low	N/A	Moderate	Negligible	N/A	Moderate	Y		
Rift Valley fever	Y	Y	Y	Y	Unknown	Unknown	Low	Very Low	Low - Moderate	Low	Very Low	High	Y		
Anthrax	Y	Y	Y	Y	Unknown	N	Very Low	N/A	Very Low	Negligible	N/A	Moderate	Y		
Bovine brucellosis	Y	Y	Y	Unknown	Unknown	Y	Very Low	Very Low	Low	Very Low	Low	Moderate	Y		
Caprine and ovine brucellosis	Y	Y	Y	Unknown	Unknown	Y	Very Low	Very Low	Low	Very Low	Low	Moderate	Y		
Bovine tuberculosis	Y	Y	Y	Y	Unknown	Y	Low	Very Low	Moderate	Very Low	Very Low	Very Low	Y		
Contagious agalactia	Y	N	N	N	N	N	N/A	N/A	N/A	N/A	N/A	N/A	N		
Contagious bovine pleuropneumonia	Y	N	N	N	N	N	N/A	N/A	N/A	N/A	N/A	N/A	N		
Contagious caprine pleuropneumonia	Y	N	Y	N	N	N	Very Low	N/A	Very Low	Very Low	N/A	Low	Y		
Mycoplasma bovis infection	N	Y	N	N	N	N	N/A	N/A	N/A	N/A	N/A	N/A	N		
Haemorrhagic septicaemia	Y	N	Y	Unknown	Unknown	N	Very Low	N/A	Moderate	Very Low	N/A	Low	Y		
Bovine anaplasmosis	Y	N	Y	N	N	N	Very Low	N/A	Very Low	Very Low	N/A	Low	Y		
Heartwater	Y	N	Y	Y	N	N	Low - Mod	N/A	Negligible	Negligible	N/A	N/A	N		
Q fever	Y	Y	Y	N	N	Y	Mod	Low	Moderate	Very Low	Very Low	Moderate - High	Y		
Besnoitia	N	N	Y	N	N	N	Very Low	N/A	Negligible	Negligible	N/A	N/A	N		
Bovine babesiosis	Y	Y	Y	Unknown	Unknown	N	N/A	N/A	N/A	N/A	N/A	N/A	N		
Theileriosis	Y	N	Y	N	N	N	N/A	N/A	N/A	N/A	N/A	N/A	N		
Surra	Y	N	Y	Y	Y	N	Low	N/A	Low	Very Low	N/A	Moderate	Y		
Tsetse fly associated trypanosomosis	Y	Y	Y	Y	Y	N	Low	N/A	Low	Very Low	N/A	Moderate	Y		
Screw-worm fly (New world)	Y	Y	Y	Y	Y	N	Moderate	N/A	Negligible	N/A	N/A	N/A	N		
Screw-worm fly (Old world)	Y	Y	Y	Y	Y	N	Moderate	N/A	Negligible	N/A	N/A	N/A	N		
Bovine spongiform encephalopathy	Y	Y	Y	N	N	N	Very Low	N/A	Negligible	N/A	N/A	N/A	N		
Internal parasites	Some	Y	Y	Y	Y	N	High	N/A	Low	Very Low	N/A	Very Low	Y		
External parasites	Some	Y	Y	Y	Y	N	High	N/A	Moderate	Low	N/A	Moderate	Y		
Seeds	N	N	Y	Y	Y	N	Moderate	N/A	Low	N/A	N/A	Low	Y		
							N: No	**Y: Yes**	**Ab: Antibodies**	**N/A: Not applicable**					

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