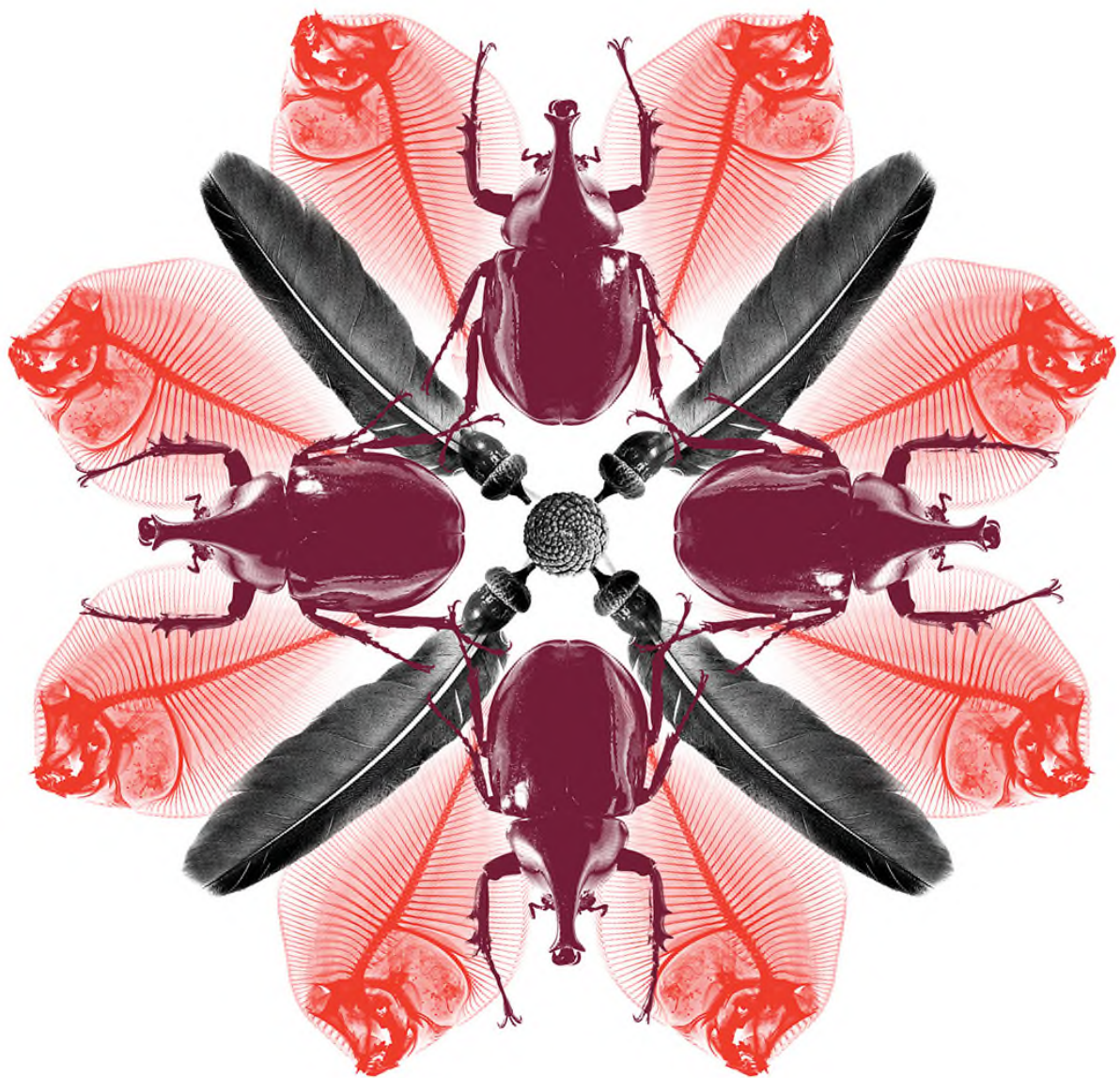


# Importation of zoo bovids and their semen from approved countries

Draft report

March 2019



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### **Stakeholder submissions on draft reports**

This draft report has been issued to give all interested parties an opportunity to comment on relevant technical biosecurity issues, with supporting rationale. A final report will then be produced taking into consideration any comments received.

Submissions should be sent to the Australian Government Department of Agriculture and Water Resources following the conditions specified within the related Biosecurity Advice, which is available at: [agriculture.gov.au/biosecurity/risk-analysis/memos](http://agriculture.gov.au/biosecurity/risk-analysis/memos).

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# Acronyms and abbreviations

Term or abbreviation	Definition
Ab	Antibody
ACT	Australian Capital Territory
Ag	Antigen
ALOP	Appropriate level of protection
AHA NAMP	Animal Health Australia National Arbovirus Monitoring Program
AQIS	Australian Quarantine and Inspection Services
AUSVETPLAN	Australian Veterinary Emergency Plan
BA	Biosecurity Advice
BICON	Australia's Biosecurity Import Condition System
BIRA	Biosecurity import risk analysis
BTEC	Brucellosis and Tuberculosis Eradication Campaign
cELISA	competitive enzyme-linked immunosorbent assay
CFT	Complement Fixation Test
CITES	Convention on International Trade in Endangered Species of Wild Fauna and Flora
EADRA	Emergency animal disease response agreement
ELISA	enzyme-linked immunosorbent assay
FAO	Food and Agriculture Organization of the United Nations
IRA	Import risk analysis
NSW	New South Wales
NT	Northern Territory
OIE	World Organisation for Animal Health
OIE Code	OIE Terrestrial Animal Health OIE Code 2018
OIE Manual	OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2018
PEQ	pre-export quarantine
PCR	polymerase chain reaction
PAQ	post-arrival quarantine
QAP	quarantine approved premises
Qld	Queensland
SA	South Australia
SPS Agreement	WTO agreement on the Application of Sanitary and Phytosanitary Measures

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<b>Term or abbreviation</b>	<b>Definition</b>
Tas.	Tasmania
Vic.	Victoria
the department	The Australian government Department of Agriculture and Water Resources
VNT	Virus Neutralisation Test
WA	Western Australia
WTO	World Trade Organization

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# Summary

This draft risk review takes into consideration the biosecurity risks for Australia associated with the importation of zoo bovids and their semen from approved countries. It includes relevant peer-reviewed scientific information, advice from international scientific experts, and relevant changes in industry practices and operational practicalities. Australia currently only permits the importation of zoo bovids from New Zealand.

This draft risk review proposes that the importation of zoo bovids and their semen to Australia from approved countries be permitted, subject to a range of biosecurity measures.

This draft risk review was conducted by the Department of Agriculture and Water Resources with the assistance of technical and scientific experts. The draft risk review identifies hazards that require biosecurity measures to manage risks to a very low level in order to achieve Australia's appropriate level of protection (ALOP). The hazards requiring specific measures are:

- Anthrax
- Brucellosis (*B. abortus* & *B. melitensis*)
- Bovine tuberculosis
- Bluetongue virus
- Bovine viral diarrhoea (type 2)
- Contagious caprine pleuropneumonia
- Foot-and-mouth disease
- Haemorrhagic septicaemia
- Heartwater
- Lumpy skin disease
- Malignant catarrhal fever (Wildebeest associated)
- Peste des petits ruminants
- Rabies
- Rift Valley fever
- Schmallenberg virus
- Screw worm fly myiasis
- Surra
- Trypanosomosis
- Transmissible spongiform encephalopathies
- Vesicular stomatitis

This draft risk review proposes a combination of risk management measures and operational systems that will reduce the biosecurity risk associated with the importation of live zoo bovids

and their semen from approved countries into Australia to achieve Australia's ALOP. Attention has been given to diseases that could have an impact on domestic livestock and diseases of trade importance.

This draft risk review recognises that general risk management measures common to most current Australian import requirements for zoo animals may achieve Australia's appropriate level of protection in relation to the likelihood of entry of a number of diseases of zoo bovids. Such measures include:

- 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 approved by the department. The residency requirement may be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.
- 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 the disease of biosecurity concern identified in this review (e.g. post-mortem records for deceased animal; disease testing programs; 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 policy.
  - The required outcome of a *Health monitoring program* 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. This underpins official certification.
- The animal must be held in pre-export quarantine for at least 30 days and isolated from all other animals not eligible for export to Australia, during which it is inspected at least daily for signs of disease, treated effectively for internal and external parasites, and tested for diseases in accordance with Australian entry requirements.
- The animal must be transported to a quarantine approved premises in Australia 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 of at least 30 days.
- The pre-export quarantine facility has documented standards of how it will meet Australian requirements.
- The receiving institution must be approved under relevant Australian state or territory legislation to hold the species being imported.

General risk measures relevant to semen are:

- The donor 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 collection, unless otherwise approved by the department. The residency requirement may

be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.

- 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 the disease of biosecurity concern identified in this review.
  - 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 policy.
  - The required outcome of a *Health monitoring program* 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 (e.g. post-mortem records for deceased animals; disease testing programs; etc.). This underpins official certification.
- The animal 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*.
- A semen collection period (or 'collection') starts on the first day semen is collected from the donor and finishes on the last day semen is collected, up to a maximum of 30 days. (A new collection period may begin the day after and is required to meet conditions applicable to that new time frame).
- The receiving institution must be approved under relevant Australian state or territory legislation to hold the imported semen.

Additional assumptions for zoo bovids and their semen this policy is based on are:

- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs including post mortem investigation of deceased animals and are under veterinary supervision.
- For the 30 days immediately before export the animal showed no clinical signs or other evidence of the diseases retained for risk review in this policy.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

The department recognises that there might be new scientific information and technologies, or other combinations of measures that may provide an equivalent level of biosecurity protection for the diseases identified as requiring risk management. Submissions requesting consideration of equivalence for alternative measures will be considered on a case-by-case basis and in light of available evidence.

This draft risk review contains details of the risk reviews for each of the identified hazards and the proposed biosecurity measures. Interested parties are invited to provide their comments on the draft in the form of submissions to the department within the consultation period.

# 1 Introduction

## 1.1 Australia's biosecurity policy framework

Australia's biosecurity policies aim to protect Australia against the risks that may arise from exotic pests entering, establishing and spreading in Australia, thereby threatening Australia's unique flora and fauna, and agricultural industries that are relatively free from serious pests and diseases, and human health.

Successive Australian Governments have maintained a conservative, but not a zero risk, approach to the management of biosecurity risks. This approach is expressed in terms of Australia's appropriate level of protection (ALOP), which reflects community expectations through government policy and is currently described as providing a high level of protection aimed at reducing risk to a very low level, but not to zero.

The risk analysis process is an important part of Australia's biosecurity system. It enables the Australian Government to formally consider the level of biosecurity risk that may be associated with proposals to import goods into Australia. If the biosecurity risks are higher than the ALOP for Australia, risk management measures are proposed to reduce the risks to an acceptable level. If the risks cannot be reduced to an acceptable level, the goods will not be imported into Australia, until suitable measures are identified.

Australia's risk analyses are undertaken by the Australian Government Department of Agriculture and Water Resources (the department) using technical and scientific experts in relevant fields, and involve consultation with stakeholders at various stages during the process.

Risk analyses may take the form of a biosecurity import risk analysis (BIRA) or a non-regulated risk analysis (such as scientific review of existing policy and import conditions, or scientific advice).

Further information about Australia's biosecurity framework is provided in the *Biosecurity Import Risk Analysis Guidelines 2016* located on the [Australian Government Department of Agriculture and Water Resources](#) website.

The Department of Agriculture and Water Resources recognises that there might be new scientific information and technologies, or other combinations of measures that may provide an equivalent level of biosecurity protection for the disease agents identified as requiring risk management. Submissions supporting equivalence measures will be considered on a case-by-case basis.

## 1.2 This policy review

### 1.2.1 Background

Family Bovidae, order Artiodactyla, contains cloven-hoofed, ruminant mammals. Bovidae covers a variety of species including several antelope species. 'Zoo bovids' are species within the family Bovidae that are exhibited in zoos, i.e. most species other than domestic cattle, water buffalo, sheep, and goat breeds.

Zoo bovids are kept in a variety of housing conditions. Some zoo collections hold small numbers of animals in open or enclosed exhibits. Other zoos may house them in large, open range style exhibits. Zoo animals are rarely housed in large herds or flocks, unlike domesticated bovidae. Standard procedure is for zoo animals to be housed in small groups or individually to allow individual animals to be closely monitored every day. Zoo bovids are segregated from domestic livestock in Australia. Generally, zoological institutions have well-developed preventative health programs with well-maintained, written health and husbandry records for each individual animal. Many zoos employ veterinarians and/or veterinary paraprofessionals with expertise in wildlife and exotic zoo animals. Some zoos without a staff veterinarian engage a local veterinary service, on a contract basis, to implement their preventative health program and respond on a case-by-case basis to health concerns in their animals.

Zoo bovids, as for most zoo animals in approved countries, are sourced from multi-generational captive stock. Direct acquisitions from the wild are subject to the requirements of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES requirements).

A common practice for zoos is to investigate the deaths of collection animals through a full post-mortem examination conducted by the zoo's veterinary service, followed, as necessary, by histopathological and other diagnostic testing. The zoo's veterinary service or animal manager may make a risk-based assessment of the intensity of investigation required. Non-collection animals (wildlife rehabilitation cases) may also receive a full post-mortem investigation.

Zoos have standard procedures for retrieving, storing and disposing of zoo animal carcasses, and for post-mortem reporting and investigation. These include appropriate use of personal and environmental protection and decontamination. As per the National Zoo Biosecurity Manual, animals culled within the zoo grounds are not fed out to other collection animals, unless the veterinary service has assessed the risk of transmissible diseases and the practice is compliant with state/ territory regulations covering swill feeding and TSE guidelines (Reiss and Woods, 2011).

Zoo bovids have a close phylogenetic relationship to domestic livestock and can potentially carry and transmit many diseases that could have an impact on domestic livestock industries. Some of these diseases could also have an immediate impact on trade, even if only reported from a single animal within a zoo setting. For example, a confirmed case of foot-and-mouth disease in a zoo may result in immediate trade restrictions and economic losses.

A further element of risk is the consequence to Australia's zoo industry, including captive breeding programs, and the commercial impact on the importing zoo. Some of the diseases reviewed have significant consequences, particularly for Australia's livestock industries, trade, and Australia's animal health status. In some instances onshore treatment of an animal may not adequately address the biosecurity risk and therefore the animal may be required to be re-exported or destroyed. Either outcome is significantly undesirable. This draft risk review considers these consequences when proposing biosecurity import conditions in addition to the general risks to Australia.

Where possible this draft risk review has reviewed literature on zoo bovids in a zoo setting. However, given the relative paucity of literature on some topics, literature from wildlife studies,

domestic livestock, and general disease principles are also considered in order to gather sufficient information to make a judgement for the context of zoo bovids.

Serosurveillance studies in wild bovids are the most common form of study for practical reasons but require care when interpreting. The presence of antibodies provides limited information as to the role of a species in the ecology of a disease, however, high seroprevalence rates within populations suggest the existence of endemic cycles (Passler et al. 2009). Gilbert et al. (2013) also discusses these considerations when interpreting wildlife serological studies.

Judgement is also required regarding the usage of tests and vaccinations in zoo bovids as they are not often validated or registered for use for those species. In addition, collection of semen from domestic cattle, water buffalo, sheep, and goats occurs in line with principles for disease management that are well established. However, collection of semen from zoo bovids is operationally different in a zoo setting. Extrapolation of applicable disease management principles for semen collected under those circumstances is required.

### **1.2.2 Scope**

The scope of this draft risk review is to consider the biosecurity risk that may be associated with the importation into Australian zoos of zoo bovids and their semen from approved, licensed or registered zoos or wildlife parks in Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Japan, Luxembourg, Netherlands, Portugal, Singapore, Spain, Sweden, the United Kingdom, and the United States. These countries are hereafter referred to as approved countries.

This policy covers all species from the Bovidae family except for those in the *Bovini* tribe and *Caprinae* subfamily (domestic cattle and buffalo, sheep and goats). Species from the genus *Connochaetes* are also excluded as a result of the risk review chapter on Malignant Catarrhal Fever – Wildebeest Associated.

### **1.2.3 Existing policy**

#### **International policy**

Import policy exists for zoo bovids from New Zealand. The import requirements can be found at the department's [online import conditions database, BICON](#).

The department has considered all the pests previously identified in the existing zoo, beef and germplasm policies and where relevant, the information in those assessments has been taken into account in this review.

#### **Domestic arrangements**

The Australian Government is responsible for regulating the movement of animals and animal products into and out of Australia. However, Australia's state and territory governments are responsible for animal health and environmental controls within their territories. Once animals and animal products have been released from biosecurity control by Australian Government biosecurity officers, they are subject to state and territory controls. It is the importer's responsibility to identify, and ensure compliance with all requirements.

### **1.2.4 Next Steps**

This draft review gives stakeholders the opportunity to comment and draw attention to any scientific, technical, or other gaps in the data, misinterpretations and errors.

The department will consider submissions received on this draft review and may consult informally with stakeholders. The department will then prepare a final report, taking into account stakeholder comments.

The final review will be published on the department's website along with a notice advising stakeholders of the release. The department will also notify the proposer, the registered stakeholders and the Secretariat of the World Trade Organization (the WTO) about the release of the final report. Publication of the final report represents the end of the process. The conditions recommended in the final report will be the basis of any import permits issued and they inform the development of negotiated veterinary health certification to be issued by an exporting country.

## 2 Method

Australia is one of the one hundred and eighty two countries that are members of the World Organisation for Animal Health (the OIE). The OIE's Terrestrial Animal Health Code (the OIE Code) describes 'General obligations related to certification' in Chapter 5.1 (OIE 2015b).

The OIE Code states in Article 5.1.2. that:

The import requirements included in the international veterinary health certificate should assure that commodities introduced into the importing country comply with the standards of the OIE. Importing countries should align their requirements with the recommendations in the relevant standards of the OIE. If there are no such recommendations or if the country chooses a level of protection requiring measures more stringent than the standards of the OIE, these should be based on an import risk analysis conducted in accordance with Chapter 2.1.

Article 5.1.2. further states that:

The international veterinary health certificate should not include measures against pathogens or diseases which are not OIE listed, unless the importing country has demonstrated through import risk analysis, carried out in accordance with Section 2, that the pathogen or disease poses a significant risk to the importing country.

The components of import risk analysis as described in Chapter 2.1. of the OIE Code are:

- hazard identification
- risk assessment (entry assessment, exposure assessment, consequence assessment and risk estimation)
- risk management
- risk communication.

Hazard identification, risk assessment and risk management are sequential steps within a risk analysis. Risk communication is conducted as an ongoing process, and includes both formal and informal consultation with stakeholders.

### 2.1 Risk review

Although not defined or described in the OIE Code, risk review is recognised by risk analysts as an essential component of the risk analysis process (Barry 2007; FSA 2006; Purdy 2010).

Australia applies a process of risk review to the biosecurity risks associated with the importation of an animal commodity (animal product or live animal) for which biosecurity measures have already been developed.

Risk review differs from *the monitoring and review component* of risk management, as described in the OIE Code, in that each component of the risk analysis process (hazard identification, risk assessment and risk management) is reviewed under the risk review process. If a change (either an increase or a decrease) in the biosecurity risk associated with a live animal or animal product

that is currently imported into Australia is identified based on updated scientific information, risk management measures can be revised accordingly.

This draft risk review has drawn on the following sources of information (this list is not exhaustive):

- the OIE Code (OIE 2018c)
- Australia's existing import policies, e.g. *Importation of zoo bovids from New Zealand*
- a review of relevant scientific literature
- expert opinion coordinated through the Australasian Zoo and Aquarium Association (ZAA).

Risk, defined by the OIE Code as 'the likelihood of the occurrence and the likely magnitude of the biological and economic consequences of an adverse event or effect to animal or human health', is dynamic in nature; it changes with time. Consequently, risk should be kept under regular review.

## 2.2 Review of hazard identification

Hazard identification is described in the OIE Code (Article 2.1.2) as a classification step that is undertaken to identify potential hazards that may be associated with the importation of a commodity.

In accordance with the OIE Code, a disease agent was considered to be a potential hazard relevant to the importation of zoo bovids and their semen if it was assessed to be:

- appropriate to the species being imported
- OIE-listed, emerging and/or capable of producing adverse consequences in Australia.

Disease agents in previous policy reviews or import risk assessments for zoo animals, conducted by the Department of Agriculture and Water Resources, were also considered as potential hazards.

A hazard was retained for further review (hazard refinement) if:

- it was not present in Australia, or present in Australia and a notifiable disease or subject to official control or eradication
- it was present in the country of export (approved countries).

OIE-listed diseases not present in the country of export were subject to further review if there were biosecurity measures for other zoo animals in Australian import requirements and evidence to associate zoo bovids and their semen in disease transmission.

Where evidence for the inclusion or exclusion of a particular disease agent was equivocal, a judgement was made based on the strength of the available evidence to implicate zoo bovids in disease transmission.

## 2.3 Review of risk assessment

Details of the risk assessment process relevant to live animals are provided in Chapter 2.1 of the OIE Code.

A review of risk factors relevant to the entry, establishment, and spread assessment of hazards identified for further review was conducted to identify any significant changes in disease agent attributes and/or geographic distribution that would be relevant to biosecurity considerations for Australia.

A review of peer-reviewed scientific literature was conducted for each hazard retained for risk review. If definitive information on risk factors was not found through literature review or contact with relevant experts, then any uncertainties were identified and documented.

Based on the information reviewed, a conclusion was reached for each hazard about whether a biosecurity risk was present that was relevant when considering the importation of zoo bovids and their semen into Australia. Any assumptions and/or judgements made in drawing conclusions for each hazard retained for further review were documented in the relevant risk review section (Chapter 4).

## 2.4 Review of risk management

This draft risk review focused on determining whether risk management was warranted for each of the hazards identified for the importation of zoo bovids and their semen.

Risk evaluation is defined in the OIE Code as the process of comparing the risk estimated in the risk assessment with the reduction in risk expected from the proposed risk management measures. The conclusions drawn from the risk reviews conducted for each hazard are used as the basis for risk evaluation during this policy review. A judgement was then made to determine whether risk management was warranted to achieve Australia's ALOP.

This draft risk review also considered the efficacy of long standing general zoo policy designed to manage the risks and animal welfare issues associated with the importation and handling of wild animal species. Those general risk management measures are implemented through application of that policy and include:

- 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 approved by the department. The residency requirement may be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.
- 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 the diseases of biosecurity concern identified in this review.

- 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 policy.
- The required outcome of a *Health monitoring program* 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 (e.g. post-mortem records for deceased animals; disease testing programs; etc.). This underpins official certification.
- The animal must be held in pre-export quarantine for at least 30 days and isolated from all other animals not eligible for export to Australia, during which it is inspected at least daily for signs of disease, treated effectively for internal and external parasites, and tested for diseases in accordance with Australian entry requirements.
- The animal must be transported to a quarantine approved premises in Australia 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 of at least 30 days.
- The pre-export quarantine facility has documented standards of how it will meet Australian requirements.
- The receiving institution must be approved under relevant Australian state or territory legislation to hold the species being imported.

General risk measures relevant to semen are:

- The donor 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 collection, unless otherwise approved by the department. The residency requirement may be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.
- 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 the disease of biosecurity concern identified in this review (e.g. post-mortem records for deceased animal; disease testing programs; 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 policy.
  - The required outcome of a *Health monitoring program* is regular monitoring, ongoing surveillance, and veterinary oversight to ensure that the health status of animals and an institution is known and monitored over time. This underpins official certification.

- The animal 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*.
- A semen collection period (or 'collection') starts on the first day semen is collected from the donor and finishes on the last day semen is collected, up to a maximum of 30 days. (A new collection period may begin the day after and is required to meet conditions applicable to that new time frame).
- The receiving institution must be approved under relevant Australian state or territory legislation to hold the imported semen.

Additional assumptions for zoo bovids and their semen this policy is predicated on are:

- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs including post mortem investigation of deceased animals and veterinary supervision.
- For the 30 days immediately before export the animal showed no clinical signs or other evidence of the diseases retained for risk review in this policy.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

## 2.5 Risk communication

Risk communication is defined in the OIE Code as:

the interactive transmission and exchange of information and opinions throughout the risk analysis process concerning risk, risk-related factors and risk perceptions among risk assessors, risk managers, risk communicators, the general public and other interested parties.

In conducting import risk analyses and policy reviews, the department consults with the Australian Government Department of Health to ensure that public health considerations are included in the development of Australia's animal biosecurity policies. Furthermore, a formal process of consultation with external stakeholders is a standard procedure for all import risk analyses and risk reviews to enable stakeholder assessment and feedback on draft conclusions and recommendations about Australia's animal biosecurity policies.

### 3 Hazard identification

The list of diseases (hazards) of potential biosecurity concern was compiled from:

- diseases listed by the OIE as diseases or multiple species diseases affecting cattle, sheep and goats (OIE 2018c)
- diseases identified in previous policy reviews and import conditions of germplasm, beef, and zoo animals conducted by the Department of Agriculture and Water Resources
- other diseases identified as occurring in zoo bovids including emerging diseases.

The method of hazard identification and refinement is described in Section 2.1. The preliminary list of diseases/disease agents is shown in Table 1. This table summarises the results of the hazard refinement process, including the reason for removal or retention of each identified hazard.

Many disease agents are ubiquitous or common pathogens and may be present in Australia. There are others that are opportunistic, not reported to be pathogenic, or of uncertain relevance in commodity due to limited or insufficient information. These agents were considered when compiling the list of hazards of potential biosecurity concern.

The diseases retained after hazard identification and refinement in Table 1 are listed at the end of this chapter.

Where the department determined that a hazard is not present in the country of export, certification of country freedom from the disease caused by the hazard may be required. For country freedom from FMD, Australia refers to the current OIE classification of the country, but also makes its own assessment due to the extreme consequences of an FMD outbreak in Australia. The department maintains an FMD-free approved country list (Department of Agriculture and Water Resources 2016b), which reflects this assessment. For other hazards for which country free status may be appropriate, the department has reviewed the evidence for each hazard and each applicant country.

**Table 1 Hazard identification and refinement**

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
<i>Anaplasma bovis</i> (formerly <i>Erhlichia bovis</i> )	Ruminants	No	Yes	No	Yes (in tick-free areas)	No	Yes	No. Not reported in zoo bovids, not reported in approved countries.
Anthrax ( <i>Bacillus anthracis</i> )	Mammals and some birds	Yes	Yes	Yes	Yes	Yes, global distribution	No	Yes. OIE-listed, nationally notifiable in Australia and control measures are in place.
Aujeszky's disease	Pigs, other mammals recorded as dead-end hosts.	Yes	Yes	No	Yes	Yes	Yes	No. Pigs are the only source of virus dissemination. Spill over into other species is typically fatal (dead-end hosts) after a short incubation period (2-10 days). Controlled by non-specific measures (pre-export quarantine period and clinical inspection).
Babesiosis ( <i>Babesia bovis</i> and <i>B. bigemina</i> )	Ruminants including non-domestic bovidae	Yes	Yes	Yes	Yes (in tick free areas)	Yes	Yes	No. Controlled by non-specific measures (ectoparasite control and inspection).
Besnoitia ( <i>Besnoitia besnoiti</i> )	Cattle and other wild ruminants	No	Yes	No	No	Yes, emerging disease in Europe	Uncertain	Yes. Not present in Australia; capable of adverse consequences.
Bluetongue disease	Artiodactyla	Yes	Yes	Yes, 12 of 27 serotypes	Yes (clinical disease only)	Yes	No	Yes. OIE listed and nationally notifiable. Australia has several serotypes of BTV but approved countries have different serotypes. Australia has a national surveillance program to detect new serotypes and maintain a bluetongue virus zone map.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Border disease virus	Sheep	No	Yes	Yes	No	Yes	n/a	No. Present in Australia, no evidence zoo bovidae involved in epidemiology.
Borna disease	Equids, sheep mainly; cattle, camelids, dogs, cats, ostriches	No	Yes	No	Yes	Yes	n/a	No. No evidence zoo bovidae involved in epidemiology. Reviewed in Perissodactyl and Horse IRAs and no specific risk management measures prescribed due to limited risk pathways and geographic distribution.
Bovine anaplasmosis ( <i>Anaplasma marginale</i> )	Ruminants including non-domestic bovidae	Yes	Yes	Yes	Yes (in tick free areas)	Yes	Yes	No. Notifiable in the tick free areas, and tick controls in place for zoning reasons where cattle move across zone boundaries. Controlled by non-specific measures (ectoparasite control and inspection).
Bovine brucellosis ( <i>Brucella abortus</i> )	Wide range of mammals	Yes	Yes	No	Yes	Yes	No	Yes. OIE listed, not present in Australia, nationally notifiable.
Bovine genital campylobacteriosis	Cattle	Yes	Yes	Yes	No	Yes	n/a	No. Present in Australia, no control measures in place.
Bovine herpesvirus 4	Ruminants	No	Possible but generally considered non pathogenic	No.	No	Yes	No	No. Not notifiable, considered non-pathogenic, existing commodity pathways (e.g. germplasm) do not apply biosecurity controls. Not noted in zoo literature.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Bovine herpesvirus 5	Cattle	No	Yes	Yes	No	Yes	No	No. Present in Australia, not controlled.
Bovine tuberculosis ( <i>Mycobacterium bovis</i> )	Wide range of mammals	Yes	Yes	No	Yes	Yes	No	Yes. OIE listed, not present in Australia; zoo bovids susceptible, cases reported in zoos.
Bovine virus diarrhoea type 2 (Pestivirus)	Bovidae	Yes	Yes	No, only Type I	Yes	Yes, worldwide	No	Yes. OIE listed, Australia is free from Type 2 bovine viral diarrhoea virus, which is more virulent.
Cache Valley virus	Sheep primarily; deer thought to be reservoir; antibodies in wide host range reported including goats, cervidae, horses, jackrabbits, etc.	No	Yes	No	No	Yes, USA & CA	N/A	No. No evidence that zoo bovidae have a role in epidemiology.
Caprine and ovine brucellosis ( <i>Brucella melitensis</i> )	Wide range of mammals	Yes	Yes	No	Yes	Yes	No	Yes. OIE listed, not present in Australia; wide range of mammals susceptible; nationally notifiable.
Caprine arthritis and encephalitis virus	Caprinae	Yes	Yes	Yes	No	Yes	No	No. Present in Australia; not a nationally notifiable disease

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Chagas' disease ( <i>Trypanosoma cruzi</i> )	Mammals, but dogs and humans most often; pigs and cats.	No	Yes	No	Yes	Yes, range expanding	No	No; no evidence that zoo bovidae are involved in epidemiology, wildlife reservoirs include nonhuman primates, raccoons, woodrats, opossums, etc.
Contagious agalactia ( <i>Mycoplasma agalactiae</i> )	Sheep, goats, some wild caprinae	Yes	Yes	Reported, but last occurrence unknown	Yes	Yes	No	No. No evidence zoo bovidae covered in this policy have a role (a couple of reports exist but only in ibex and chamois – caprinae subfamily).
Contagious bovine pleuropneumonia ( <i>Mycoplasma mycoides subsp. mycoides SC</i> )	Bovids from the genus <i>Bos</i> and the genus <i>Bubalus</i>	Yes	Yes	No	Yes	No. Sub Saharan Africa, possibly Asia	No	No. Species specific disease and no reports in the zoo bovidae species covered by this policy.
Contagious caprine pleuropneumonia ( <i>Mycoplasma capricolum subsp. capripneumoniae</i> )	Goats and other non-domestic bovidae, occasionally sheep	Yes	Yes	No	Yes	No; Africa, Middle East, possibly Asia; exact distribution not known	No	Yes. OIE listed, not present in Australia, reported in wild bovids of this policy, exact geographical distribution not known.
Cybernetic Congo haemorrhagic fever	Mammals including humans	Yes	Yes	No	Yes	Yes, Mediterranean and southern and eastern Europe, Asia etc.	Uncertain	Yes. OIE listed, not present in Australia

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
<i>Cysticercus bovis</i> ( <i>Taenia saginata</i> )	Cattle	No	Yes	Yes	Yes	Yes	Yes	No. Whilst not present in Australia and notifiable, zoo bovidae are dead end hosts as there is no access to them from definitive, carnivore hosts.
East Coast Fever and Mediterranean Theileriosis ( <i>Theileria parva</i> and <i>T. annulata</i> )	Ruminants including cattle, buffalo and other antelope.	Yes	Yes	No	Yes	No. <i>T. parva</i> in eastern and southern Africa. <i>T. annulata</i> in North Africa, southern Europe and Asia	No	Yes. OIE listed, not present in Australia. <i>T. parva</i> reported in waterbuck. <i>T. annulata</i> present in some approved countries.
Eastern, Western and Venezuelan Equine encephalomyelitis	Equines and humans, occasionally birds and other mammals	Yes	Yes	No	Yes	Yes	No	No. No evidence Zoo bovidae have any role in epidemiology. One dead-end host report in literature (domestic cow).
<i>Echinococcus granulosus</i>	Cattle and multiple other species including humans	Yes	Yes (limited)	Yes	No	Yes	Yes	No. Whilst not present in parts of Australia and notifiable, zoo bovidae are dead end hosts as there is no access to them from definitive, carnivore hosts.
<i>Echinococcus multilocularis</i>	Cattle and multiple other species including humans	Yes	Yes	No	Yes	Yes	Yes	No. Whilst not present in Australia and notifiable, zoo bovidae are dead end hosts as there is no access to them from definitive, carnivore hosts.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Enzootic abortion of ewes ( <i>Chlamydomphila abortus</i> )	Goats, sheep, less commonly cattle, pigs, horses and deer.	Yes	Yes	Yes pathogen present but not syndrome of enzootic abortion in ewes	Yes	Yes	No	No. No evidence that zoo bovidae have any role. Wild caprinae were shown to have antibodies in a recent study, but a literature search, AUSVETPLAN, OIE, and other sources don't have any evidence of a role for zoo bovidae.
Enzootic bovine leucosis (bovine leukaemia virus)	Cattle primarily; sheep, water buffalo, capybara	Yes	Yes	Yes (but with a free compartment )	Yes	Yes; Europe, N America	No	No. No evidence zoo bovidae have a role in epidemiology. There is a set of experimental infections in the literature only. Present in Australia.
Epizootic haemorrhagic disease (clinical disease)	Cervidae primarily; cattle, caprinae, occasional other species including rhinos, black bears, oryx, etc.	Yes	Yes, strains present in Australia but no clinical disease reported	Yes. All serotypes except #4. No clinical disease reported	Yes (clinical disease)	Yes.	No	No. No evidence zoo bovidae have a role in epidemiology. All but serotype 4 in Australia; serotype 4 is only reported in Africa. Clinical disease notifiable only, not under official controls.
External parasites	Wide range of mammals	No	Yes	Yes	Not specifically listed but novel detections are under Australian veterinary systems	Yes	Yes	No. General zoo conditions require external inspection of animals and treatment of external and internal parasites. External inspection also occurs in post-arrival quarantine.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Flavivirus encephalitides (tick-borne)	Ruminants, goats, sheep, cows, dogs	No	Yes	No	Yes	Yes, Central Europe and North America	Yes	No. No evidence zoo bovidae have a role in epidemiology. Mammals other than rodents are spill over dead-end hosts. Large ruminants serve as feeding hosts for ticks but are not involved in actual virus epidemiology. Baseline zoo conditions require parasite treatment and visual inspection for wounds, ticks, etc.
Foot and mouth disease	Artiodactyla	Yes	Yes	No	Yes	No	No	Yes. OIE-listed, not present in Australia, zoo bovidae are susceptible, disease of significant adverse consequences to Australia.
Getah virus infection	Horses and pigs, other warm blooded species, including humans occasionally	No	Yes	No	Yes	Yes. East Asia, including Japan	N/A	No. No evidence zoo bovidae have a role in epidemiology. Rare spill over events into multiple species types (including humans).
Haemorrhagic septicaemia ( <i>Pasteurella multocida</i> serotypes B2 6:B & E2 6:E)	Ruminants, camels, deer, hares, horses, pigs, elephants, birds, potentially humans	Yes	Yes	No	Yes	Yes	No	Yes. OIE listed, nationally notifiable, not present in Australia.
Heartwater ( <i>Cowdria ruminantium</i> )	Many species ruminants including wildlife	Yes	Yes	No	Yes	No	No	Yes. OIE listed, not present in Australia.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Infectious bovine rhinotracheitis ( <i>Bovine herpesvirus 1</i> )	Cattle, sheep, goats, wild artiodactyla	Yes	Yes	Yes, but only subtype 1.2b. Subtypes 1.1 and 1.2a are absent	No  Not specifically listed but novel detections are under Australian veterinary systems	Yes	No	Yes. Infection is lifelong and potential transmission from species of this policy. Adverse consequences if the more virulent strains are introduced to Australia.
Internal Parasites	Wide range of mammals	No	Yes	Yes		Yes	Yes	No. General zoo conditions require treatment of external and internal parasites.
Jembrana disease	Cattle ( <i>Bos</i> spp.) and water buffalo	No	Yes	No	Yes	No. Only in Indonesia	No	No. No evidence zoo bovidae have a role in epidemiology or are susceptible. Not in approved countries.
Leishmaniasis	Humans and dogs primarily. Occasional reports in other species.	Yes	Yes	Yes, a single novel species found in macropods in a discrete location.	Yes	Yes.	No	No. No evidence that zoo bovidae are involved in epidemiology. Occasional reports in zoo animals but for species of Carnivora order or rodents and nonhuman primates. Australia lacks the competent vectors for the species important to trade.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Leptospirosis	Wide range of mammals	No	Yes	Yes	No	Yes	No	No. Minimal domestic controls on existing leptospirosis strains. Not a very infectious disease. Various reviews including the 2013 Dog & Cat IRA have so far only determined <i>L. Canicola</i> serovar of concern to Australia for imports. Few wild animal reservoirs have been identified and wild bovids have a low seroprevalence.
Louping ill	Sheep and grouse. Other warm blooded species occasionally infected.	No	Yes	No	Yes	Yes, UK and Norway	Yes	No. No evidence zoo bovidae have a role in epidemiology. The 2012 Perissodactyl policy and a literature search do not reveal any suitable vectors in Australia. Only sheep and grouse are known to develop sufficient viremia to transmit to ticks. Disease transmission primarily via tick and transstadial pathways.
Lumpy skin disease virus	Cattle, wild artiodactyla, occasionally sheep and goats	Yes	Yes	No	Yes	No, Africa	No	Yes. OIE-listed, not present in Australia and nationally notifiable. Emerging transboundary disease.
Maedi-visna virus	Sheep and goats primarily; occasionally wild caprinae species	Yes	Yes	No	Yes	Yes	No	No. No evidence zoo bovidae are involved in epidemiology.
Malignant catarrhal fever (wildebeest-associated) Alcelaphine herpesvirus-1 (AIHV-1)	Artiodactyla	No	Yes	No, only ovine form	Yes	Yes	Uncertain	Yes. Nationally notifiable; not present in Australia.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
MERS-CoV (Middle East respiratory syndrome coronavirus)	Dromedary camels, humans	No	Yes	No	No	No	N/A	No. Whilst emerging disease several studies have confirmed dromedary camels to be the reservoir. No evidence zoo bovidae are involved in epidemiology. Not in approved countries.
Nairobi sheep disease	Sheep, goats primarily but also wild artiodactyla	Yes	Yes	No	Yes	No, Africa, India	Uncertain	Yes. OIE-listed, not present in Australia and nationally notifiable.
Ovine epididymitis ( <i>Brucella ovis</i> )	Sheep	Yes	Yes	Yes	No	Yes	N/A	No. Present in Australia, disease only in domestic sheep.
Ovine herpesvirus-2 (sheep associated MCF)	Sheep. Spill over into other artiodactyls	No	No	Yes	No	Yes	N/A	No. Present in Australia, no control measures in place. Also covered in risk review on Malignant Catarrhal fever (wildebeest-associated).
Paratuberculosis (Johne's disease) ( <i>Mycobacterium avium subsp. paratuberculosis</i> )	Ruminants	Yes	Potentially; subject to control in regions	Yes	Yes	Yes	Yes	No. Managed by non-specific import requirements.
Peste des petits ruminants	Goats and sheep, wild artiodactyla	Yes	Yes	No	Yes	No (other than Bulgaria 2018). Africa and Asia	No	Yes. OIE listed, not present in Australia.
Pulmonary adenomatosis (Jaagsiekte)	Sheep. Rarely goats.	No	Yes	No	Yes	Yes. Europe and North America	No	No. No evidence that zoo bovidae are involved in epidemiology.
Q fever ( <i>Coxiella burnetii</i> )	Wide range of mammals	Yes	No (already present)	Yes	No	Yes	No	No. Present in Australia. Existing control measures are for human health purposes, not biosecurity.

Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Rabies	All mammals including humans	Yes	Yes	No	Yes	Yes	Uncertain	Yes; OIE-listed, not present in Australia.
Rift Valley fever virus	Artiodactyla, primates, rodents, humans	Yes	Yes	No	Yes	No. Africa, Middle East.	No	Yes; OIE listed, not present in Australia.
Salmonellosis (S. abortus-equi)	Equids	No	Yes	No	Yes	Yes	No	No. No evidence that zoo bovidae are involved in epidemiology
Salmonellosis (S. abortus-ovis)	Sheep primarily; occasionally goats and rabbits.	Yes	Yes	No	Yes	Yes	No	No. No evidence that zoo bovidae are involved in epidemiology.
Schmallenberg virus	Ruminants, pigs, camelids, elephants, perissodactylids	No	Yes	No	No	Yes. Europe.	No	Yes. Emerging disease of concern, zoo bovidae susceptible but epidemiological role uncertain.
Screw-worm fly - New World ( <i>Cochliomyia hominivorax</i> )	All mammals including humans	Yes	Yes	No	Yes	No	Uncertain	Yes. Whilst general zoo conditions require external inspection of animals and treatment of external and internal parasites, disease of significant consequence and resistant to many common anti-parasitics.
Screw-worm fly - Old World ( <i>Chrysomya bezziana</i> )	All mammals including humans	Yes	Yes	No	Yes	No	Uncertain	Yes. Whilst baseline zoo conditions require external inspection of animals and treatment of external and internal parasites, disease of significant consequence and resistant to many common anti-parasitics.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Sheep pox and goat pox	Sheep and goats	Yes	Yes	No	Yes	No	No	No. Caprinae species, mainly domestic sheep and goats, are the only known susceptible species.
Sheep scab (Psoroptes)	Sheep primarily.	No	Yes	No	Yes	Yes.	Yes	No. General zoo conditions require external inspection of animals and treatment of external and internal parasites. Literature search shows psoroptes is susceptible to common anti-parasitics. Psoroptes mites found from non-sheep species do not seem to be capable of causing sheep scab. Sheep psoroptes may not be able to survive on non-sheep hosts.
Surra ( <i>Trypanosoma evansi</i> )	Mainly equids, camels, occasionally bovidae	Yes	Yes	No	Yes	No	No	Yes. OIE listed, not present in Australia.
Transmissible gastroenteritis	Pigs	Yes	Yes	No	Yes	Yes	No	No. Infection only in pigs.
Transmissible spongiform encephalopathies	Wide range of mammals	Yes, (BSE and scrapie)	Yes	No	Yes	Yes	No	Yes. OIE listed, not present in Australia and some cases reported in zoo bovidae.
Trichinellosis	Canids, pigs, horses, other flesh-eating mammals	Yes	Yes	No	Yes	Yes, N America and Europe	No	No. No evidence that zoo bovidae are involved in epidemiology as they are herbivores. The 2012 Perissodactyl policy and the horse IRA did not find a risk pathway for those herbivorous species.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Trichomonosis ( <i>Tritrichomonas foetus</i> )	Cattle	Yes	Yes	Yes	No	Yes	Yes	No. Not notifiable, present in Australia, not reported in zoo bovidae and managed by non-specific zoo import requirements.
Tsetse fly associated trypanosomiasis ( <i>Trypanosoma brucei</i> , <i>T. vivax</i> )	Wide range of mammals including non-domestic bovidae	Yes	Yes	No	Yes	No. Africa	No	Yes. OIE listed, not present in Australia.
Tuberculosis ( <i>Mycobacterium tuberculosis</i> )	Wide range of mammals, humans and non-human primate primarily.	No	Possible, if multi resistant strains introduced	Yes	No	Yes	N/A	No. Disease present in Australia in humans. Included in scope of some IRAs predominantly for zoonotic risk (e.g. non-human primates). Bovidae species are an unlikely source for <i>M. tuberculosis</i> ; testing overlaps with <i>M. bovis</i> and is covered under that chapter.
Tularaemia	Wide range of mammals including humans.	Yes	Yes	Yes	Yes	Yes	No	No. No evidence that zoo bovidae involved in epidemiology. Typically smaller mammals such as lagomorphs and rodents. Cattle seem resistant to infection with only a few clinical cases reported.
Vesicular exanthema	Pigs	No	Yes	No	Yes	No	No	No. No evidence that zoo bovidae are involved in epidemiology.
Vesicular stomatitis	Artiodactyla, horses, camelids, humans	No	Yes	No	Yes	Yes. USA and the Americas in general	No	Yes; nationally notifiable, not present in Australia. Clinically resembles FMD.

## Hazard identification

Disease (disease agent)	Susceptible species	OIE listed disease?	Adverse consequences in Australia?	Present in Australia?	Nationally Notifiable in Australia?	Present in approved countries?	Managed by non-specific import requirements?	Retained for risk review; and provide reasons
Warble-fly myiasis	Wide range of mammals	No	Yes	No	Yes	Yes	Yes	No. General zoo conditions require external inspection of animals and treatment of external and internal parasites. Literature search shows warble-fly is susceptible to common anti-parasitics.
Wesselsbron virus	Sheep, goats, rodents, birds, pigs, humans, wild artiodactyla	No	Yes	No	Yes	No. Disease only in sub Saharan Africa.	No	No. No evidence zoo bovidae are involved in epidemiology. There is a single study in 1997 that found antibodies to Wesselsbron in a variety of African wildlife (including elephants, zebras, and a couple species of zoo bovidae) but nothing else in literature recording disease, virus, etc. for these species (Barnard 1997).
West Nile virus infection – clinical disease	Horses, humans, birds, occasionally other mammals	Yes	Yes	Yes, other strains	Yes (clinical disease)	Yes	No	No. No evidence zoo bovidae are involved in epidemiology. Most mammals are dead-end hosts that cannot transmit virus to mosquitoes.
Yersinia ( <i>Y pestis</i> , <i>Y enterocolytica</i> , <i>Y paratuberculosis</i> )	Many mammal species including humans, however ungulates considered highly resistant to infection	No	Possibly, if new virulent strains enter Australia (or <i>Y pestis</i> )	Yes, except for <i>Y pestis</i>	No	Yes	No	No. Not nationally notifiable and ungulates including bovidae are considered highly resistant to infection. Fleas and rodents and lagomorphs are the principal hosts with other mammals being incidental hosts.

The following diseases were retained for risk review on the basis of the information provided in Table 1:

- Anthrax
- *Besnoitia besnoiti*
- Bluetongue disease
- Bovine tuberculosis
- Bovine viral diarrhoea (Type 2)
- Brucellosis (*B. abortus* & *B. melitensis*)
- Contagious caprine pleuropneumonia
- Crimean-Congo haemorrhagic fever
- East Coast fever & Mediterranean theileriosis
- Foot-and-mouth disease
- Haemorrhagic septicemia
- Heartwater
- Infectious bovine rhinotracheitis virus
- Lumpy skin disease
- Malignant catarrhal fever (wildebeest associated)
- Nairobi sheep disease
- Peste des petits ruminants
- Rabies
- Rift Valley fever
- Schmallenberg virus
- Screw-worm fly myiasis
- Surra
- Transmissible spongiform encephalopathies
- Trypanosomosis (tsetse fly associated)
- Vesicular stomatitis

## 4 Risk reviews

### 4.1 Anthrax (*Bacillus anthracis*)

#### 4.1.1 Background

Anthrax is an infectious bacterial disease of humans, animals and several species of birds. It is caused by a spore-forming bacterium, *Bacillus anthracis*, and is characterised by rapidly fatal septicaemia with widespread oedema, haemorrhage and necrosis.

Domesticated and wild ruminants are most susceptible, equids less susceptible and omnivores and carnivores relatively resistant. Although *B. anthracis* occurs worldwide, outbreaks occur most commonly in parts of Africa, Asia and the Middle East, with sporadic cases in Australia, Europe and the United States (OIE 2014a; Spickler 2007). Outbreaks can affect wildlife, and many wild bovidae are confirmed to be susceptible to anthrax (Bengis 2012; De Vos & Turnbull 2004; Gates, Elkin & Dragon 2001; Hugh-Jones & De Vos 2002).

Anthrax is an OIE-listed disease (OIE 2018c). It is present, although uncommon in Australia where it occurs in specific regions and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016).

#### 4.1.2 Technical information

##### Epidemiology

*B. anthracis* is thought to multiply almost exclusively inside the body and exists in the environment as dormant spores, which remain viable in the soil or in animal products for decades. However, there is experimental evidence of vegetative *B. anthracis* multiplying in soils on or around roots of grass seedlings (Saile & Koehler 2006) and of bacteriophages and earthworms providing *B. anthracis* with alternatives to sporulation for survival and possibly multiplication in the soil (Schuch & Fischetti 2009). Once the soil has been contaminated by spores, it is very difficult to decontaminate. Transmission occurs by entry through skin lesions, ingestion or inhalation of spores in soil or on plants. Live animals are only infectious to others once they have died, and bacteria or spores contaminate the environment (Animal Health Australia 2012). Contaminated bone meal and other feed can also spread anthrax, and flies can disseminate anthrax mechanically. Outbreaks are often associated with heavy rainfall, flooding, or drought (Spickler 2007).

The incubation period is generally 1–7 days, but spores can germinate in the lungs for up to six weeks post-infection (Spickler 2007). Anthrax does not form a carrier state in animals (with the possible exception of pigs). The movement of live animals can only spread anthrax to a new location if animals are in the incubation period of infection when moved, and subsequently die and release the bacteria or spores (Animal Health Australia 2012). There are no reports in the literature of the infectious agents of anthrax being present in semen. It is considered extremely unlikely that semen would be collected from a male during the bacteraemic period (Williams 2003).

The OIE Code recommends that cattle semen should be considered be a safe commodity with respect to anthrax, with no conditions imposed (OIE 2014a).

**Clinical signs**

Infection with *B. anthracis* in susceptible species generally results in acute or peracute systemic illness, often resulting in death. Affected animals usually die within 1–3 days, with some surviving up to seven days. *B. anthracis* is readily isolated from blood or tissues of a recently dead animal that died of anthrax.

**Diagnosis**

Detection of *B. anthracis* in blood smears from infected carcasses is the main method of diagnosis. A PCR assay has been developed to detect anthrax spores in soil. The organism can also be cultured from infected carcasses.

**Prevention**

Control measures for anthrax include vaccination, premises quarantine, movement controls and surveillance (Animal Health Australia 2012).

**4.1.3 Current biosecurity measures**

Australia's current biosecurity measures for anthrax include premises freedom. The OIE Code recommendations include premises freedom or vaccination (OIE 2014a).

**4.1.4 Risk review**

Anthrax is present in approved countries. It is present in Australia and is a nationally notifiable disease. For control purposes the implementation of disease control measures as described in Australia's emergency animal disease response plan (EADRP) are used and in accordance with relevant legislation.

The following key points are relevant to the biosecurity risk of anthrax in zoo bovidae:

- Anthrax has a wide host range including ruminants, rodents and primates. It causes significant mortalities and is also a human health risk.
- Anthrax is present, albeit uncommon, in Australia. It occurs in specific regions and is subject to biosecurity control measures when it occurs.
- All species of bovids are considered highly susceptible to anthrax and outbreaks have been reported in wild bovids.
- Affected animals are only considered infectious to others once they have died and the carcass releases spores or bacteria.
- There is no carrier state recognised in bovids.
- There is no evidence that semen or artificial insemination poses a risk for transmission of anthrax between hosts.
- The only disease transmission risk in movement of animals is if they are in the short incubation period at the time of movement, and subsequently succumb to the infection.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.1.5 Conclusion

Anthrax is present in approved countries. Based on the preceding information, risk management measures are warranted for live animals. No measures are warranted for zoo bovidae semen.

Australia's biosecurity measures for anthrax for zoo bovids are:

- For 20 days immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of anthrax has occurred in any species during the previous 20 days and the disease is compulsorily notifiable.

## 4.2 *Besnoitia besnoiti*

### 4.2.1 Background

Besnoitiosis is a protozoal disease of cattle ('elephant skin disease') and other wild ruminants caused by *Besnoitia besnoiti*, a cyst-forming apicomplexan parasite in the family *Sarcocystidae*. There are several species of *Besnoitia* affecting domestic and wild animals. *B. besnoiti* is the only species known to infect both domestic and wild bovidae (Bigalke & Prozesky 2004; Leighton et al. 2001). *B. besnoiti* has several strains (differentiated only by the pathology and clinical signs they produce and the typical species they infect), including a cattle strain, a blue wildebeest strain and an impala strain. Only the cattle strain is known to cause significant disease. However, the importation of other strains of *B. besnoiti* would prompt a change to the country's animal health status.

*B. besnoiti* is endemic in cattle in Africa, Russia, Asia and South America (European Food Safety Authority 2010; Soulsby 1982). In Europe, the disease is endemic in southern countries (including Portugal, Spain, France and Italy) and is considered an emerging disease in the remainder of the continent, with the spread of cases into new areas over the past decade (European Food Safety Authority 2010; Mehlhorn et al. 2009). Domestic cattle trade, including movement of solitary infected animals, is cited as a major reason for European spread (Basso et al. 2013; Bigalke et al. 1967; Olias, Schade & Mehlhorn 2011). The disease is of economic importance in many countries due to the morbidity it causes (Bigalke & Prozesky 2004; Cortes et al 2014).

Besnoitiosis is not an OIE listed disease. It is not reported in Australia (Nasir et al. 2012). It is not nationally notifiable (Department of Agriculture and Water Resources 2016).

### 4.2.2 Technical information

#### Epidemiology

Many aspects of the epidemiology of bovine besnoitiosis remain uncertain. *Besnoitia* is a two-host parasite. Domestic cattle and other wild bovidae are considered the intermediate hosts for *B. besnoiti*, although this is still under discussion. Although the life cycle and definitive host for *B. besnoiti* are unknown, studies of other *Besnoitia* spp. and the genus's morphological similarity to *Toxoplasma gondii* strongly suggests that the definitive host for *B. besnoiti* is a carnivore (Basso et al. 2011; Bigalke & Prozesky 2004; Ellis et al. 2000; Kiehl et al. 2010; Olias, Schade & Mehlhorn 2011). In a typical transmission pathway the definitive host becomes infected by ingesting tissue cysts within the carcass of an intermediate host. The intermediate host becomes infected after ingesting sporulated oocysts excreted in the faeces of the definitive host. The incubation period after initial exposure is approximately 14 days (Bigalke & Prozesky 2004).

Initial infection of cattle with *Besnoitia* leads to an acute febrile stage that may last for up to 10 days. Rapidly multiplying tachyzoites are present in circulating blood during, and for a few days beyond, the febrile period. Cysts then develop in tissues containing slowly multiplying bradyzoites (Alvarez-Garcia *et al.* 2014a; Duvallet & Boireau 2015; Olias, Schade & Mehlhorn 2011). A large proportion of infected animals become seropositive but do not exhibit clinical signs (Liénard *et al.* 2013).

Transmission may occur between intermediate hosts, with biting flies acting as mechanical vectors, although the epidemiological significance of this method of transmission is unclear. This mode of transmission has been demonstrated experimentally for the cattle strain of *B. besnoiti* (Bigalke 1960; Liénard *et al.* 2013). Seasonal increases in transmission and the rapid infection of naïve animals when introduced to infected herds also support the role of mechanical transmission (Alvarez-Garcia *et al.* 2014b; Cortes *et al.* 2014). Flies may ingest the organism from cysts on skin or mucous membranes, or from blood under experimental conditions (Bigalke & Prozesky 2004; Frey *et al.* 2013a; Liénard *et al.* 2013). As parasitaemia only occurs during the short febrile stage the source of parasite for uptake would be bradyzoites released from tissue cysts e.g. from rupture (Alvarez-Garcia *et al.* 2014b; Gutiérrez-Expósito *et al.* 2015). Gutiérrez-Expósito *et al.* (2015) discusses a case of detectable parasitaemia in chronically infected cattle.

The importance of horizontal transmission (e.g. mating, close contact with wounds, ingestion of ruptured cysts associated with mammary gland suckling) has become apparent in recent years (Álvarez-García *et al.* 2013; Basso *et al.* 2013; Esteban-Gil *et al.* 2017). Chronically and subclinically infected animals may play an important role in the transmission of the disease by acting as reservoirs for intra-herd spread (Alvarez-Garcia *et al.* 2014a; Basso *et al.* 2013; Esteban-Gil *et al.* 2017; Frey *et al.* 2013a). Irrespective of the actual transmission method, parasite transmission appears to be maintained over time within infected herds (Gutiérrez-Expósito *et al.* 2015).

Non-domestic bovidae intermediate hosts for *B. besnoiti* are reported to include blue wildebeest (*Connochaetes taurinus*), impala (*Aepyceros melampus*) and kudu (*Tragelaphus strepsiceros*) (Bigalke *et al.* 1967; McCully *et al.* 1966). Besnoitiosis was reported in a single blue duiker in a zoo in the United States but the causative species was not reported (Foley, Anderson & Steinberg 1990). A serosurvey in Canada detected *Besnoitia* antibodies in wild muskox and cervidae but due to cross-reactivity it could not be determined whether they were *B. besnoiti* or *B. tarandi* (Gutierrez-Exposito *et al.* 2012). Other serosurveys have detected antibodies to *Besnotia* spp. in cervidae but not in non-domestic bovidae (Gutierrez-Exposito *et al.* 2016; Gutierrez-Exposito *et al.* 2013).

The strains of *B. besnoiti* identified in wild bovidae appear to be different from those in domestic cattle; these strains demonstrate low pathogenicity when tested in cattle and are used in vaccines for cattle (Leighton *et al.* 2001). Antelope strains are reported to be viscerotropic whilst the cattle strains are dermatotropic (Bigalke & Prozesky 2004; Le Blancq *et al.* 1986; McCully *et al.* 1966). Skin lesions have been experimentally produced in blue wildebeest using both the cattle and antelope strains of *B. besnoiti*, however no cases of natural disease in non-domestic bovidae have been reported (Bigalke *et al.* 1967; Olias, Schade & Mehlhorn 2011). Domestic cattle experimentally infected by the antelope strains did not produce cysts or evidence of disease other than a mild febrile reaction (Bigalke *et al.* 1967). A wide range of mammals

including rabbits, sheep, goats and black wildebeest have been infected experimentally with the cattle strain of *B. besnoiti*, however, no naturally infected hosts other than cattle have been found (Gutierrez-Exposito et al. 2013). The cattle strain of *B. besnoiti* has only been reported to occur, under natural conditions, in domestic cattle and non-domestic bovidae are not known to be involved in transmission of the cattle strain.

Orchitis and permanent sperm changes are reported in infected bulls and goats (Bigalke & Prozesky 2004). A risk of transmission may be present from rupture of cysts and intermittent or active shedding, however, the transmission of *B. besnoitia* in semen has not been reported even in clinically affected animals (Esteban-Gil et al. 2014; Gollnick et al. 2015; Hornok et al. 2015; Kumi-Diaka et al. 1981). Comparison to toxoplasmosis life cycles and current knowledge of *Besnoitia* epidemiology indicates semen is an unlikely pathway for transmission in otherwise healthy animals.

### **Clinical signs**

Infected intermediate hosts may demonstrate a range of signs from acute febrile disease with oedema to chronic disease associated with formation of tissue cysts (Gollnick et al. 2015; Leighton et al. 2001). Disease in domestic cattle may be severe (but usually not fatal) or mild. Clinical signs in cattle tend to occur in two phases, either acute or chronic. The severe form of the disease is characterised in the acute phase by fever, inappetance, hyperaemia and orchitis. The chronic phase is characterised by scleroderma, hyperkeratosis, alopecia, loss of necrotic epidermis and atrophy of the testes of bulls. A significant proportion of infected domestic cattle do not develop clinical signs (Alvarez-Garcia et al. 2014b; Duvallet & Boireau 2015; Olias, Schade & Mehlhorn 2011).

Natural infections in non-domestic bovidae species are not known to be associated with clinical disease. Clinical signs produced by experimental inoculation are similar to those seen in domestic cattle.

### **Diagnosis**

Tissue histology has been the traditional method for confirmatory diagnosis. Tissue cysts may only be pin-point in size. Recent improvements in ELISA and IFAT serological methods have improved the ability to detect asymptomatic *B. besnoiti* infections (Cortes et al. 2006; García-Lunar et al. 2013; Lienard et al. 2015; Liénard et al. 2011; Schares et al. 2013). Chronically infected animals may not develop antibodies (García-Lunar et al. 2013; Gutiérrez-Expósito et al. 2015; Schares et al. 2016). Some tests have low or poor sensitivity and cross-reactions may occur with other species of *Besnotia* affecting ungulates (*B. besnoiti*, *B. tarandi*, and *B. bennetti*) (Cortes et al. 2014; Gutierrez-Exposito et al. 2012; Gutiérrez-Expósito et al. 2015; Ness et al. 2012; Olias, Schade & Mehlhorn 2011). Cross-reactions due to the presence of antibodies to *Neospora caninum* and *Sarcocystis* spp. are also reported (Garcia-Lunar et al. 2015). The Western Blot test is highly sensitive and specific and is used both as an assay and as a confirmatory test (Basso et al. 2013; Gutierrez-Exposito et al. 2013; Nasir et al. 2012). The modified agglutination test (MAT) is comparable to the IFAT in reliability (Waap et al. 2011). A combination of serological tests is recommended, especially for individual animals, to improve sensitivity and specificity (Cortes et al. 2014; García-Lunar et al. 2013; Gutiérrez-Expósito et al. 2015).

Molecular testing methods are used but may have similar limitations as described above, especially in detection of sub clinical cases (Cortes et al. 2014; Schares et al. 2011).

## Prevention

A vaccine derived from blue wildebeest *B. besnoiti* strain is used in cattle, but is not commercially available in many countries. It is primarily aimed at preventing clinical signs and does not prevent development of asymptomatic carriers. Treatment of besnoitiosis is generally unrewarding (Bigalke & Prozesky 2004).

### 4.2.3 Current biosecurity measures

There are no previous biosecurity measures for live animals and *B. besnoiti*. There are no recommendations in the OIE Code.

### 4.2.4 Risk review

*B. besnoiti* is present in approved countries. It is not present in Australia and is not a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of *B. besnoiti* in non-domestic zoo bovidae:

- Several key factors in the epidemiology of *B. besnoiti* are still not understood.
- The disease is of economic importance in many countries due to the morbidity it causes.
- The lifecycle of *B. besnoiti* involves both a definitive and intermediate vertebrate host, however the definitive host has not been identified, but is presumed to be a carnivore. A variety of ruminant species, as well as other mammalian species, have been identified as intermediate hosts.
- Horizontal spread within herds of intermediate hosts, including the presence of long-term carriers, is highly likely as one mechanism through which population infection is maintained over time.
- There are several strains of *B. besnoiti*, including a cattle strain and at least two different antelope strains. Strains are differentiated by the pathology and clinical signs they produce, and the typical species they infect.
- The antelope strains of *B. besnoiti* are of low pathogenicity but produce obvious internal pathology. They have been reported in wild African antelope but not in zoo antelope in approved countries.
- The antelope strains have not been reported in domestic cattle. An attenuated form of the antelope strain is used to vaccinate livestock in endemic regions against the cattle strain.
- The cattle strain of *B. besnoiti* is pathogenic but under natural conditions is only reported in domestic cattle. Transmission may occur when the intermediate host ingests sporulated oocysts excreted in the faeces of the definitive host. The definitive host is infected by ingesting tissue cysts within the carcass of the immediate host. Horizontal transmission between intermediate hosts may also occur through direct contact or mechanical transmission by biting arthropods from skin and mucous membrane lesions.
- Wild antelope probably have a role in transmission of their respective strains of *B. besnoiti*. Non-domestic bovidae are not known to be involved in transmission of the cattle strain of *B. besnoiti*.
- A transmission risk via semen has not been demonstrated.

- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs including post mortem investigation of deceased animals and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.2.5 Conclusion

Accordingly and based on the preceding information, specific risk management measures for *B. besnoiti* are warranted for live animals. Specific measures are not warranted for zoo bovid semen.

Australia's biosecurity measures for *B. besnoiti* for zoo bovids are:

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of *B. besnoiti* has occurred in any species.

### 4.3 Bluetongue

#### 4.3.1 Background

Bluetongue disease is an insect-borne viral disease of all ruminant species (including antelope, buffalo, cattle, deer, goats and sheep) in the order *Artiodactyla*, caused by bluetongue virus (BTV), a member of the *Orbivirus* genus of the family *Reoviridae* (Verwoerd & Erasmus 2004). Bluetongue disease is endemic in most countries between 53° N and 34° S with occasional outbreaks occurring outside these latitudes. It is endemic to many countries, including North America. It is considered an emerging disease in northern Europe, appearing in 2006 and spreading over subsequent years to involve Britain, and many northern European and Scandinavian countries (Carpenter, Wilson & Mellor 2009).

Several serotypes of BTV have been identified in certain regions of Australia, from vectors and sentinel cattle, but clinical disease from BTV has not been reported (Biosecurity Australia 2011). Exotic serotypes could be introduced by live importation of viraemic ruminants, inoculation of infected imported biological products into ruminants, use of live attenuated vaccines or wind dispersal of infected vectors. The introduction of exotic serotypes to Australia is undesirable as negative impacts would be expected, for example, BTV serotype 8 can cause clinical disease in domestic cattle (Falconi, López-Olvera & Gortázar 2011; Spickler 2015a; Vöggtlin et al. 2013). Certification of Australia's BTV status is important for several export markets.

Bluetongue disease is a multiple species OIE-listed disease (OIE 2018c). Clinical bluetongue disease is a nationally notifiable disease (Department of Agriculture 2015).

#### 4.3.2 Technical information

##### Epidemiology

There are 27 serotypes of BTV and strains differ in virulence and pathogenesis. Twelve serotypes (1, 2, 3, 5, 7, 9, 12, 15, 16, 20, 21 and 23) of BTV have been isolated in Australia from *Culicoides* spp. or clinically healthy cattle (Animal Health Australia 2015b; Jenckel et al. 2015). Clinical disease has not been observed in commercial flocks or herds of any susceptible species in Australia (Animal Health Australia 2015b). Serotype classification is related to the genes that determine specific surface antigens. While vaccination is protective for all identical serotypes, the genetic makeup of these viruses can be quite different in regions of the genome that influence virulence. Serotypes cannot be directly compared without genome sequencing.

Bluetongue disease is non-contagious and transmission of BTV occurs via the bites of *Culicoides* midges. Transmission requires a population of competent vectors, favourable climatic conditions for virus amplification in the vector and sufficient viral load to initiate infection in a new host. Infection in sheep and goats is usually preceded by widespread infection and amplification of the virus in cattle. The disease is most prevalent during the warmer months, especially in wet seasons, when the *Culicoides* vectors are most active. Late summer or early autumn are identified as the highest risk transmission periods due to build-up of virus numbers in cattle and increasing vector populations with warmer weather.

There are over 1400 species of *Culicoides* worldwide. Whilst less than 50 species are known vectors of BTV, the expansion of BTV8 in Europe has demonstrated an ability for BTV to be transmitted by *Culicoides* spp. not previously recognised as BTV vectors, when spreading into new areas (Sanderson 2011). The *Culicoides* vectors remain infective for life (10–90 days). Transovarial infection of the vector does not occur; subsequent generations of vectors can only be infected by feeding on a viraemic host during the infective period (Animal Health Australia 2015b).

*Culicoides* are biological vectors of BTV. They become infected by feeding on viraemic animals and remain infective for the duration of their lives, up to 90 days. Virus replication occurs in several stages, leading to virus in the vector's salivary glands and replication every 6–8 days (USDA 2016). Environmental temperature is a major influence on the rate of BTV transmission. Temperature not only influences vector activity but also the time required for the vector to digest a blood meal and incubate the virus, as well as the rate of viral replication itself, which ceases below 12° C (Carpenter et al. 2011; Wilson & Mellor 2009; Wittmann, Mellor & Baylis 2002). BTV is able to overwinter in some regions, with transmission resuming when climatic conditions once again become favourable (Animal Health Australia 2015b; Wilson & Mellor 2009). Possible explanations for this phenomenon include the activity of small populations of *Culicoides* in warmer microclimates such as animal barns; long-lived infected females in mild-winter regions; or non-vector related pathways of transmission. Persistent, active transmission in such scenarios has not been demonstrated (Animal Health Australia 2015b).

*Culicoides* activity and virus transmission have seasonal peaks during summer and autumn. Incursions of BTV may occur periodically outside of these peak times as influenced by vector distribution and sensitivity to frost and cooler environments. The OIE provides guidelines for zones or countries to be recognised as 'seasonally free of BTV'. Factors include BTV being a notifiable disease and that surveillance is conducted to demonstrate no BTV transmission nor adult *Culicoides* vector activity (Animal Health Australia 2015b; Nielsen 2017).

The BTV is introduced to the mammalian host by the bite of an infected vector and then replicates in a regional lymph node. BTV is highly cell-associated, particularly in blood cells and endothelial cells, and disseminates to a variety of tissues (MacLachlan & Gard 2008). BTV is found transiently in serum and monocytes but is present within red blood cells, even in the presence of high antibody titres, for up to eight weeks (Schwartz-Cornil et al. 2008). Infection with one serotype does not confer immunity to other serotypes, however, serotype-specific antibodies are believed to persist for the life of the animal following infection. Consecutive infections with a second and especially a third serotype normally give rise to a comparatively short-lived, broad-reacting neutralising antibody response. Animals eventually clear the virus, and there is no evidence that they remain persistently infected, even when infected *in utero*.

(Animal Health Australia 2015b; Biosecurity Australia 2011). Reports of a carrier state are generally confined to older literature prior to recognition of multiple re-infection of animals with different serotypes (Geering, Forman & Nunn 1995).

Direct transmission between infected ruminants is reported for some serotypes (for example, 1, 8, 26), but is not considered to be epidemiologically important (Spickler 2015a). Other minor routes of transmission include vertical transmission via the placenta, semen or colostrum, and by live attenuated vaccines or vaccines contaminated by BTV (Sperlova & Zendulkova 2011). BTV does not survive for long outside the host or vector and products (for example, meat, wool) and fomites are not a risk for transfer of infection.

The incubation period generally ranges from four to seven days. Viraemia is detectable two to three days post-infection and usually lasts less than four weeks, but may rarely persist for up to eight weeks (Bonneau et al. 2002; Gard & Melville 1992; Koumbati et al. 1999; Richards et al. 1988; Singer, MacLachlan & Carpenter 2001). The OIE considers the infective period for BTV in domestic species to be 60 days (OIE 2018c). Breed and species variations exist but in general terms the range of viraemia and incubation times across bovidae and cervidae species appears to be consistent (Falconi, López-Olvera & Gortázar 2011; Hoff & Hoff 1976; Niedbalski 2015; Tessaro & Clavijo 2001).

Although all ruminants are considered susceptible to BTV infection, clinical disease is primarily seen in sheep, and in wild deer and pronghorn in North America. BTV replicates in many species of ruminants, often asymptotically. Clinical disease is occasionally seen in cattle, goats, South American camelids, non-domestic ruminants, farmed cervids and some carnivores (Spickler 2015a). The role of carnivores and other wildlife species in BTV transmission is unknown or of minimal significance (Animal Health Australia 2015b; Biosecurity Australia 2011).

BTV has probably been endemic in wild African ruminants since antiquity and it is likely that some African wildlife species act as reservoir hosts for the virus (Verwoerd & Erasmus 2004). Serological surveys of African wildlife have demonstrated antibodies to BTV in a wide variety of ungulate species including blue wildebeest, black wildebeest, Cape buffalo, red hartebeest, eland, springbok, blesbok, impala, kudu, waterbuck (*Kobus ellipsiprymnus*), sable, reedbuck, tsessebe, Coke's hartebeest, Grant's gazelle, Thomson's gazelle, oryx, oribi, giraffe and African elephant (Anderson & Rowe 1998b; Barnard 1997; Davies & Walker 1974). Other than one case after experimental infection in a Cape buffalo, there are no reports of clinical disease as a result of BTV infection in African wildlife species (Howerth, Stallknecht & Kirkland 2001; Sanderson 2011; Young 1969).

North American ruminant species including white-tailed deer, pronghorn and desert bighorn sheep may develop clinical disease and suffer mortalities (Howerth, Stallknecht & Kirkland 2001; Jessup 1985; Thorne et al. 1988; Verwoerd & Erasmus 2004). Evidence of BTV infection has also been reported in American bison and mountain goats (Dulac et al. 1988; Howerth, Stallknecht & Kirkland 2001; Robinson et al. 1967).

In Europe, serological evidence of BTV infection has been detected in a variety of wild ruminants including Spanish ibex (*Capra pyrenaica*), mouflon (*Ovis aries musimon*), chamois (*Rupicapra pyrenaica*) and aoudad (*Ammotragus lervia*) (Lorca-Oró et al. 2014). In most European wild ruminant species, infection with BTV does not appear to result in clinical disease. BTV infection in mouflon in Spain resulted in clinical disease and mortalities (Fernández-Pacheco et al. 2008).

Wild European ruminants may be implicated in the epidemiology of BTV in Europe, with red deer the most likely (García et al. 2009; Lorca-Oró et al. 2014; Nielsen 2017; Rossi et al. 2014).

Clinical bluetongue is very rare in zoo collections but has occurred (Miller & Fowler 2014; Spickler 2015a). Seropositivity and viremia may be common in certain herds (S. Citino, White Oak Conservation, 2017, pers. comm.).

Some BTV serotypes (for example, 8, 23) can be shed in semen (for varying durations, depending in part on serotype) and can cross the placenta (Sperlova & Zendulkova 2011; Vanbinst et al. 2010). Semen that is collected during a period of low vector activity, when BTV transmission is reduced, is less likely to be infected with virus. A risk analysis of importation of BTV8 infected ruminant semen into Australia from the European Union determined that risk management was required (Biosecurity Australia 2011).

### **Clinical signs**

In most species, including cattle and many wild ruminant species, infection is generally subclinical or mild, with little more than a febrile reaction. Sheep may experience a wide range of clinical signs and severe, often fatal disease; signs may be peracute, acute or chronic and include hyperaemia of buccal and nasal mucosae, increased salivation, lacrimation and nasal discharge. Oedema of the tongue, lips, face and ears may be followed by cyanosis ('bluetongue'), and sub-mandibular oedema. Foot lesions and lameness may also be seen along with anorexia and loss of body condition (Geering, Forman & Nunn 1995).

Clinical bluetongue disease has been reported in a variety of zoo bovidae with signs similar to those reported in domestic sheep (Hoff, Griner & Trainer 1973; Howerth, Stallknecht & Kirkland 2001; Ramsay et al. 1985; Spickler 2015a).

### **Diagnosis**

Diagnosis of infection is by virus isolation in cell culture, animal inoculation, molecular methods or a number of serological tests including competitive ELISA (cELISA), complement fixation test (CFT), agar gel immunodiffusion and virus neutralisation.

Both the cELISA and several RT-PCR tests are highly sensitive and specific tests for BTV and are effective in determining an individual animal's BTV status (Batten et al. 2008; Batten et al. 2009). The cELISA may be used to survey population status. Antibodies may take one or two weeks post infection to be detected using the cELISA, but remain detectable for at least two years post infection (Biosecurity Australia 2011). RT-PCR enables detection closer to the point of infection. Serotyping of BTV is generally limited to laboratory studies with several weeks turnaround time, however, some RT-PCR formats are available that enable faster serotype identification (OIE 2014b).

### **Prevention**

BTV is difficult to control and efforts are generally focused on preventing the introduction of infective animals and vectors into new areas. Risk management may include movement of animals only from 'seasonally free' areas, although other measures such as vaccination and diagnostic testing may still be recommended (OIE 2016d). Direct management of *Culicoides* vectors, for example use of insect traps, may form part of a control strategy although the effectiveness of such methods may be difficult to assess.

Vaccination is a key preventative measure for BTV. Over 2000 individuals of 57 species of zoo species (primarily bovidae) were vaccinated in 47 zoos in nine European countries during 2008. Thirty-seven species of bovidae were tested post-vaccination, with 100 per cent seroconversion, and no vaccinated zoo animals showed signs of disease post-vaccination, despite ongoing circulation of the virus (Sanderson 2011). Vaccines should be matched to the viral serotype; protection against other serotypes can be limited or non-existent. Both attenuated and killed vaccines are currently manufactured (although not necessarily sold in all regions), and multivalent vaccines are available to limit livestock losses and reduce circulation of BTV. Attenuated vaccines are considered to be more effective than killed vaccines; but may have adverse consequences, including teratogenicity during early pregnancy, and ongoing spread by vectors with possible re-assortment of vaccine virus genes with those of wild type virus. Inactivated vaccines require two doses to reach a protective antibody titre (Animal Health Australia 2015b; OIE 2014b; Spickler 2015a).

#### 4.3.3 Current biosecurity measures

There are previous biosecurity measures for bluetongue disease in semen, embryos and live animals. Australian policies and OIE Code recommendations include country or zone freedom, testing or vaccination for multiple species (Biosecurity Australia 2011; OIE 2018c).

Australia's *National Arbovirus Monitoring Program* monitors the distribution of economically important arboviruses of ruminant and associated insect vectors, including BTV (Animal Health Australia 2015c).

#### 4.3.4 Risk review

Exotic strains of BTV are present in approved countries that are not present in Australia. Bluetongue (clinical disease) is a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of bluetongue in non-domestic zoo bovidae:

- BTV is primarily a vector borne virus. Transmission and distribution of BTV depends on the density of animal reservoirs, presence of amplifying hosts, and suitable *Culicoides* vector activity. Other modes of transmission are epidemiologically insignificant.
- Australia has many serotypes of BTV but some remain exotic.
- Australia's BTV status is important for export certification.
- BTV may be transmitted through semen in infected ruminants.
- BTV activity increases seasonally, generally during summer and autumn, when climatic factors promote vector activity, spread and efficiency of transmission.
- Domestic cattle play an important role in local disease transmission. Wild ruminants may have a role in maintenance of BTV within a geographic space.
- Clinical disease and production of antibodies have been reported in non-domestic bovidae (including those in zoos) however, a distinct epidemiological role for non-domestic bovidae has not been identified.
- Infection results in viraemia of up to 60 days, followed by natural, long term immunity to that serotype. There is no recognised carrier state.

- There is no cross-protection between serotype infections or vaccinations.
- Control is difficult and prevention relies primarily on sourcing virus-free animals. Country freedom, or seasonal freedom, and use of molecular and serological tests may aid this process.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- The quarantine facilities used in zoo pre-export quarantine and post-arrival quarantine are not vector proof.

#### 4.3.5 Conclusion

Accordingly and based on the preceding information risk management measures for zoo bovidae are warranted.

Australia's proposed risk management measures for BTV for live zoo bovidae are:

##### Option ONE

- The animal was not vaccinated against BTV in the 60 days prior to export.

AND

- In the 7 days immediately before export, a blood sample was taken from the animal and tested by an approved PCR method for bluetongue virus. The test result was negative.

AND

- Immediately on arrival into Australia, during transit to post-arrival quarantine site, and during post-arrival quarantine, the animal was protected from vector attacks. Details of how this will be accomplished should be detailed in the travel plan for approval.

AND

- At least 7 days post-arrival into Australia, a blood sample was taken from the animal in PEQ and tested by an approved PCR method for bluetongue virus.
  - If the test result is negative, vector protection may cease.
  - If the test result is positive, vector protection is maintained and the Department of Agriculture and Water Resources is contacted as soon as possible (and no later than 48 hours) following the result for further direction.

##### Option TWO

- The animal was kept in a country free or seasonally free from BTV as recognised by Australia\* at least 60 days prior to export (and within the period the country is considered free from BTV).

\*Countries recognised as free from BTV or having seasonally free periods from BTV:

- Canada (not including the Okanagan Valley of British Columbia)—country seasonally free between 1 January and 15 May.
- New Zealand – country free from BTV.

AND

- The animal was not vaccinated against BTV in the 60 days prior to export.

Australia's proposed risk management measures for BTV for zoo bovidae semen are:

### Option ONE

Blood samples were drawn from the donor:

- Between 28 and 60 days immediately after the semen collection period finished. The blood samples gave negative results to the competitive ELISA for BTV antibodies.

OR

- On the first day, the last day and at least every 7 days during the semen collection period. The blood samples gave negative results to a virus isolation test for BTV.

OR

- On the first day and the last day of the semen collection period. The blood samples gave negative results to an approved RT-PCR test for BTV.

[The veterinary health certificate must indicate the option that applies].

### Option TWO

- All donors were kept in a country free or seasonally free from BTV as recognised by Australia\* at least 60 days prior to, and at the time of, semen collection

\*Countries recognised as free from BTV or having seasonally free periods from BTV:

- Canada (not including the Okanagan Valley of British Columbia)—country seasonally free between 1 January and 15 May.
- New Zealand – country free from BTV.

AND

- The animal:
    - was not vaccinated
- OR
- was vaccinated, and the vaccine was: inactivated, approved by the competent authority in the exporting country, and administered more than 60 days prior to semen collection.

[The veterinary health certificate must indicate the option that applies].

## 4.4 Bovine tuberculosis

### 4.4.1 Background

Tuberculosis is caused by *Mycobacterium* spp. in the family Mycobacteriaceae. They are non-motile, non-spore forming, weakly Gram-positive, acid fast, facultative intracellular bacteria. Those species pathogenic for humans and animals belong to the slow-growing group and are referred to as the *Mycobacterium tuberculosis* complex (Gibson 1998; Good & Shinnick 1998). Members of the *M. tuberculosis* complex include: *M. africanum*, *M. bovis*, *M. canettii*, *M. caprae*,

*M. microti*, *M. orygis*, *M. pinnipedii* and *M. tuberculosis*. Of these agents, *M. bovis* is the primary agent of biosecurity concern considered in this review.

Bovine tuberculosis (BTB), caused by *M. bovis*, is an infectious, chronic respiratory disease in cattle, deer, goats, pigs, water buffalo (*Bubalus arnee*) and a wide range of other animal species. *M. bovis* has been identified in non-domestic ungulates and other mammalian species (Montali, Mikota & Cheng 2001). Many cloven hoofed species have established natural infections with *M. bovis*. In the wild, the badger in the United Kingdom and Australian brush tail possum in New Zealand have also established reservoir infections (Radostits et al. 2007b).

Bovine tuberculosis is widespread throughout the world. Eradication campaigns in many countries have effectively reduced the incidence of bovine tuberculosis (OIE 2018c). Australia eradicated BTB after a 27 year campaign at approximately \$840 million, declaring freedom in accordance with the OIE Code in 1997 (More, Radunz & Glanville 2015). In order to maintain that status, it is essential that a definitive diagnosis be made for any animal in Australia with signs that could be due to this organism.

BTB is a disease of significant economic importance to Australia. In the context of zoo bovidae, there may be difficulty distinguishing between tuberculosis species, including *M. bovis* and *M. tuberculosis*. The implication of this is that Australia must assess animals that exhibit clinical signs or test results consistent with undefined tuberculosis as potentially infected with *M. bovis* and therefore apply protections on that basis.

Tuberculosis (from all causative agents) is a zoonosis and a nationally notifiable disease of public health concern (Department of Health 2015).

BTB is an OIE-listed disease (OIE 2018c). BTB is not present in Australia and it is a nationally notifiable animal disease (Department of Agriculture 2016).

#### 4.4.2 Technical information

##### Epidemiology

Domestic cattle are considered the natural host species for *M. bovis* (Cousins et al. 2004). It may also infect a wide range of mammal species including humans, other domestic species and free-living and captive wildlife. Wildlife reservoirs of *M. bovis* include white-tailed deer (*Odocoileus virginianus*) and bison (*Bison bison*) in North America, brushtail possums (*Trichosurus vulpecular*) in New Zealand, badgers (*Meles meles*) in the United Kingdom and Ireland, and African buffalo (*Syncerus caffer*) and lechwe (*Kobus lechwe*) in Africa (Clifton-Hadley et al. 2008; Kaneene et al. 2010; Miller 2008).

Other non-domestic bovidae species may have the potential to act as reservoirs of *M. bovis*, but their role in the epidemiology of the disease has not been confirmed. *M. bovis* infection has been confirmed in a wide range of bovidae species, including greater kudu (*Tragelaphus strepsiceros*), feral water buffalo (*Bubalis bubalis*), Arabian oryx, European wild goat (*Capra aegagrus*), impala (*Aepyceros melampus*), sitatunga (*Tragelaphus spekii*), wildebeest (*Connochaetes* spp.), lesser kudu (*Tragelaphus imberbis*), topi (*Damaliscus korrigum*), yak (*Bos grunniens*), common duiker, east African oryx (*Oryx gazelle beisa*), bushbuck, mountain goat, addax, sable and eland (*Taurotragus* spp.) (Kaneene et al. 2010; Miller 2008; Smith et al. 2017). All cloven-hoofed ungulates are considered susceptible (Miller 2008). Single or multiple cases of *M. bovis* have

frequently been reported in zoo bovidae (Lecu & Ball 2015; Miller 2008). For the purposes of this review, all bovids are regarded as susceptible to infection with *M. bovis*.

Transmission of *M. bovis* in bovidae occurs most commonly through inhalation or ingestion. The bacteria is aerosolised from the respiratory tract of infected hosts and is also shed in excretions and secretions including milk.

Transmission via the congenital, cutaneous, or venereal route has also been reported. The bacteria may be excreted in respiratory discharges, saliva, faeces, milk, urine, vaginal and uterine discharges as well as discharges from draining peripheral lymph nodes. Animals with gross tuberculous lesions communicating with airways or the intestinal tract are likely to excrete large numbers of bacteria into the environment (Cousins et al. 2004; Radostits et al. 2007b). Infected individuals may shed bacteria in the absence of clinical signs. Indirect inhalation and oral transmission via contaminated feed, infected sputum, or infected dust particles have been suspected to be the mode of transmission in several cases (Cousins et al. 2004; Lepper & Pearson 1973; Neill et al. 1994; Palmer, Waters & Whipple 2004; Phillips et al. 2003). A single colony forming unit is sufficient to cause disease via the respiratory route (Dean et al. 2005) and disease severity reflects the size of infectious dose received and the immune status of the host (Menzies & Neill 2000; Risco et al. 2014). Infection with *M. bovis* generally results in the development of chronic granulomatous lesions.

The incubation period of *M. bovis* may be prolonged and difficult to detect. Not all infected animals go on to develop clinical disease, which is influenced by the individual's ability to mount a successful cell-mediated immune response to infection.

Semen from infected donors can pose a transmission risk, either through intrinsic infection (bacteria is present in seminal fluid), or extrinsic infection (BTB lesions in the prepuce contaminate the seminal sample) (Ahmed, Khan & Ganai 1999). Although uncommon, miliary tuberculosis and chronic testicular tuberculosis have been reported in testes of bulls (Hein & Tomasovic 1981).

### **Clinical signs**

BTB has a chronic, variable, and often subclinical course (Cousins et al. 2004). In zoo animals infection is often very advanced before clinical signs are detectable (Lecu & Ball 2015; Lyashchenko et al. 2006; Montali, Mikota & Cheng 2001). If clinical signs develop they are generally non-specific and include ill-thrift, weakness, chronic wasting and eventually death.

### **Diagnosis**

Diagnostics for BTB fall into three categories: direct, cell-mediated-immunity (CMI), and serological (humoral) techniques. Diagnosis is complex as many tests lack both sensitivity and specificity, and shedding of *M. bovis* organisms is intermittent. No single test is 100 per cent effective at diagnosing infection with BTB. Test specificity and sensitivity depend on the test used, the stage of disease, distribution of infection and other confounding factors. Most diagnostic tests for *M. bovis* have not been validated for zoo animals or non-domestic bovidae. Tests based on immunological response (both cell-mediated and humoral) may vary in reliability with host species and should be interpreted carefully. Many tests may show cross-reaction between *M. bovis*, *M. tuberculosis*, and other species of Mycobacteria. Due to the protection Australia requires from BTB, Australia must assess animals that exhibit clinical signs

or test results consistent with having undefined tuberculosis as potentially infected with *M. bovis* (More, Radunz & Glanville 2015).

For zoo and non-domestic ungulates, several authors and organisations support the general recommendation to perform a single cervical tuberculin skin test (TST) in combination with at least one alternate testing method (Miller & Fowler 2014; Modise 2012; Thoen 2013). Repeat testing and the use of tests that target different parts of the immune response is a recommended strategy to overcome the limitations of BTB diagnostic tests (Lécu & Ball 2011; Lerche et al. 2008; Modise 2012). Knowledge of herd health aids in interpretation of diagnostic tests for tuberculosis in bovidae (both domestic and non-domestic). Many zoos in approved countries have ongoing BTB screening programs utilising a range of recommended or 'in-house' protocols.

Generally, the CMI modalities are more reliable earlier in the course of the disease and become less sensitive once advanced pathology—and greater infectivity—occur, due to anergy of the CMI response. By the same rationale, humoral techniques become more reliable as pathology develops (De la Rua-Domenech et al. 2006; Modise 2012).

Culture and sensitivity is the gold standard for confirmatory diagnosis of *M. bovis*. However, culture can take 2–12 weeks for a result, there is often difficulty in obtaining suitable specimens for culture and the test has low sensitivity (Lécu & Ball 2011). Microscopy and molecular techniques can be used for confirmatory diagnosis to detect mycobacterial DNA in biological samples, but both techniques, while highly specific, have poor sensitivity (Lecu et al. 2013).

The TST uses tuberculin protein to elicit a delayed-type hypersensitivity reaction (Cousins et al. 2004). The TST in ungulates involves intradermal injection of 0.1 mL bovine purified protein derivative (PPD) tuberculin (1 mg/mL) in the cervical skin fold or caudal skin fold with reactions measured 72 hours after injection (Miller & Fowler 2014; Thoen 2013).

The comparative tuberculin skin test (CTST) compares the response to avian tuberculin with the response to mammalian tuberculin and helps rule out false positives from non-specific reactions due to exposure to atypical mycobacteria. The CTST is performed similar to the TST but avian tuberculin at the same concentration is also injected at a site approximately 12–15 cm away (OIE 2009). The reaction is measured 72 hours after injection.

Interpretation of the TST and CTST in non-domestic bovidae can be undertaken according to protocols detailed by the OIE Manual and standard textbooks for interpretation of tests in domestic cattle (Cousins et al. 2004; Miller & Fowler 2014; OIE 2009).

The TST and CTST rely on a local inflammatory cell response which may be low or absent due to concurrent immunosuppression, latent infections, a state of anergy associated with advanced or generalised disease, recently acquired infections, tegument cellular organisation for that species, or environment factors (De la Rua-Domenech et al. 2006; Gyles et al. 2008; Lécu & Ball 2011). False negatives may also occur due to incorrect injection technique, subjectivity in the interpretation of the TST, or the use of suboptimal reagent concentration. False positives may be due to previous exposure to injection adjuvant or non-specific reactions to contaminants or exposure to atypical or saprophytic mycobacteria (Bushnitz et al. 2009). Further limitations include variable sensitivity and specificity and the need to immobilise certain animals in order to both administer and read the test (Cousins & Florisson 2005; Cousins et al. 2004; Lécu & Ball 2011). Because reading methods vary between veterinarians, it is a good practice to have the

same veterinarian perform the initial and subsequent readings, perform a close examination of the test site, palpation, and measure any swelling by callipers. The concentration of bovine and avian tuberculin used should be no lower than 2,000 IU and the injection volume should be a maximum of 0.2 mL.

The TSTs have been developed as herd tests and are most useful when applied to herds rather than individuals (De la Rua-Domenech et al. 2006; Maas, Michel & Rutten 2013). After a TST is performed, it is recommended that an interval of time is allowed to elapse before the next test, to reduce the possibility of desensitisation by antigen overload. This is most applicable to follow-up skin tests but is also considered for other test modalities. The recommended time interval between tests varies by host species, context and diagnostic test, but is a minimum of 42 days for cattle (Bourne 2014; De la Rua-Domenech et al. 2006; Lécu & Ball 2011; Radunz & Lepper 1985).

Intradermal tuberculin injections have been used to elicit an anamnestic rise in general immunity and increase the sensitivity of diagnostic tests (Katial 2004; Lécu & Ball 2011). While this technique was shown to increase the sensitivity of serological tests in heavily infected animals, it also resulted in an increase in false-positive reactions in other animals (Chambers 2009).

A blood test (BOVIGAM®<sup>™</sup>), which measures CMI via the production of gamma interferon (IFN- $\gamma$ ) by sensitised lymphocytes, has been developed for cattle, with good sensitivity and specificity (Lipiec, Krajewska & Szulowski 2013). Studies also support the extension of this test to exotic species (Cousins & Florisson 2005; Goosen et al. 2015; Grobler, De Klerk & Bengis 2002; Katale et al. 2017; Lecu & Riquelme 2008; van der Heijden et al. 2016). Whole blood is incubated overnight with PPD and, if activated, lymphocytes produce IFN- $\gamma$  in response to the PPD stimulation (De la Rua-Domenech et al. 2006). There are host species-specific differences in IFN- $\gamma$  response that need to be taken into consideration, including the choice of positive control antigens (De la Rua-Domenech et al. 2006; Lécu & Ball 2011). The IFN- $\gamma$  also suffers by some of the limitations that affect all tests based on CMI, including an anergic state in some animals (De la Rua-Domenech et al. 2006). Bovine gamma IFN tests may be used as an alternative testing method following a positive TST, or may be used in parallel with TST to increase sensitivity and specificity (Lecu & Ball 2015; Miller 2008).

Serological tests that detect antibodies against *Mycobacterium*-specific antigens have shown diagnostic potential for a variety of both captive and free-ranging wildlife hosts (Duncan et al. 2009; Jurczynski et al. 2011; van der Heijden et al. 2016). Serological techniques that detect antibodies are generally more useful at later stages of infection as the humoral response is limited in early stages and minimal pathology (De la Rua-Domenech et al. 2006). Secreted antigen 85 (Ag85) is produced during active infection with *M. bovis*. It can be detected in serum using a dot blot immunoassay and has been shown to be diagnostic for *M. bovis* infections in some species (Chambers 2009). ELISA techniques have been used for the diagnosis of BTB in many non-domestic bovidae and have the advantage of being simple, rapid, and low cost (Chambers 2009; Cousins & Florisson 2005). They may be used in conjunction with CMI tests, to increase sensitivity and specificity, and to assist in interpretation of results in anergic individuals (Miller & Fowler 2014; OIE 2009). When used alone, they may have low sensitivity and specificity, and require species-specific antigens for test accuracy (Modise 2012). A rapid card serological test (Dual-Path Platform (DPP®) VetTB Assay) is marketed for use in cervids and elephants by Chembio. The DPP® VetTB Assay allows the rapid detection of antibodies to *M.*

*bovis* and *M. tuberculosis* and has been employed in non-domestic bovidae with apparently high sensitivity and specificity (Cousins & Florisson 2005; Modise 2012). The multi-antigen print immunoassay (MAPIA, Chembio) is a laboratory based test able to distinguish between presence of *M. bovis* or *M. tuberculosis* antigens in a sample but it is currently only available in the United States.

Diagnostic imaging may be used to assist diagnosis but cannot reliably distinguish between tuberculosis and pneumonia due to other causes (Keeling, Wolf & Bourne 1975; Lecu et al. 2013). Digital and film-screen radiography have a limiting spatial resolution between 0.17 and 0.08 mm, making them unsuitable for screening for microscopic disease or carrier animals that do not have extensive lesions or pathology patterns (Thrall 2013).

### **Treatment**

Mycobacterial diseases in any species are difficult to treat successfully. A variety of chemotherapy methods have been attempted, usually to preserve or prolong the life of valuable animals. Ante-mortem verification that treatment has eliminated all of the mycobacteria is difficult to prove definitively.

### **Prevention**

BTB in a domestic herd is typically addressed by isolation, quarantine, testing and culling strategies. In zoos, BTB risk is managed through isolation, compartmentalisation, pre- and post-transfer testing and health checks of individual animals; routine screening (e.g. annual) of herds; investigation of sick individuals and post-mortem examination of deceased animals.

## **4.4.3 Current biosecurity measures**

Australia's biosecurity measures for bovine tuberculosis in zoo bovids from New Zealand include premises freedom and testing but is also predicated on a country control program and specific assessment of their zoo systems at the time of review. Recommendations in the OIE Code for bovid species include country, zone or compartment freedom and testing (OIE 2018c).

## **4.4.4 Risk review**

*M. bovis* is present in exporting countries and it is not present in Australia, where it is a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of BTB in zoo bovidae:

- Bovine tuberculosis generally has a worldwide distribution, but has been eradicated from Australia.
- Bovine tuberculosis is a nationally notifiable animal disease.
- Tuberculosis (*M. tuberculosis* complex) is a nationally notifiable disease of public health concern.
- The reintroduction or reporting of bovine tuberculosis into Australia would place at risk the large industry and public investment in the national eradication of this disease. Australia must assess animals that exhibit clinical signs or test results consistent with undefined tuberculosis as potentially infected with *M. bovis*.
- Transmission of *M. bovis* primarily occurs via respiratory aerosols. It can also occur via contaminated respiratory secretions, pasture and other fomites.

- Tuberculosis caused by *M. bovis* has been reported in a wide range of zoo and wild bovidae.
- Infections in bovidae may not always progress to clinical disease, but are usually lifelong.
- Diagnostic testing is complex; with many limitations in zoo bovidae.
- The use of multiple tests of differing modality is recommended to increase diagnostic sensitivity and specificity in individual zoo animals.
- A knowledge of herd health aids in interpretation of diagnostic tests for tuberculosis in bovidae (both domestic and non-domestic). BTB screening programs exist in many zoos to aid this.
- Treatment of tuberculosis is not reliable at eliminating infection.
- *M. bovis* may be transmitted in semen and may remain viable in frozen semen.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.4.5 Conclusion

Accordingly and based on the preceding information, risk management measures for live zoo bovidae and semen are warranted.

Australia's proposed biosecurity measures for bovine tuberculosis for live zoo bovidae are:

##### Option ONE

- For 12 months immediately before export the animal has not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals for export were each subject to a test for bovine tuberculosis performed between 210 and 72 days immediately before export, with negative results. The test must be:
  - a TST or CTST. The test was read 72 hours post-inoculation, with negative results
 OR
  - 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.

AND

- The animals for export were each subject to a tuberculin skin test (TST) *or* comparative tuberculin skin test (CTST) performed in the 30 days immediately before export. The test was read 72 hours post-inoculation, with negative results.

AND

- The animals for export were each subject to *either* a gamma interferon assay approved by the department *or* a serological test approved by the department, with negative results. The

test was performed in the 30 days immediately before export on blood taken during this period.

### Option TWO

- For 12 months immediately before export the animal has not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals for export each received three separate skin tests for tuberculosis in the 210 days prior to export. The tests were performed a minimum of 42 days apart from each other and one was *during* pre-export quarantine. Each test was *either* a TST *or* CTST. Each test was read 72 hours post-inoculation, with negative results.

### Option THREE

- For 12 months immediately before export the animal has not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - Diagnostic testing of all zoo 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).
  - That the collection must have been a 'closed-herd'<sup>1</sup> during that time.
  - That the collection must contain at least four zoo bovids.
  - That full post mortems investigations were conducted on any dead ungulate species to determine the cause of death.

AND

- The animals for export were each tested for bovine tuberculosis between 210 and 72 days immediately before export. If the test for the collection screening program occurs during this time, it will fulfil this requirement. The test must be either:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative resultsOR
  - 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.

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<sup>1</sup> A 'closed-herd' in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

AND

- The animals for export were each tested with a TST *or* CTST performed during the 30 days immediately before export. The test was read 72 hours post-inoculation, with negative results.

#### Option FOUR

- For 12 months immediately before export the animal has not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - diagnostic testing of all zoo 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).
  - That the collection must have been a ‘closed-herd’<sup>2</sup> during that time.
  - That the collection must contain at least four zoo bovids.
  - That full post mortem investigations were conducted on any dead ungulate species to determine the cause of death.

AND

- The animals for export were each tested via a TST *or* CTST, performed between 210 and 72 days immediately before export. The test was read 72 hours post-inoculation, with negative results. If the test for the herd screening program occurs during this time, it will fulfil this requirement.

AND

- The animals for export were each tested for bovine tuberculosis, performed during the 30 days immediately before export. The test must be either:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative results.
 OR
  - 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.

Australia’s proposed biosecurity measures for bovine tuberculosis for zoo bovidae semen are:

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<sup>2</sup> A ‘closed-herd’ in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

**Option ONE**

- 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 bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals from which the semen for export was collected were each subject to a test for bovine tuberculosis performed between the 210 and 72 days immediately before semen collection. The test must be:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative resultsOR
  - 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.

AND

- The animals from which the semen for export was collected were each subject to a tuberculin skin test (TST) *or* comparative tuberculin skin test (CTST) performed in the 30 days immediately before semen collection. The test was read 72 hours post-inoculation, with negative results.

**Option TWO**

- 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 bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - Diagnostic testing of the zoo 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).
  - The collection must have been a 'closed-herd'<sup>3</sup> during that time.
  - The collection must contain at least four zoo bovids.
  - Full post mortem investigations were conducted on any dead ungulate species to determine the cause of death.

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<sup>3</sup> A 'closed-herd' in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

AND

- The animals from which the semen for export was collected were subject to a test for bovine tuberculosis, performed in the 30 days immediately before semen collection. This test must be separate to the herd screen program test. The test must be either:
  - a TST or CTST. The test was read 72 hours post-inoculation, with negative results. If the herd test was also a TST or CTST, then this second test must not be performed within 42 days of the herd test
- OR
  - on a blood sample taken in the 30 days immediately before semen collection and tested using *either* a gamma interferon assay approved by the department *or* a serological test approved by the department, with negative results.

## 4.5 Bovine viral diarrhoea (Type 2)

### 4.5.1 Background

Bovine viral diarrhoea (BVD), is caused by bovine viral diarrhoea virus (BVDV), belonging to the genus *Pestivirus*, family *Flaviviridae* (Potgieter 2004). The virus causes a range of clinical syndromes in domestic cattle, most commonly bovine viral diarrhoea (BVD) and mucosal disease (MD). It is primarily a pathogen of cattle however BVD may infect a wide range of domestic animals and free-living ruminants, including sheep, pigs, alpacas, deer and non-domestic bovidae. Infection due to BVDV is worldwide in distribution although some European countries have eradicated it.

The literature and most control programs at a country or herd level do not typically distinguish between BVDV genotypes because both may be present within a country and the epidemiology and management are identical. This review predominantly differentiates BVD1 and BVD2 at the level of risk management, given Australia's freedom from the clinically more costly BVD2.

BVD is an OIE listed disease (OIE 2018c).

BVD1 is widespread in Australia however the more virulent BVD2 has not been reported in Australia (Animal Health Australia 2016). BVD2 is a notifiable disease of animals in Australia (Department of Agriculture 2016).

### 4.5.2 Technical information

#### Epidemiology

Bovine viral diarrhoea leads to major losses in dairy and beef herds. Two genotypes, BVD1 and BVD2, have been identified. BVD1 is widespread globally, including in Australia. BVD2 is more pathogenic, causing severe haemorrhaging and loss of condition, with high mortality rates, and is increasingly recognised in North America and Europe (Collins et al. 2009). A third putative genotype, atypical BVDV (a 'HoBi-like' pestivirus), has recently been proposed (Bauermann, Flores & Ridpath 2012). Infection due to atypical BVDV has been reported in Asia, Europe and South America (Bauermann et al. 2013; Weber et al. 2014).

The epidemiology and pathogenesis of BVD is complex but is well studied in domestic cattle. Pathogenesis depends on a number of host factors and the specific properties of the infecting BVDV isolate (Radostits et al. 2007b). BVDV is lymphotropic, leading to immunosuppression and increased susceptibility to concurrent disease (Walz et al. 2010). BVD is characterised by

high morbidity (80 to 100 per cent) and low case fatality (5 to 10 per cent) (Harkness & Van der Lugt 1994).

BVDV isolates may be non-cytopathic (NCP) or cytopathic (CP) biotypes. NCP biotypes appear to be the cause of acute infections, although CP isolates have induced acute infection under experimental conditions (Potgieter 2004). The most important source for introducing BVDV infection into a herd is Persistently Infected (PI) animals (Potgieter 2004).

PI animals arise from foetal infection with NCP isolates, early in gestation (Lanyon et al. 2014). Most PI calves are smaller and die at a young age. Some show no clinical signs and may survive, to be the major source of infection in a herd. They are immunotolerant to the virus and can be difficult to detect using serological methods, some mounting no detectable antibody response whilst others demonstrate antibodies without any resolution of viral shedding. A herd with one or more PI animals is expected to have a high proportion of antibody positive animals.

An epidemiologically significant scenario arises when a non-PI cow carries a PI foetus, known colloquially as a 'Trojan-cow'. The dam appears healthy but the PI calf, once born, begins shedding significant amounts of virus. Trojan cows are noted to have antibody titres, in mid-late pregnancy, significantly higher than that of seropositive cows carrying non-infected calves, probably as a result of continual antigenic challenge from the foetus (Brownlie et al. 1998; Lanyon et al. 2014; Lindberg et al. 2001).

Mucosal disease (MD) develops when superinfection with the CP biotype occurs in PI animals. MD is characterised by low morbidity (5 to 10 per cent) and high fatality (90 to 100 per cent). Most cases and fatalities due to MD occur during the first 2 years of life (Harkness & Van der Lugt 1994; Radostits et al. 2007b).

BVDV can be transmitted by a variety of routes, including direct transmission between animals by aerosol, transplacental transmission, semen and embryo transfers, mechanical transmission by biting flies, venereal transmission, fomites, and by iatrogenic means including hypodermic needles and equipment shared between rectal examinations. Transmission of BVDV and the other pestiviruses via these indirect methods occur not infrequently, for example contaminated vaccine (OIE 2017d; Taus et al. 2014). BVDV can be excreted via nasal discharge, saliva, semen, faeces, urine, tears and milk (Radostits et al. 2007b). Animals infected after birth are most likely to acquire infection via the respiratory route, with increased stocking density favouring transmission. Even after development of protective immunity following acute disease, infective BVDV remains in animal tissues for many months and these animals may still be a risk source for transmission (Collins et al. 2009). Acutely infected animals are an inefficient means of horizontal transmission though it is possible for groups to act as a reservoir for infection; PI animals are significantly more effective at spreading the virus (Collins et al. 2009; Lanyon et al. 2014; Potgieter 2004; Walz et al. 2010).

Viraemia occurs 2–4 days after exposure and, in acute infections, the virus can be isolated from serum or leucocytes for 3–10 days post infection (Potgieter 2004).

Other than domestic cattle, natural pestivirus infections and disease also occur in sheep, pigs, goats and a range of captive and free living ruminant species (Wolff et al. 2016). Whilst numerous serosurveys have confirmed exposure to BVDV in Bovidae, as well as other ruminant families, there are very few reports of isolation of the viruses from non-domestic ruminants

(Anderson & Rowe 1998b; Frölich & Flach 1998; Passler & Walz 2010; Potgieter 2004; Ridpath & Neill 2016; Wolff et al. 2016). Free-ranging herds of many African bovidae species (buffalo, wildebeest, kudu, eland, nyala and bushbuck) have been shown to have moderate to high seroprevalence rates (Anderson & Rowe 1998a; Depner, Hubschle & Liess 1991; Potgieter 2004). Virus has been isolated from nilgai, waterbuck, serow (*Capricornis sumatraensis*), pygmy goat (*Capra hircus*) and African buffalo (Doyle & Heuschele 1983a; Van Campen, Frölich & Hofmann 2001).

Evidence suggests BVDV or closely related viruses cause clinical disease in buffalo, eland and water buffalo. Lesions associated with BVDV have been reported, often in captive settings, in red-fronted gazelle (*Eudorcas rufifrons*), Dorcas gazelle (*Gazella dorcas*), banteng, Grant's gazelle (*Gazella granti*), gaur (*Bos gaurus*), American bison (*Bison bison*), and yak (*Bos grunniens*) (Van Campen, Frölich & Hofmann 2001).

Evidence suggests non-domestic ungulates may develop Persistent Infections. The efficiency with which BVDV crosses the placental barrier differs between host species with domestic cattle, white tailed deer, and possibly also red deer, the most efficient at creating PIs (Anderson & Rowe 1998b; Passler et al. 2010; Passler et al. 2014; Passler et al. 2007; Rodríguez-Prieto et al. 2016; Uttenthal et al. 2005). Non-domestic bovidae are not known to be efficient in this regard.

Although transmission of BVDV between species has been reported, in most cases cattle are the source of the virus (Passler et al. 2009; Potgieter 2004). There is evidence that the virus can be maintained in wildlife populations (Passler et al. 2007; Rodríguez-Prieto et al. 2016; Uttenthal et al. 2005; Wolff et al. 2016). One study found evidence of endemic viral circulation in free-ranging eland (Anderson & Rowe 1998b). Endemic BVD1 and BVD2 infection has been demonstrated in sympatric herds of free-ranging bighorn sheep, mountain goats and mule deer (Wolff et al. 2016). PI individuals have been reported in free-ranging mule, and white-tailed deer, and captive mountain goats (Chase et al. 2008; Duncan et al. 2008; Nelson et al. 2008). MD has also been reported in some wildlife species, supporting the presence of persistent infections in some of these species (Potgieter 2004). Cattle remain the most significant species in the epidemiology of BVDV. The role of some deer species continues to be studied. The importance of other species remains unknown.

Bulls excrete BVDV in semen during acute and persistent infection. Viral presence is detectable in semen for over 5 months post infection (Givens et al. 2003). The virus can be isolated from whole semen, seminal plasma and washed cell fraction of fresh and frozen semen (Revell et al. 1988). Infected bulls that are neither PI nor viraemic may have persistently infected testicular tissues, and may shed virus in semen intermittently (Givens et al. 2009; OIE 2015a; Voges et al. 1998).

### Diagnosis

A wide range of molecular, antigen (Ag), and antibody (Ab) tests exist to assist diagnosis of BVDV and many have been employed in wild ruminant studies. Tests should be selected based on the epidemiology of BVDV and the desired goal of testing, for example identification of PI animals. Animals that have never been exposed to BVDV will test negative for Ab, Ag, and virus. Animals or late-term, immunocompetent foetuses that have experienced an acute infection will test Ab positive and, generally, Ag or virus negative. PI individuals will return a positive Ag or virus test and negative Ab result (Lanyon et al. 2014). The most commonly used tests are the Ag ELISA and the real-time reverse-transcriptase polymerase-chain-reaction (real-time RT-PCR),

which are sufficiently sensitive to detect viral particles in tissue samples several months after infection (Hanon et al. 2014; Lanyon et al. 2014).

Seroconversion is generally detectable by day 14 post-infection and Ab titres continue to rise beyond day 50 post-infection. The ELISA and the Virus Neutralisation Test (VNT) are the most widely used tests for detection of antibody (Bauermann, Flores & Ridpath 2012; Lanyon et al. 2014). Seronegative animals either have never been exposed to the disease *or* are PI animals. Seropositive animals indicate exposure and also suggest that the individual is not PI. A high prevalence of seropositivity in a herd indicates a high likelihood of ongoing infection (and the presence of a PI animal). A herd of seronegative adult animals is unlikely to contain a PI animal, and likely represents a naïve herd (Anderson & Rowe 1998a; Lanyon et al. 2014; Potgieter 2004). Ab ELISAs are sufficiently sensitive to be used on pooled serum samples (Lanyon et al. 2010).

Multiple Ag ELISAs exist commercially, including pen-side SNAP® tests. Whilst specificity is excellent, sensitivity may be lower, although advances in test technology have improved this (Aduriz, Atxaerandio & Cortabarria 2015; Hanon et al. 2014; Lanyon et al. 2014; OIE 2015a).

The short viraemic period can make it difficult to directly demonstrate virus, other than in PI animals. Isolation of virus is generally not possible beyond 14-21 days post-infection, though the use of sensitive detection techniques such as RT-PCR may extend this period (Collins et al. 2009). Several RT-PCR methods have high sensitivity (Lanyon et al. 2014; Mari et al. 2016; OIE 2015a; Potgieter 2004; Yan et al. 2011). RT-PCR has also been applied to pooled samples with good sensitivity.

Detection of BVDV in reproductive material is challenging. Semen can be tested by virus isolation or RT-PCR but is sensitive to sample collection, handling, and transport conditions and thus careful preparation is required (OIE 2015a).

### **Clinical signs**

BVDV causes a wide variety of clinical manifestations but may be characterised by inappetance, depression, fever and mild diarrhoea. Viraemia is transient and cases recover rapidly within a few days. Infection with BVDV2 may result in more severe and sometimes haemorrhagic diarrhoea, severe lymphopenia, severe alimentary epithelial necrosis and lymphoid depletion (Kelling et al. 2002). Small vesicle-ulcers develop in epithelial cells, resulting in erosions throughout the oral cavity and gastrointestinal tract. Ill thrift is common and death usually follows within 2 weeks of onset of clinical signs (Harkness & Van der Lugt 1994). MD may be acute or chronic; both are associated with severe clinical signs and pathology and are typically fatal. As in cattle, clinical signs and pathogenesis are variable depending on host and virus associated factors, though respiratory and reproductive signs are common.

### **Prevention**

BVDV vaccines are primarily used for disease control purposes (OIE 2015a). Live, killed, and modified-live vaccines are available and are able to provide good protection unless the animal is exposed to constant viral challenge from a PI animal (Rodning et al. 2010).

Control programs typically focus on PI animal identification, eradication, and prevention. Herd eradication processes have been hampered by the ability of acutely infected animals to sometimes act in a reservoir fashion and the prolonged presence of virus even in recovered

animals (Collins et al. 2009). In cattle, failure to maintain virus free herds is generally due to inadvertent procurement of PI cattle, or pregnant cows with unknown BVDV foetal status. All newly acquired animals should be isolated and their offspring tested to ensure they are free of BVDV, prior to introduction to the herd. Semen should only be used from bulls known to be free from BVDV infection. The rare but important occurrence of persistent testicular infection, sometimes lifelong, means that semen should also be screened using PCR methods, unless there is confidence that the bull is completely naïve to BVDV exposure (OIE 2015a; Walz et al. 2010).

#### **4.5.3 Current biosecurity measures**

There are no recommendations in the OIE Code. Australian conditions for live camelids include a blood test in pre-export quarantine (PEQ) with a negative result to either an antigen-ELISA, a VNT, or a PCR. Australian conditions for semen include premises freedom, donor isolation, and diagnostic testing of the donor.

#### **4.5.4 Risk review**

BVD2 is present in approved countries. In Australia, only the low virulent BVD1 strain has been reported. BVD2 is a nationally notifiable disease.

BVD1 and BVD2 have similar epidemiology and both cause BVD and MD, but BVD2 is more pathogenic and is not present in Australia. Much of the available literature does not distinguish between BVD1 and BVD2 strains, when referring to infection in non-domestic bovidae.

The following key points are relevant to the biosecurity risk of BVDV2 in non-domestic zoo bovidae:

- Infection with BVDV2 may cause BVD and MD, both of which cause serious disease in cattle. Significant reproductive losses and morbidity can occur in infected herds. Infection due to BVDV2 is more severe than BVDV1 although there is some cross-over in presentation.
- BVDV2 causes disease primarily in cattle, but a range of non-domestic bovidae may be infected by BVDV2 and may show signs of disease similar to those seen in cattle. Interspecies transmission is known to occur.
- The epidemiology of BVDV is complex. BVDV is primarily transmitted through exposure to PI animals, which shed significant amounts of virus for the duration of their life.
- Acutely (transiently) infected animals may also transmit the virus, which persists in tissues and blood for many months post-infection.
- There are many horizontal and vertical transmission pathways; respiratory aerosols and transplacental transmission are the greatest risk pathways. The virus may spread via fomites, semen and other biological materials used in artificial insemination.
- There is evidence that some non-domestic bovidae species may become acutely infected or give birth to persistently infected animals.
- Vaccination has limited effectiveness in preventing disease transmission and is used primarily for control purposes.
- The diagnosis of BVDV infection is complex, but the range of tests and procedures available allows for reliable identification of risk animals and semen.

- Infected bulls that are neither PI nor viraemic may have persistently infected testicular tissues, and may shed virus in semen intermittently.
- There is a risk pathway for BVD2 (and pestiviruses in general) via fomites from an infected zoo animal.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### **4.5.5 Conclusion**

Accordingly and based on the preceding information, risk management measures are warranted for live animals and semen.

Australia's proposed biosecurity measures for BVD2 for live zoo bovidae are:

##### **Option ONE**

- Since birth the animal was continuously resident in a country free from BVD. The disease must be compulsorily notifiable.

##### **Option TWO**

- During pre-export quarantine, a blood sample was drawn from the animal and tested by an RT-PCR test approved by the department. The test was negative to BVD.

AND

- During pre-export quarantine, a haired skin sample was taken from the animal (ear notch or caudal tail fold) and tested using an antigen ELISA test approved by the department. The test was negative to BVD.

##### **Option THREE**

- During pre-export quarantine, a blood sample was drawn from the animal and tested by:
  - A RT-PCR test or antigen ELISA test approved by the department. The test was negative to BVD.

AND

- An antibody ELISA test or VNT approved by the department. The test was negative to BVD.

##### **Option FOUR**

- For 180 days immediately before export the animal for export was part of a zoo collection subject to a documented BVD screening program. The screening program must include:
  - Diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department. The testing regime must establish that neither infection nor persistently infected animals are present.
  - The collection must have been a 'closed-herd' during that time.
  - The collection must contain at least four zoo bovids.

AND

- The animal was tested by the screening program during this time.

Australia's proposed biosecurity measures for BVD2 for zoo bovidae semen are:

#### Option ONE

- The semen was tested by an approved virus isolation or approved RT-PCR test for BVD, with negative results.

#### Option TWO

- On the *last* day of the semen collection period, a blood sample was drawn from the donor animal and tested by:
  - A RT-PCR test or antigen ELISA test approved by the department. The test was negative to BVD.
 AND
  - An antibody ELISA test or VNT approved by the department. The test was negative to BVD.

## 4.6 Brucellosis (*B. abortus* and *B. melitensis*)

### *B. abortus*

#### 4.6.1 Background

Bovine brucellosis is caused by the bacterium *Brucella abortus*, in the family *Brucellaceae*. It is an important cause of disease in livestock and exhibits a broad host range. Bovine brucellosis is highly contagious, causing abortion and infertility in cattle; animals often remain chronically infected unless treated. It is an important zoonosis, causing undulant fever in humans.

Bovine brucellosis is widespread throughout the world but was eradicated from Australia as part of the Brucellosis and Tuberculosis Eradication Campaign (BTEC), with freedom in accordance with the OIE Code declared in 1989 (DAFF 2009). Other countries reporting eradication of bovine brucellosis include Austria, Belgium, Canada, Denmark, Finland, Germany, Japan, the Netherlands, New Zealand, Norway, Sweden, Switzerland and the United Kingdom. In the United States the disease is limited to free-ranging bison and wapiti (*Cervus Canadensis*) (OIE 2016g). It is a disease of significant economic importance.

*B. abortus* is an OIE-listed disease (OIE 2018c) and is not present in Australia. It is a nationally notifiable disease in Australia (Department of Agriculture and Water Resources 2016). It is a zoonotic disease and nationally notifiable in humans (Department of Health 2015). *B. abortus* is a Category 2 disease under the emergency animal disease response agreement (EADRA), with potential for rapid spread, severe production losses and potential export trade losses.

#### 4.6.2 Technical information

##### Epidemiology

Serosurveillance is the major diagnostic tool used in monitoring wildlife populations for disease. However, the serological tests for brucellosis cannot distinguish between antibodies to *B. abortus*, *B. melitensis* and *B. suis*, and it is not always feasible for wildlife studies to follow serology with PCR in order to distinguish which type of *Brucella* antibody is detected. *B. abortus* is noted to have a broader host range in wildlife compared to *B. melitensis*.

The major reservoir of *B. abortus* is domestic cattle, though some wild ruminants (bison, African buffalo and elk) are also considered reservoirs of the infection, with the potential for transmission to cattle (Godfroid et al. 2013; Rhyan 2013). Pigs, sheep, goats and farm dogs are also occasionally infected.

The most significant epidemiological features of brucellosis are the variable incubation period, latency and the delay in seroconversion following infection, which makes detection of early infection problematic. About 15 per cent of cattle in herds infected with *B. abortus* abort before seroconversion occurs, and about 5 per cent of progeny of infected dams retain infection and become seropositive only after first parturition (Nicoletti 2010).

Abortions induced by brucellosis result in a large amount of infectious material being shed into the environment which is the primary mode of transmission. Bacteria may be shed intermittently in milk after abortion or calving. Bacteria also may be shed in urine and faeces. Shedding of bacteria declines rapidly, following calving or abortion, and infected cows may then remain non-infectious until the next pregnancy, which induces a rapid build-up of infectious organisms within the reproductive tract.

*B. abortus* is transmitted between individuals by ingestion, inhalation, through skin abrasions and through mucous membranes. The source of infection may include direct contact with infected cattle, or contact with infected reproductive material and discharges, or contaminated pasture and feed (Corbel & MacMillan 1998; Godfroid et al. 2004a). Transmission can also occur in utero and from suckling milk from infected dams. The organism may survive for many months in soil, faeces, and aborted fetuses, but does not replicate outside the host. Transmission between herds occurs through movement of infected animals.

The incubation period in cattle may vary from weeks to months depending on the stage of pregnancy when infection occurs (Godfroid 2004). In cattle, individuals may carry the infection for years (often localised in udder and lymph nodes) and the organism may be excreted in vaginal discharge at the time of calving and in milk. The duration of infection in bison is unknown, but is likely to be prolonged (Rhyan et al. 2013).

Serological evidence of infection with *Brucella* (assumed and sometimes confirmed to be due to *B. abortus*) has been found in a variety of wild bovidae including eland, wildebeest, impala, waterbuck, bushbuck, oryx and African buffalo (Bishop, Bosman & Herr 1994; Godfroid 2004; Gradwell et al. 1977; Madsen & Anderson 1995; Paling et al. 1988; Waghela & Karstad 1986). The organism has been isolated from African buffalo and American bison and these two species are considered important reservoirs for the disease in their range states (Rhyan et al. 2013). Transmission between bison and cattle has been demonstrated experimentally (Rhyan 2000). Natural infection (with typical clinical signs) has been seen in bighorn sheep held in a wildlife research facility and housed close to infected elk (Kreeger et al. 2004). The organism has also recently been isolated from goral (*Naemorhedus goral raddeanus*) and wild Chinese water deer (*Hydropotes inermis*) in South Korea (Lam Quang et al. 2011; Truong et al. 2016).

None of the bovid species covered in the scope of this import risk analysis (IRA) have been identified as reservoirs of *B. abortus*. Godfroid, Nielsen and Saegerman (2010) comment that 'in countries where bovine brucellosis eradication programmes are close to their end, there are no known sustainable reservoirs of *B. abortus* in wild species other than bison and elk'. This is consistent with epidemiology described in the literature, where transmission to other species

groups is possible, but maintenance or a significant role is rarely found outside the preferred host range (Boeer et al. 1980; Muñoz et al. 2010; Schnurrenberger et al. 1985; Truong et al. 2016). There are no published reports of *B. abortus* in zoo bovids in the countries covered by this IRA.

*B. abortus* can be found in the necrotic and desquamating seminal epithelial cells and in macrophages shed in semen, seminal fluid and urine from bulls (Bendixen & Blom 1947; Robison et al. 1998). Infected bulls usually excrete the organism in the semen during the acute stage but as the disease becomes chronic, excretion may cease or become intermittent.

*B. abortus* was isolated from 80 consecutive ejaculates collected from a bull over an 18 month period (Manthei, DeTray & Goode, Jr. 1951). *B. abortus* was able to be cultured from the semen of several Yellowstone National Park bison bulls (Frey et al. 2013b). The organism can survive freezing and be transmitted by artificial insemination (Williams 2003). Data more specific to the semen of species of zoo bovids covered by this policy was not located.

### **Clinical signs**

Clinical signs in cattle are related to infection of the reproductive organs and include late term abortion (primarily of the first gestation after infection), birth of weak offspring, retained foetal membranes and metritis. In bulls, infection may result in orchitis, which may be manifest by scrotal swelling. Swollen joints may be seen in chronically infected animals.

Infections in wildlife species may be asymptomatic, or animals may present with similar clinical signs to those seen in domestic cattle (Godfroid 2004; Rhyan 2013).

### **Diagnosis**

Diagnosis of brucellosis can be challenging in both domestic livestock and wildlife. It is recommended that test protocols should be developed based on the epidemiological circumstances. Careful interpretation of results is required (Godfroid, Nielsen & Saegerman 2010). Brucellosis tests can have important limitations in detecting infected individuals despite being adequate for group situations (Godfroid et al. 2013).

Serological testing is widely used for screening of wildlife. Most tests have been transposed from domestic livestock to wild species without species validation because the immunodominant antigens are associated with the surface 'smooth' lipopolysaccharide that is common to all the naturally occurring *Brucella* biovars (Davis et al. 1990; Godfroid et al. 2013; Godfroid, Nielsen & Saegerman 2010). This also means that serological tests cannot distinguish between antibodies to *B. abortus*, *B. melitensis* and *B. suis*. Some tests (e.g. Rose Bengal) also have cross-reactivity with non-*Brucella* organisms. Antibody levels are known to wane after infection. The duration of antibody persistence is not known for most wildlife species (Godfroid, Nielsen & Saegerman 2010).

The Buffered *Brucella* Antigen Tests (Rose Bengal and buffered plate agglutination tests; BBAT) are among the tests prescribed for trade by the OIE, and are rapid tests, with good sensitivity and specificity in cattle. The Rose Bengal test requires high-quality serum, which may be a consideration for sampling in field situations (Godfroid et al. 2013).

Indirect and competitive ELISAs (iELISA and cELISA, respectively) are prescribed tests for trade by the OIE, demonstrating good sensitivity and specificity (Animal Health Australia 2005a; Mainar et al. 2005). The iELISA is noted to be more sensitive but more vulnerable to non-specific

reactions than the cELISA (Godfroid, Nielsen & Saegerman 2010; Perrett et al. 2010). Both ELISAs are being increasingly employed in the screening of wildlife populations (Truong et al. 2016).

The complement fixation test (CFT), in parallel with the brucellin skin test, recently showed good sensitivity in detecting water buffaloes inoculated with RB51 brucellin (Tittarelli et al. 2015). Some authors regard the CFT as a low sensitivity test, and difficult to standardise, hence the shift towards ELISAs (Godfroid, Nielsen & Saegerman 2010). A fluorescence polarisation assay (FPA), used in brucellosis control and certification programs in North America and Europe, is a prescribed test for trade by the OIE. Other tests include a PCR, the CFT, and serum agglutination test (SAT).

Bacteria may be detected in foetal tissues and membranes, and vaginal and uterine swabs of clinically affected animals, using culture, staining or molecular techniques (Godfroid, Nielsen & Saegerman 2010). Culture of *Brucella* organisms remains the gold standard of diagnosis.

### **Prevention**

Prevention of bovine brucellosis at a herd level focuses on ensuring newly acquired animals are free of infection, since transmission between herds occurs via movement of infected animals. Facilities previously housing infected individuals should be left empty for one month, prior to introduction of new animals (Animal Health Australia 2005a).

Vaccination is used in domestic livestock to minimise infection risk and has been a cornerstone for national or regional bovine brucellosis eradication programs. Vaccination has been used in commercial bison herds and to a limited extent in wildlife (Cheville, McCullough & Paulson 1998; Davis & Elzer 2002; Thorne 2008). The effectiveness of cattle vaccines in non-domestic bovidae is largely untested.

### **Treatment**

Successful treatment of infected animals is difficult. Antibiotics including long acting tetracyclines and streptomycin have been used parenterally and by intra-mammary infusion, but eradication of the agent is not guaranteed, and treatment is rarely seen as a reliable option for control of the disease (Animal Health Australia 2005a). Infected semen should be destroyed (Williams 2003).

## **4.6.3 Current biosecurity measures**

Current biosecurity measures for bovine brucellosis include country freedom and thermal treatments of susceptible commodities (e.g. milk). The OIE Code recommendations include country or zone freedom and testing for bovids (OIE 2016a).

## **4.6.4 Risk review**

Brucellosis due to *B. abortus* is present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of bovine brucellosis in zoo bovidae:

- Bovine brucellosis is a disease of significant economic and human health importance. It was eradicated from Australia as part of the BTEC campaign.

- Bovine brucellosis is transmitted through direct or indirect contact with infectious material, generally produced post-abortion.
- Infection with brucellosis may persist for years in cattle, and presumably for similar times in affected bison and other bovidae.
- Evidence of infection with bovine brucellosis has been reported in a non-domestic bovid species. Although buffalo, bison and wild sheep species may be considered reservoirs of infection, there is no evidence that any of the species of non-domestic bovids covered by this policy act as reservoirs of infection.
- Bovine brucellosis has not been reported in zoo animals in approved countries.
- The effectiveness of bovine brucellosis vaccinations in zoo bovid species is largely untested.
- Infection with bovine brucellosis in zoo bovidae may be subclinical.
- Treatment of infected animals is problematic, with a poor cure rate.
- Diagnosis of bovine brucellosis can be challenging and an appropriate combination of tests may be required to aid detection. Antibody levels are known to wane after infection.
- Semen is a risk material. Bacteria may be shed from infected animals for prolonged periods, survive freezing, and be transmitted through artificial insemination.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

## ***B. Melitensis***

### **4.6.5 Background**

Caprine and ovine brucellosis is caused by the bacterium *Brucella melitensis*, in the family *Brucellaceae*. *B. melitensis* is an important cause of disease in sheep and goats. Infection results in abortion, poor milk yield and orchitis, and Malta fever in humans.

*B. melitensis* has a worldwide distribution, albeit more restricted than *B. abortus*. It occurs mainly in the Mediterranean region, west and central Asia, Central America, South America and Africa. Disease due to *B. melitensis* has not been reported in Southeast Asia and does not occur in Australia, New Zealand and Canada. In USA, an outbreak was reported in goats and a cow in Texas in 1999. The disease, probably introduced from Mexico, has since been eradicated.

*B. melitensis* is an OIE-listed disease (OIE 2018c) and is not present in Australia.

It is a nationally notifiable disease (Department of Agriculture and Water Resources 2016). It is a serious zoonotic disease and nationally notifiable in humans (Department of Health 2015). *B. melitensis* is a Category 2 disease in the EADRA, with the potential to spread widely, cause production losses and disrupt trade.

### **4.6.6 Technical information**

#### **Epidemiology**

Although serosurveillance is a major diagnostic tool used in monitoring wildlife populations for disease, the serological tests for brucellosis cannot distinguish between antibodies to *B. abortus*, *B. melitensis* and *B. suis*. Reports of infection based solely on serological results are omitted from

this chapter but included instead in the chapter on *B. abortus*, which has a much broader host range in wildlife, whilst *B. melitensis* is noted to be restricted in host range in comparison. The epidemiological information presented in this risk review should also be considered alongside that of the *B. abortus* review.

*B. melitensis* is normally a disease of goats and sheep. All caprine breeds are natural hosts for *B. melitensis* and are generally susceptible. Disease due to *B. melitensis* has also been reported in cattle, alpacas and camels. Cattle herds are at risk of infection when grazing on pasture previously grazed by migrating infected flocks of sheep and goats, or fodder harvested from such fields.

Animals become infected after exposure to infected placenta, foetal fluids or vaginal discharges from infected animals. The organism continues to be shed in vaginal discharges for up to two months in sheep and three months in goat's post-partum (Garin-Bastuji et al. 1998). The young acquire latent infection by ingesting colostrum and milk of infected dams and manifest the disease after sexual maturity. Abortions do not usually occur in cattle but cases have been documented in cattle in close contact with infected goats and sheep (Kahler 2000). Most animals remain chronically infected, usually for life.

Uterine infection persists for up to five months after abortion, while the mammary glands and associated lymph nodes remain infected for several years. Flocks remain infected for years, often at high prevalence, even in the absence of clinical signs (Godfroid et al. 2004b). Spontaneous recovery has occurred, particularly in goats infected while not pregnant. Animals generally abort once, although re-infection of the uterus occurs in subsequent pregnancies with organisms being shed with membranes and foetal fluids.

*B. melitensis* is not commonly diagnosed in non-domestic ruminants (Godfroid et al. 2004b). Infection has been demonstrated, with concurrent disease, in wild ibex (*C. ibex*) and chamois (*R. rupicapra*) in Europe (Ferroglio et al. 1998; Garin-Bastuji et al. 1990; Munoz et al. 2010). However the majority of these infections seem to occur as spill over events from domestic sheep and goats and wild ruminants are generally not considered to be significant to the epidemiology of the disease. Recent studies have suggested that re-emergent bovine and human brucellosis cases in France (due to *B. melitensis*) may have occurred as a result of spill over from Alpine ibex, indicating that free-ranging non-domestic caprinae in Europe may act as a reservoir for this infection (Garin-Bastuji et al. 2014; Mick et al. 2014). *B. melitensis* has been reported in an impala, a captive Arabian oryx and also sable antelope (Schiemann & Staak 1971; Spickler 2018). Multiple authors conclude that the bovid species covered by this policy do not play a significant role in the maintenance of either *B. abortus* or *B. melitensis* (Coelho, Díez & Coelho 2015; Garin-Bastuji et al. 2014; Godfroid et al. 2005; Munoz et al. 2010).

It is generally accepted that domestic billy goats and rams do not play an important role in the epidemiology of *B. melitensis*, however orchitis and epididymitis are common sequelae of infection (Godfroid et al. 2004b). The bacterium is noted to be shed in milk and urine. Semen is noted to be a risk source for infection though specific duration of infectivity was not located (Spickler 2009b; Williams 2003). Artificial insemination is considered a risk for spread of brucellosis. *B. melitensis* in the seminal fluid fraction of semen of bulls and rams have been identified by both PCR and direct culture method (Amin, Hamdy & Ibrahim 2001). OIE Code

recommendations include ongoing herd testing to ensure freedom and appropriate processing (OIE 2016a).

### **Clinical signs**

Clinical signs in affected animals are similar to those seen in bovine brucellosis. Infection causes placentitis with subsequent abortion, followed by vaginal discharge. Abortion rates are lower in sheep than in goats. Subsequent infertility may occur. Mastitis, loss of body condition, lameness and chronic cough are less commonly observed signs. In males, orchitis may occur.

### **Diagnosis**

Diagnosis is based on isolation and identification of *B. melitensis* organisms and on serological tests. Serological tests are the same as those developed for detection of bovine brucellosis, and are not able to distinguish *B. melitensis* infection from *B. abortus* infection (Blasco & Molina-Flores 2011; Rhyan 2013). The rose Bengal test and the CFT are recommended tests for screening herds and individual animals. An ELISA is also available. The Native Hapten (NH) gel precipitation test can distinguish infected from vaccinated animals (Díaz-Aparicio et al. ; OIE 2016a).

### **Prevention**

The live, attenuated Rev-1 vaccine is available for use in domestic livestock (Blasco & Molina-Flores 2011). There is no brucellosis vaccine that demonstrates satisfactory safety and efficacy in wildlife and thus good management practices are most effective at preventing *B. melitensis* (Godfroid et al. 2013).

## **4.6.7 Current biosecurity measures**

There are no previous biosecurity measures for caprine and ovine brucellosis. The OIE Code recommendations include country or zone freedom and testing for caprinae (OIE 2016a).

## **4.6.8 Risk review**

*B. melitensis* is present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of *B. melitensis* in zoo bovidae:

- *B. melitensis* has the potential to spread widely, cause production losses and disrupt trade. It is a serious zoonosis.
- Ovine and caprine brucellosis is transmitted through direct or indirect contact with infectious material, generally produced post-abortion.
- Infection with brucellosis may persist for years in sheep and goats without overt clinical signs.
- Diagnosis of caprine and ovine brucellosis can be challenging and an appropriate combination of tests may be required to aid detection.
- Evidence of infection with *B. melitensis* has been reported in a range of non-domestic bovid species. There is no evidence that any of the species of non-domestic bovids covered by this policy have an important epidemiological role. There is no evidence to suggest that zoo systems in approved countries would be exposed to this disease.

- Semen is a risk for transmission, however specific details of infective period are not known. The OIE Code recommends sourcing semen only from herds verified free of the disease.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### **4.6.9 Conclusion – *B. abortus* & *B. melitensis***

Accordingly, based on the preceding information, risk management measures for brucellosis (*B. abortus* & *B. melitensis*) are warranted for zoo bovids.

Australia's proposed biosecurity measures for brucellosis for zoo bovids are:

##### **Option ONE**

- For 12 months immediately before export the animal was continuously resident in a country *and* premises where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* and *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable

##### **Option TWO**

- For 12 months immediately before export or since birth the animal has only resided on premises where no clinical, epidemiological or other evidence of brucellosis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

AND

- In the 30 days immediately prior to export the animal was subjected to an approved ELISA or BBAT test for brucellosis (*B. abortus* and *B. melitensis*). In the case of post-parturient females, the test was carried out at least 30 days after giving birth. The test result was negative.

Australia's proposed biosecurity measures for bovine brucellosis for zoo bovidae semen are:

##### **Option ONE**

- For 12 months immediately before semen collection or since birth the animal was continuously resident in a country *and* premises where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* or *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable.

##### **Option TWO**

- For 12 months immediately before semen collection or since birth the animal has not resided on any premises in the country of export where clinical, epidemiological or other evidence of brucellosis (*B. abortus* or *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable.

AND

- The donor animal was subjected to an approved ELISA or BBAT test for brucellosis (*B. abortus* or *B. melitensis*) within the 30 days immediately after collection. The test result was negative.

## 4.7 Contagious caprine pleuropneumonia

### 4.7.1 Background

Contagious caprine pleuropneumonia (CCPP) is caused by the bacterium *Mycoplasma capricolum* subsp. *capripneumoniae* (Mccp). CCPP is present in much of Africa and some countries in Asia, including Middle Eastern countries. A distinct Asian cluster strongly indicates that CCPP was not recently imported to continental Asia but has been endemic in the area for a long time (Manso-Silvan et al. 2011; Samiullah 2013). Since 2005 the disease has been present in the European part of Turkey (Thrace), with potential to move into the Balkans.

There have been few declarations of CCPP outbreaks to the OIE in the last 15 years, partly due to lack of awareness and confusion with other *Mycoplasma*, *Pasteurella* and peste des petits ruminants infections, and also due to the difficulty in definitively diagnosing the disease. An accurate distribution of the disease is unknown (Prats-van der Ham et al. 2015).

CCPP causes major economic losses where it is endemic. It is one of the most severe diseases of goats, being extremely contagious and frequently fatal.

CCPP is not present in Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016). CCPP is an OIE-listed disease (OIE 2018c).

### 4.7.2 Technical information

#### Epidemiology

Due to the genetic similarity of Mccp to other mycoplasma species, the overlap in clinical signs, and the difficulty of diagnosis, there is much confusion about geographic distribution, diagnosis, and best methods for control of this disease (Prats-van der Ham et al. 2015). Many previous reports of Mccp have recently been re-attributed to other pathogens (Hussain et al. 2012). False diagnoses of CCPP may continue to occur in Asia, the Middle East and Africa due to presence of several other *Mycoplasma* spp., such as *M. m. capri*, which is endemic in Greece (Nicholas & Churchward 2012).

CCPP occurs primarily in goats and for close to a century it was thought to be highly species specific (Thiaucourt, Van der Lugt & Provost 2004). More recently the pathogen has been isolated from healthy sheep in contact with Mccp-positive goats in Africa (Bölske et al. 1995; Litamoi, Wanyangu & Simam 1990) and CCPP has emerged as a significant disease in a variety of free-ranging and captive species.

Naïve animals are severely affected by CCPP with widespread morbidity and mortality reaching 100 per cent (Nicholas & Churchward 2012; Srivastava et al. 2010). In the last decade numerous outbreaks have occurred in goat herds in Oman, Iran, Ethiopia, Tajikistan, Yemen, Thrace and Mauritius with mortalities numbering in the hundreds and thousands (Arif et al. 2007; Spickler 2015b).

There is significant evidence that naturally acquired CCPP can result in clinical disease in a range of bovidae sub-families. In 2012, approximately 2,400 free-ranging Tibetan antelope (*Pantholops hodgsonii*), around 15 per cent of the entire estimated species population, died from CCPP

infection (Yu et al. 2013). Fatalities and clinical disease due to CCPP have been reported in captive non-domestic bovidae including wild goat (*Capra aegagrus*), Nubian ibex (*Capra ibex nubiana*), Laristan mouflon (*Ovis orientalis laristanica*) and gerenuk (*Litocranius walleri*) (Arif et al. 2007). A fatal case of CCPP was reported in captive Arabian oryx housed in close, but not direct, proximity to two densely stocked captive populations of sand gazelle (*Gazella subgutturosa marica*) suffering an outbreak (Chaber et al. 2014). Only one of the 14 oryx in the enclosure was affected and none of the 85 oryx in the enclosure beyond. In the 2012 Tibetan antelope outbreak, close contact with wild goats and sheep was considered an important driver. The high mortalities suggest that the antelope population was naïve, and is consistent with CCPP's impact on naïve populations (Yu et al. 2013). The outbreak reported by Arif et al. (2007) proposes either contact with domestic goats outside the conservation area or three Nubian ibex that had been imported 12 months prior as the source of infection. In the United Arab Emirates three separate outbreaks in captive gazelle and antelope species are attributed to the introduction of sick goats and indirect contact with the gazelles at feed stations (Molnar et al. 2014; Nicholas & Churchward 2012). An experimentally infected Thomson's gazelle developed antibody but did not develop any clinical signs nor infect a goat kept in close contact with it (Paling, Macowan & Karstad 1978).

The existence of a chronic carrier state for Mccp is still uncertain (Lefevre & Thiaucourt 2004; Tigga et al. 2014). Apparently healthy carriers can excrete *Mycoplasma* spp. (Geering, Forman & Nunn 1995). Historically, outbreaks of disease in naïve regions have been associated with the introduction of apparently healthy (presumed carrier) goats from endemic areas (Nicholas & Churchward 2012; Srivastava et al. 2010). Goats and sheep may continue to shed bacteria once clinically recovered and perpetuate disease in herds (Atim et al. 2016; Thiaucourt & Bölske 1996; Wesonga et al. 2004a). Mccp has been isolated from asymptomatic sheep held in affected goats herds (Lefevre & Thiaucourt 2004). A single seropositive goat was shown to infect all 10 disease-free contacts within 2 weeks in close confinement (Ozdemir et al. 2006).

CCPP is transmitted via inhalation of infected materials, including respiratory fluids. Although highly contagious, it generally requires close contact between individuals. The indirect transmission path of CCPP to a captive oryx suggests that during periods of high bacterial load, other methods of disease transmission may occur (Chaber et al. 2014). Overcrowding and confinement are known to favour the effective circulation of *Mycoplasma* (Atim et al. 2016).

The incubation period is generally 6 to 14 days, but may be as long as four weeks. The OIE Code prescribes 45 days as the incubation period but notes that chronic carriers occur (OIE 2015c).

Mccp are fragile organisms that do not persist in the environment (Spickler 2015b). The pathogen is not known to cause septicaemia nor pathology of the genital tract and there is no evidence to suggest that embryos are infected, although other species of mycoplasma (e.g. contagious agalactica) have been shown to have infection risk in semen (Alves et al. 2013; AQIS 2000; Gregory et al. 2012).

### **Clinical signs**

The disease may present in acute or chronic forms. Signs in infected goats and antelope include fever, nasal discharge, cough, other respiratory signs and up to 80 per cent mortality. Abortion is reported in goats. Chronic CCPP is characterized by a cough, nasal discharge and debilitation. Characteristic pathology includes sero-fibrinous pleuropneumonia, sometimes unilateral, and

severe pleural effusion. Clinical signs in wild or captive wild ungulates are similar to those seen in goats (Arif et al. 2007; Spickler 2015b; Yu et al. 2013).

### Diagnosis

The fastidious nature of Mccp makes it difficult to culture and it is often missed on routine bacteriological analysis. The distribution of lesions is important for diagnosis. As CCPP does not spread beyond the thoracic cavity, the development of lesions in other anatomical sites (including septicaemia) suggests a different *Mycoplasma* species. There is no suitable test for the suspected chronic carrier state.

Multiple PCR assay techniques are available (Lorenzon, Manso-Silvan & Thiaucourt 2008; Prats-van der Ham et al. 2015; Samiullah 2013; Settypalli et al. 2016).

The Manual recommends the Complement Fixation Test (CFT) or C-ELISA for the purpose of verifying an individual or population as free from infection (OIE 2014d). Serology may be problematic when there is slow, absent, or undetectable development of antibodies in both acute and chronic cases (March, Harrison & Borich 2002; Thiaucourt & Bölske 1996; Wesonga et al. 2004b). Cross-reactivity remains an issue (Peyraud et al. 2014; Wesonga et al. 2004b). Sero-diagnosis using the Latex agglutination test (LAT) is relatively easy and is more sensitive than the CFT (Nicholas & Churchward 2012; Samiullah 2013). The c-ELISA has high specificity without cross reaction but the true sensitivity is unknown (Peyraud et al. 2014).

### Prevention

CCPP spreads to new areas by the movement of infected animals. In endemic areas, susceptible species should be kept from direct and indirect contact with goats.

Vaccination has been successful in managing outbreaks in captive antelopes and is highly effective at preventing reinfection in goats (Atim et al. 2016; Samiullah 2013). During outbreaks, antibiotic treatment and reduction in animal density may assist control. Complete elimination of Mccp by use of antibiotics is rare. Treated animals should be considered potential carriers until the true risk of this situation is understood (Nicholas & Churchward 2012).

## 4.7.3 Current biosecurity measures

There are no previous biosecurity measures for live animals and CCPP. The OIE Code recommends country, zone or premises freedom, testing, isolation and vaccination for wild and domestic goats (OIE 2018c).

Australian import conditions for caprine semen require donors to have lived since birth in countries or zones that meet OIE Code requirements for freedom from CCPP (AQIS 2000). The OIE Code does not specify measures for semen, although it does allow for countries to prohibit import or transit of goat semen from countries considered infected with CCPP (OIE 2015c).

## 4.7.4 Risk review

CCPP is not present in approved countries. It is not present in Australia and is a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of CCPP in non-domestic zoo bovidae:

- CCPP is present in western Turkey (Thrace), Africa, the Middle East and Asia. The true distribution in these countries is not clear.
- CCPP is primarily a disease of goats, but has been demonstrated to cause disease in sheep and several non-domestic bovidae species.
- A carrier state for goats is suspected but not confirmed.
- Spread of the pathogen into new regions is often associated with the movement of asymptomatic carrier goats.
- Outbreaks in naïve populations result in high morbidity and mortality. Outbreaks in non-domestic ungulates have been similar. Direct or indirect contact with goats has been implicated in most of these outbreaks.
- CCPP is one of the most severe diseases of goats but also impacts other industries.
- Clinical signs in susceptible bovidae are similar to those seen in goats.
- Transmission occurs through close contact and inhalation of aerosolised bacteria.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.7.5 Conclusion

Accordingly and based on the preceding information, risk management measures are warranted.

Australia's proposed biosecurity measures for CCPP for live zoo bovidae are:

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of CCPP has occurred during the previous 12 months and the disease is compulsorily notifiable

Australia's proposed biosecurity measures for CCPP for zoo bovidae semen are:

- Since birth, the animal was continuously resident in a country (or countries) where no clinical, epidemiological or other evidence of CCPP has occurred in any species and the disease is compulsorily notifiable.

## 4.8 Crimean-Congo haemorrhagic fever

### 4.8.1 Background

Crimean-Congo haemorrhagic fever (CCHF) is a zoonotic disease caused by infection with a Crimean-Congo haemorrhagic fever virus (CCHFV), a tick-borne virus in the genus *Nairovirus*, family *Bunyaviridae* (Swanepoel & Burt 2004). Infection results in sub clinical viraemia in a range of mammalian species including cattle, sheep and small mammals such as hares and hedgehogs. It is an important zoonosis and is the most important tick borne viral disease of humans.

CCHF is present in many countries of Eastern Europe, the Middle East, Asia and Africa. It is an emerging disease in Eastern Europe and the Middle East and recent evidence also suggests the virus may be present in Spain, Portugal and France (Bente et al. 2013; ECDC 2016). Because of

the ubiquitous nature of the vector tick species, CCHF is considered an important transboundary zoonotic disease (Yadav et al. 2015).

CCHF is an OIE multiple species listed disease (OIE 2018c). It is not present in Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016).

#### 4.8.2 Technical information

##### Epidemiology

The epidemiology of CCHF has not been fully elucidated. CCHF virus is transmitted primarily via tick bite. Transmission may also occur through direct contact with infected blood and tissues. A large number of tick species have been confirmed as vectors, however Ixodid ticks of the genus *Hyalomma* are the primary vectors (Panayotova et al. 2016; Wasfi et al. 2016). The virus may be passed between ticks by both horizontal and vertical transmission, surviving from larva to nymph to adult, and to the tick egg, and surviving between seasons, in several tick species (Meegan, Hoogstraal & Moussa 1979). Since many of the studies of CCHFV in non-*Hyalomma* genera are either experimental or focused purely on demonstration of the virus in the tick rather than transmissibility, the evidence for non-*Hyalomma* species involvement in natural transmission or maintenance of CCHF is limited (Messina et al. 2015; Papa et al. 2015). However, local transmission and circulation of CCHFV in non-*Hyalomma* species may be possible (Spickler 2007; Bente et al. 2013).

Serological studies show a large number of vertebrate species may play a role as hosts for the virus, however the key reservoir host species are not clearly understood. Not all mammalian species are susceptible to CCHF infection, or develop levels of viraemia sufficient for virus transmission via ticks (Bente et al. 2013). The incubation period is usually 1-3 days with a viremia of up to a week (Aidaros 2010; Spickler 2007).

Small wild mammals, such as lagomorphs, rodents or hedgehogs, are the first blood meal host for many larval or nymphal *Hyalomma* ticks, which then, as adults, feed on humans or livestock, enabling the virus to cycle between small mammals and livestock. Viraemia in mammalian hosts is short-lived, but sufficient to infect ticks. Ticks may remain infected for extended periods of time (several years) and they effectively act as the reservoir host (Bente et al. 2013). *Hyalomma* ticks are not known to be present in Australia (Barker, Walker & Campelo 2014).

Ticks spread the virus to a variety of wild and domestic mammalian host species. Many *Hyalomma* ticks feed preferentially on artiodactyla, as well as humans, which enables transfer of the virus to these key hosts (Meegan, Hoogstraal & Moussa 1979). Many wild and domestic animals, including cattle, goats, sheep and hare, have a role as amplifying hosts (Nalca & Whitehouse 2007; Yadav et al. 2015). Migratory birds have been implicated in the spread of ticks carrying CCHFV (although not capable of carrying the virus themselves) and further spread of the virus through Western Europe has been predicted (England et al. 2016).

Serological evidence of CCHF infection has been found in a number of non-domestic bovidae including eland, buffalo, blesbok, hartebeest, springbok, nyala, kudu (*Tragelaphus strepciceros*), sable antelope, waterbuck, reedbuck, mountain reedbuck, gemsbok (oryx) and common duiker (Spengler, Bergeron & Rollin 2016). A range of other non-bovid ungulates and other small mammal species show serological evidence of CCHF infection (Shepherd et al. 1987). Prevalence of antibodies to CCHF in wild African bovidae is generally low except in larger species (kudu, eland and buffalo) (Burt, Swanepoel & Braack 1993). This may relate to the fact that larger

animals typically host greater numbers of ticks which increases the likelihood of seroconversion, as noted with cattle for CCHF (Spengler, Bergeron & Rollin 2016). There is no evidence of a carrier state in ungulate hosts. There are no reports to suggest that wild ungulates play a significant role in the epidemiology of the disease other than acting as hosts for ticks.

The virus is highly infectious to humans. Humans primarily acquire infection through the bite of infected ticks, or through exposure to blood or other contaminated body fluids from infected livestock or human patients. Humans remain viraemic for 7-10 days but are considered dead-end hosts for the virus as they are not a source of infection for ticks (Bente et al. 2013).

Until recently, indigenous cases of CCHF had not been reported in humans in Europe west of the Balkans. However, previous evidence of CCHFV infection (antibodies) had been detected in bats in France (Meegan, Hoogstraal & Moussa 1979) and more recently, molecular evidence of CCHFV was found in ticks collected from red deer in Spain, indicating the presence of the virus in south-western Europe (Estrada-Pena et al. 2012). In 2016 two related human cases of CCHF occurred in Spain, one apparently originating from a tick bite, and the second from exposure to the first patient. A risk assessment concluded future sporadic tick-acquired cases are likely to occur in Spain (ECDC 2016).

There is no evidence of sexual transmission of CCHF in animals and its role in semen is not considered further.

#### **Clinical signs**

In humans, CCHF infection results in a range of presentations, ranging from mild to severe, with a mortality rate reaching 30 per cent or higher. The most common presentation is a febrile, flu like syndrome, with signs suggestive of haemorrhagic fever (Bente et al. 2013).

Infection in cattle, sheep and other small mammals rarely causes clinical signs. Clinical signs have not been reported in non-domestic bovidae (Bente et al. 2013; Shepherd et al. 1987).

#### **Diagnosis**

Molecular techniques can be used to detect the virus in the blood of the host during viraemia. A variety of serological tests are available to detect antibodies to CCHF virus, in both animals and humans, including the fluorescent antibody technique (FAT) and an ELISA in cattle with high sensitivity and specificity. CCHF virus is difficult to grow in cell cultures.

#### **Prevention**

A vaccine is available for use in humans in high risk situations. Movement of livestock (and their accompanying ticks) has been implicated in the geographic spread of CCHF (Yadav et al. 2015). Livestock and other animals should undergo tick control prior to movement from CCHF endemic areas. Tick control may be utilised in endemic areas (Aidaros 2010).

### **4.8.3 Current biosecurity measures**

There are no previous biosecurity measures for CCHF. There are no recommendations in the OIE Code (OIE 2018c).

### **4.8.4 Risk review**

CCHF is present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of CCHF in zoo bovidae:

- CCHF is present in many countries of Eastern Europe, the Middle East, Asia and Africa. It is an emerging disease in Eastern Europe and the virus may be present in Spain and France.
- CCHF is a transboundary disease, transmitted predominantly by ticks but also infected blood and tissues, and is a serious zoonotic disease.
- A wide range of mammalian species may act as hosts to the virus, including humans and bovidae species.
- Serological evidence of CCHF has been detected in a wide range of wild ruminant species, including bovid species held in zoos.
- Zoo bovidae species may have a role as amplifying hosts for CCHF, in common with a wide variety of mammalian species.
- Viraemia in bovidae is brief and there is no evidence of a carrier state. CCHF rarely causes symptoms in animals.
- Persistence of the virus in the environment is largely through survival and transmission within the tick vector.
- Zoo bovid import conditions include management for ticks during the pre-export quarantine period and inspection of the animal in post-arrival quarantine.
- There is no evidence that CCHF can be transmitted in mammalian semen.
- Spread and establishment of CCHF in Australia is unlikely as *Hyalomma* ticks (the primary vector) are not present. Local spread and circulation in other tick species may be possible.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.8.5 Conclusion

Based on the preceding information, risk management measures for zoo bovidae and their semen are not warranted. The risk of tick transmission is managed by pre and post arrival quarantine inspections and acaricide treatment during the pre-export quarantine period.

## 4.9 East Coast fever & Mediterranean theileriosis

### 4.9.1 Background

Theileriosis is caused by tick-transmitted protozoal parasites in the genus *Theileria*, order *Piroplasmida*, phylum *Apicomplexa*. This includes parasites previously in the genera *Cytauxzoon*, *Gonderia* and *Haematoxenus*, which are now included in the genus *Theileria*. Bovine theileriosis primarily affects cattle and both African and Asian buffalo.

A number of different species of *Theileria* cause disease, primarily in cattle, which are of major economic importance to cattle industries due to movement restrictions, production losses, and high mortality rates in susceptible animals (Spickler, 2009). The most important is *T. parva* which occurs in East Africa, Malawi, areas of Zambia and Zimbabwe, South Africa, Botswana and Angola. Under the previous taxonomy, *T. parva* was divided into subspecies; *T. parva* caused East Coast fever, *T. parva lawrenci* caused corridor disease and *T. parva bovis* caused Zimbabwe theileriosis, all significant diseases of cattle. These subspecies are no longer formally used. *T. annulata*, another economically important *Theileria*, causes Mediterranean theileriosis (also

known as tropical theileriosis) in cattle. *T. annulata* occurs in North Africa, southern Europe, the Middle East and central Asia.

*Theileria parva* and *T. annulata* are not present in Australia and are OIE-listed diseases (OIE 2018c). They are nationally notifiable animal diseases (Department of Agriculture and Water Resources 2016). The other species of *Theileria* discussed in this risk review are not OIE-listed diseases and are not nationally notifiable animal diseases.

East Coast fever, caused by *T. parva* is an EADRA category 4 disease and if uncontrolled, is expected to cause production losses with domestic and export effects (Animal Health Australia, 2018).

## 4.9.2 Technical information

### Epidemiology

*T. parva* is considered likely to be originally a buffalo parasite that has become adapted to cattle (Lawrence, Perry & Williamson 2004a). African buffalo are considered asymptomatic maintenance hosts. Waterbuck were experimentally infected with *T. parva*, without evidence of clinical disease, but there is no evidence that they are important in the epidemiology of the disease. Additionally, natural infection has not been found in waterbuck in *T. parva* endemic areas (Githaka et al. 2014). *T. parva* has not been found to be infective to other members of the family bovidae (Githaka et al. 2014; Stagg et al. 1994). Non-domestic bovidae (other than African buffalo) are not reported to be involved in the epidemiology of *T. parva* (Bishop et al. 2004; Lawrence, Perry & Williamson 2004a).

Transmission of *T. parva* and *T. annulata* occurs only via ticks. Ticks of the genus *Rhipicephalus*, particularly *R. appendiculatus*, are the main vectors of *T. parva* and related theilerioses in Africa (Lawrence, Perry & Williamson 2004a; Lawrence et al. 2004). *T. parva* is restricted to those areas in sub-Saharan Africa where *R. appendiculatus* occurs. Australia has several species of *Rhipicephalus*, (but not *R. appendiculatus*) but there is no evidence that species other than *R. appendiculatus* have a significant role in natural transmission. *T. annulata* is transmitted by ticks of the genus *Hyalomma* and a single infected tick is considered capable of transmitting a fatal infectious load in cattle (Pipano & Shkap 2004). No species of *Hyalomma* ticks occur in Australia (Barker, Walker & Campelo 2014).

East Coast fever maintains a cycle within cattle, without the involvement of buffalo or evidence that other wildlife play a role in its epidemiology (Grootenhuys 1989; Lawrence, Perry & Williamson 2004a; Lawrence et al. 2004; Oura et al. 2011). Zimbabwe theileriosis cycles within cattle populations in Africa and there is no evidence that wildlife are involved in the epidemiology (Grootenhuys & Olubayo 1993; Lawrence, Perry & Williamson 2004b). Buffalo-associated theileriosis in cattle (commonly called corridor disease) occurs in areas where African buffalo and cattle intermingle, within the natural range of the transmitting tick *R. appendiculatus*. There is no evidence that other wildlife are involved in the epidemiology (Gachohi et al. 2012; Grootenhuys & Olubayo 1993; Maritim et al. 1989; Young, Grootenhuys & Irvin 1985).

It is believed that *T. annulata* evolved in the Asiatic water buffalo and this species is considered the natural host of the parasite. Infection does not typically cause clinical signs in water buffalo (Robinson 1982). *T. annulata* is maintained in nature by a cattle-tick-cattle cycle and has a seasonal occurrence, with most cases in summer months. Yaks are considered highly susceptible

to infection with severe clinical signs and high fatality rates (Pipano & Shkap 2004). Other species of wildlife are not considered susceptible (Robinson 1982). Reports of *T. annulata* in the zoo bovid species covered in this policy were not located.

The incubation period for *T. parva* is generally 15 d but may range 8–25 days (Lawrence, Perry & Williamson 2004a) and for *T. annulata* is 9–25 days (Pipano & Shkap 2004).

A variety of species of *Theileria* other than *T. parva* and *T. annulata* (some previously termed *Cytauxzoon* spp.) have been implicated in clinical disease and fatalities in African bovidae including sable, roan, greater kudu (*Tragelaphus strepsiceros*), grey duiker (*Sylvicapra grimmia*), tsessebe (*Damaliscus lunatus*) and eland (*Taurotragus oryx*) (Nijhof et al. 2005). Infection with these species, with no evidence of clinical disease, has also been reported in a wider range of bovidae including African buffalo (*Syncerus caffer*), klipspringer (*Oreotragus oreotragus*), reedbuck (*Redunca arundinum*), blue wildebeest (*Connonhaetes taurinus*), blesbok (*Damaliscus pygargus*) and also in cattle in southern Africa and eastern Africa (Nijhof et al. 2005; Oura et al. 2011; Thomas, Wilson & Mason 1982). *T. taurotragi* usually causes a benign infection in cattle but has been reported to cause occasional cases of clinical disease. *T. taurotragi* has been reported in eland and this host species may be important in the epidemiology of this agent. However this agent is not a nationally notifiable disease in Australia (Department of Agriculture and Water Resources 2016) and is not an OIE-listed disease (OIE 2018c).

Data on the presence of *T. parva* and *T. annulata* in semen were not located in the literature. Both organisms are fragile outside of the host and the only known means of transmission of the organisms is via ticks.

#### **Clinical signs**

East Coast fever (*T. parva*) is characterised by fever, enlarged lymph nodes, anaemia, increased respiratory rate, dyspnoea and sometimes diarrhoea. The clinical signs for other forms of *T. parva* infection are similar, however the duration of illness may be shorter and death may occur more quickly. Infection with *T. annulata* causes fever, swollen lymph nodes, anorexia, weight loss, anaemia, jaundice, abortion and often proceeds to death in susceptible individuals. The disease may present with peracute through to chronic forms (Pipano & Shkap 2004).

There is a high mortality rate for both *T. parva* and *T. annulata* in cattle, with up to 90 per cent mortality in naive cattle. In endemic areas, effects are less dramatic however infection is associated with reduced growth and productivity.

#### **Diagnosis**

Infection in cattle can be confirmed by identification of the schizonts in lymph node smears or small piroplasms in blood smears. PCR can be used to detect the pathogen and the indirect fluorescent antibody (IFA) test can be used to identify antibody, although cross-reactivity with other *Theileria* may occur (Mans, Pienaar & Latif 2015; OIE 2014g).

### **4.9.3 Current biosecurity measures**

There are no previous biosecurity measures for Theileriosis. The OIE Code recommendations for bovine theileriosis caused by *T. parva* and *T. annulata* in cattle and buffaloes includes zone or country freedom, freedom from clinical signs, testing and treatment for ticks (OIE 2018c).

#### 4.9.4 Risk review

Bovine theileriosis is not present in Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016).

The following key points are relevant to the biosecurity risk of Theileriosis in non-domestic zoo bovidae:

- *T. parva* does not occur in approved countries.
- *T. annulata* occurs in southern Europe and along the Mediterranean coast but not in other approved countries.
- Transmission of these organisms only occurs via ticks.
- Australia does not have *Hyalomma* ticks that are required for *T. annulata* transmission and spread.
- Australia has several species of *Rhipicephalus* ticks, but not *R. appendiculatus*, the primary vector. It is unclear whether Australian *Rhipicephalus* can act as competent vectors for *T. parva*.
- *T. annulata* infection has not been demonstrated in the zoo bovid species covered by this policy.
- *T. parva* infection was demonstrated in waterbuck in a single experimental study, however the rest of the literature consulted conclude that only species within the *Bovini* tribe are involved in the epidemiology of the disease.
- Theileriosis is of major economic importance.
- General zoo import conditions require physical inspection of animals for ticks and acaricide treatments.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- There is no evidence the organisms are found or transmitted in semen.

#### 4.9.5 Conclusion

Accordingly, based on the preceding information, specific risk management measures for the live zoo bovid species and their semen covered in this policy are not warranted. The risk of tick transmission is managed by pre and post arrival quarantine inspections and acaricide treatment during the pre-export quarantine period.

### 4.10 Foot-and-mouth disease

#### 4.10.1 Background

Foot-and-mouth disease (FMD) is caused by the FMD virus (FMDV), genus *Aphthovirus*, family *Picornaviridae*. It is a highly contagious viral disease that primarily affects cloven-hoofed animals. FMD is endemic in most of Africa, Asia, the Middle East and parts of South America. Much of Europe is free, as is all of North America and the Australasian region. FMD is primarily a disease of artiodactyls (even toed ungulates), but it has also been reported in Asian elephants, hedgehogs and some rodents. Clinical FMD has been reported in a wide range of even toed

wildlife species, including many species from the *Bovidae* family (Schaftenaar 2002; Thomson & Bastos 2004; Thomson, Bengis & Brown 2008; Weaver et al. 2013).

FMD is not present in Australia and is a nationally notifiable disease (Department of Agriculture 2016). FMD is classed in the EADRA as a Category 2 disease (a disease with potential to cause major national socio-economic consequences through significant international and domestic market disruptions and very severe production losses in the affected livestock industries) (Animal Health Australia 2014). Modelling performed in 2013 estimated that a small, quickly contained outbreak could directly cost upwards of five billion Australian Dollars (AUD) over ten years and a large, multi-state outbreak upwards of fifty billion AUD (Buetre et al 2013). Any vesicular hoof disease in cloven-hoofed animals should be regarded as suspicious of FMD until proven otherwise. Any report of FMD in Australia, including in a zoo setting, would have an immediate impact on the general livestock industry.

FMD is an OIE-listed disease (OIE 2018c).

#### **4.10.2 Technical information**

##### **Epidemiology**

FMD events in wild or zoo bovidae generally occur as spill over events from FMD outbreaks in domestic livestock. FMD spreads by direct contact between animals and contact with infected animal products, the airborne virus or contaminated fomites. In Kruger National Park in South Africa, FMD cycles in wild bovidae species, with no possible spill over from livestock. Outbreaks of clinical disease, particularly in impala, are regularly observed (Thomson, Bengis & Brown 2008). There are numerous reports of FMD outbreaks spilling over from domestic livestock and affecting zoo animals in Europe and Asia (Schaftenaar 2002).

The incubation period of FMD is variable, ranging from 18 hours to 21 days. Seven serotypes of FMD virus are recognised — A, O, C, SAT1, SAT2, SAT3 and Asia 1. The serotypes do not confer cross immunity to each other and each have many subtypes and variants (Mahy 2005; Teklehiorghis et al. 2014). Animals generally recover from acute infection in one to two weeks, however a carrier-state may persist in some species (Weaver et al. 2013).

The carrier-state in FMD is defined as unapparent infection with virus able to be isolated beyond 28 days post-infection. In domestic cattle and sheep the pharynx and upper oesophagus generally become persistently infected for four to five months but have lasted as long as 42 months (Animal Health Australia 2014). Following this definition, carrier-status has been documented in many species, however the majority of FMD outbreaks are due to direct contact with acutely infected animals. Despite numerous studies, there has been no documented successful infection of naïve animals with FMD following contact with those in a carrier-state (Samara & Pinto 1983; Weaver et al. 2013). Nevertheless, the risk of transmission from carrier-state animals cannot be completely ruled out.

A carrier-state of limited duration has been demonstrated in many of the bovidae species covered by this policy in a wild setting. Greater kudu have the longest duration, able to carry the virus for up to 160 days post infection. Other carrier-state examples are blue wildebeest (45 days), eland (32 days) and sable (28 days) (Ferris et al. 1989; Hedger, Condy & Golding 1972; Weaver et al. 2013). Compared to domestic stock, many of these species also tend to have high mortality rates when infected (Weaver et al. 2013).

The African buffalo (*Syncerus caffer*) is noteworthy as it may act as a maintenance source of FMD, able to carry and transmit the FMDV intermittently for months or, rarely, as long as five years (Condy et al. 1985; Dawe et al. 1994; Thomson, Bengis & Brown 2008). Contact between buffalo and cattle and/or impala is believed to be important for the transmission of FMDV in southern Africa (Dawe et al. 1994; Teklehiorghis et al. 2014; Thomson & Bastos 2004). Several studies have shown that infected impala (and other antelope) do not become long term carriers (Anderson et al. 1975; Bastos et al. 2000; Hedger, Condy & Golding 1972) but they may have a role in propagating FMD outbreaks by acting as an intermediary host species (Bastos et al. 2000; Weaver et al. 2013). Kudu and wildebeest have been implicated performing a similar role for a few outbreaks (Letshwenyo, Mapitse & Hyera 2006; Vosloo et al. 2005; Weaver et al. 2013).

Investigations after the 2010/2011 outbreak of FMD in Bulgaria suggested that wild boar and deer populations have a limited capacity to maintain and spread FMDV (Alexandrov et al. 2013), and it is considered unlikely that these species could maintain FMD infection in the absence of domestic animals (Dhollander et al. 2014; Robinson & Knight-Jones 2014). Studies in Mongolian gazelles also found that the continued circulation of FMDV in domestic livestock results in the virus entering the susceptible gazelle population and there is no evidence for the persistence of virus in the gazelle population between outbreaks (Bolortsetseg et al. 2012; Nyamsuren et al. 2006; Weaver et al. 2013).

With the exception of African buffalo, transmission by persistently infected livestock, zoo animals or wildlife has not been demonstrated (Elnekave et al. 2016; Knight-Jones et al. 2016; Weaver et al. 2013).

Semen from FMDV infected bulls can contain virus. The virus was detected in the seminal fluid of infected cattle and boars (McVicar et al. 1977). It was also isolated from the semen of 12 of 16 experimentally infected bulls for up to ten days post-infection. In another study, viral antigen was detected in bovine semen for up to 60 days post infection (Gajendragad et al. 2000). Virus was isolated from the semen and sheath wash of a wild seropositive African buffalo showing no clinical signs of FMD (Bastos et al. 1999). FMD virus may be present on the prepuce and coat of vaccinated bulls (Sellers et al. 1969; Sellers, Herniman & Gumm 1977) and may contaminate semen during ejaculation. There are no published studies investigating the FMD transmission risk of semen in bovidae species covered by this policy. However, given the overlap of susceptibility and clinical signs within species of the family Bovidae to FMD, it is assumed that the risk is similar, although undefined.

### **Clinical signs**

Clinical signs in non-domestic and zoo ruminants are similar to those seen in domestic livestock and include anorexia, pyrexia, lameness and vesicles on oral and nasal mucous membranes and feet. The disease may also present subclinically.

### **Diagnosis**

Diagnosis of FMD is by virus isolation or by the demonstration of FMD viral antigen or nucleic acid in samples of tissue or fluid. Detection of virus-specific antibody can also be used as indicators of infection. Antibodies to viral non-structural proteins can be used to differentiate natural exposure from vaccination response (OIE 2017a).

**Prevention**

The wide antigenic variation of the virus and lack of cross-protection between serotypes makes the disease difficult to control. Whilst vaccination may be useful at reducing clinical cases and slowing spread, it is not sufficient for complete protection against the disease in susceptible animals. The effectiveness and shortcomings of vaccination against FMD have been reviewed extensively by the department and many authors (Brückner & Saraiva-Vieira 2010; Paton et al. 2009; Uttenthal et al. 2010).

The most effective method for preventing FMD remains the requirement of country freedom for import of products from animals susceptible to FMD.

**4.10.3 Current biosecurity measures**

Australia's previous biosecurity measures for FMD included country freedom or zone freedom and testing. The OIE Code recommendations include country freedom or zone freedom and testing (OIE 2016b).

**4.10.4 Risk review**

FMD is not present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of FMD in zoo bovidae:

- FMD is an OIE-listed disease and is nationally notifiable in Australia.
- FMD is the greatest animal disease of economic concern to Australia.
- It is a highly infectious disease that causes widespread morbidity and, in some species of bovidae, high mortality.
- Virus is typically transmitted by close contact from acutely infected animals, or contact with infected animal products, airborne virus or contaminated fomites.
- FMD events in wild or zoo bovidae generally occur as spill over events from FMD outbreaks in domestic livestock.
- A short duration carrier-state exists in many wild and zoo bovidae species and several species may have roles in propagating FMD outbreaks as intermediate hosts.
- Other than African buffalo, transmission from persistently infected cattle or wildlife has not been demonstrated.
- There is evidence that FMD virus can be transmitted via semen.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

**4.10.5 Conclusion**

Accordingly, based on the preceding information and the recommendations in the OIE Code (OIE 2016b) risk management measures continue to be warranted.

Australia's proposed biosecurity measures for FMD for zoo bovids are:

- For 270 days immediately before export the animal was continuously resident in a country on the department's FMD-free approved country list.

Australian's proposed biosecurity measures for FMD for zoo bovid semen are:

- For 90 days immediately before collection, the animals from which the semen for export was collected resided in a country on the department's FMD-free approved country list.

## 4.11 Haemorrhagic septicaemia

### 4.11.1 Background

Haemorrhagic septicaemia (HS) is a highly fatal disease of cattle and water buffalo caused by the B2 and E2 serotypes (Carter and Heddleston system; equivalent to 6:B and 6:E in Namioka-carter system) of the bacterium *Pasteurella multocida*, a gram-negative anaerobic bacterium belonging to family *Pasteurellaceae*. Outbreaks are associated with high morbidity and mortality rates. HS is endemic in tropical and subtropical regions including South-East Asia, India, the Middle East, regions of Africa, and southern and central Europe (OIE 2016b; Völker et al. 2014). Outbreaks of septicaemic pasteurellosis in wild ruminants caused by the other *P. multocida* serotypes have been reported in countries free from HS, including Australia.

HS is an OIE-listed disease (OIE 2018c). HS is not present in Australia and is a nationally notifiable disease (Animal Health Australia 2016; Department of Agriculture and Water Resources 2016). HS is a category 4 disease in the EADRA with the potential to cause severe economic and production losses.

### 4.11.2 Technical information

#### Epidemiology

The Asian form of HS is caused by serotype B2 while the African form is caused by serotype E2. Outbreaks occur sporadically and tend to be associated with high levels of physiological stress in hosts. *P. multocida* is shed in respiratory aerosols, saliva, urine, faeces and milk by both active carriers and clinical cases (Annas et al. 2014a). Transmission is through direct contact with infected animals, or indirect methods such as exposure to infected fomites or aerosols. Contaminated feed and vectors such as ticks and biting insects have also been implicated in transmission (Radostits et al. 2007a). Close contact between animals is generally required for transmission by ingestion or inhalation. HS-causing strains of *P. multocida* have been identified in many tissues of clinically affected animals (Annas et al. 2014b; Bastianello & Jonker 1981; Khin, Zamri-Saad & Noordin 2010; Lane et al. 1992) and in the respiratory, gastrointestinal and urinary tracts of carrier animals (Annas et al. 2014a). Moist environmental conditions may prolong survival and the bacteria may persist in animal carcasses for a few days (De Alwis 1999).

HS outbreaks in Asia usually occur in areas with a high seasonal rainfall, particularly in marshy zones or along river deltas (Bastianello & Henton 2004). Epidemic outbreaks have been associated with high morbidity and mortality rates (OIE 2012b; Shivachandra, Viswas & Kumar 2011). Rare and sporadic outbreaks of HS have been reported in the United States, however since 2010 HS has been considered absent from the United States and is nationally notifiable in that country (OIE 2018c; USDA:APHIS 2016). Eradication of HS is generally not considered feasible from Asian countries (Benkirane & De Alwis 2002).

HS has occurred in zoo populations of deer, zebra, eland and *Bos* spp. with obvious clinical signs including death, but overall the disease is rarely reported in zoos (Eriksen et al. 1999; Happy et al. 2013; Okoh 1980; Vellayan & Jeferry 2014).

The incubation period is generally two to five days but may be as short as a few hours (Spickler 2009a).

Carrier status is an important epidemiological feature of HS. Carriers are animals that have survived a previous outbreak and tend to harbour the bacteria in tissues associated with the respiratory tract (Annas et al. 2014a; De Alwis 1999). Not all infected animals become carriers and the number of carriers declines over the months following an outbreak; up to 20 per cent of outbreak survivors are reported to become carriers, though by 6 months the carrier rate is five per cent or less (Spickler 2009a). Maximum duration of the carrier state is not known but up to 12 months has been reported (De Alwis 1999; Spickler 2009a). Carriers may be latent or active. Latent carriers harbour the bacteria in the tonsils but are not known to shed (Annas et al. 2014a; Bastianello & Henton 2004). Active carriers harbour the bacteria in the tonsils and nasopharynx and may actively secrete the organism for up to six weeks before returning to a latent state. The bacteria localises in the tonsillar crypts, which significantly inhibits clearance of the carrier state (Bastianello & Henton 2004). Physiological stress is another important epidemiological feature (for example, concurrent disease, poor nutrition, high stocking density), and is known to induce latent carriers to become active and shed the bacteria (Moustafa et al. 2015). Explosive outbreaks may occur in herds that have minimal immunity.

Water buffalo are considered much more susceptible than cattle, with a higher mortality rate (Bastianello & Henton 2004). Young animals are more susceptible than adults (Benkirane & De Alwis 2002). Fatal epidemics of HS are reported in Kazakhstan in free-ranging saiga antelope (*Saiga tatarica*), resulting in deaths of tens of thousands of individuals within days. A recent epidemic occurred in the northern summer of 2015. The herds were suffering the stress of the imminent seasonal calving period and particularly adverse weather events occurred immediately before the die-off began (Zhusypbekovich et al. 2016).

HS has been described in wild mammals, and was either reported or suspected, in African buffalo, bison, pigs, goats, sheep, eland, yak and saiga antelope (Okoh 1980; Rimler 1992). Outbreaks of B2 HS-causing strains in wild bison occurred in the United States in 1992, 1912 and 1965 (Corn & Nettles 2001). Other outbreaks of septicaemic pasteurellosis in bison and cattle in the United States have been attributed to non HS-causing strains of *P. multocida*. There is no evidence of transmission between bison and domestic ruminants. Of the non-domestic bovidae, HS has only been confirmed in bison, water buffalo, saiga antelope and yak (De Alwis 1999; Joshi et al. 1997; OIE 2015d). HS has also been reported in a wide range of non bovidae species including camel, cervids, donkeys, horses, pigs, elephants and poultry, although some of these may have been due to B1 or B3,4 serotypes (Bastianello & Henton 2004; De Alwis 1999).

Williams (2003) did not locate any records of *P. multocida* being present in or transmitted by semen. No evidence of presence or transmission in semen was located in the literature.

### **Clinical signs**

Infection may be peracute, acute or subacute. Variable clinical signs are associated with HS, ranging from pyrexia, respiratory distress, nasal discharge and dependent oedema in the submandibular or brisket regions, to recumbency and sudden death. Peracute infection is

characterised by sudden death, while acute and subacute infections are characterised by fever, anorexia, depression, profuse salivation and nasal discharge (Chung et al. 2015). Clinical signs may last as little as a day or as long as 10 days before the animal dies. Chronic clinical disease is not known to occur; animals either die from the disease or survive to become active or latent carriers (Spickler 2009a).

### Diagnosis

Diagnosis of HS is based on clinical signs, gross lesions, and patterns of morbidity and mortality. Confirmation requires isolation and characterisation of the pathogen using conventional and molecular techniques. True septicaemia in HS only occurs at the terminal stage of the disease. *P. multocida* may not be present in the blood of animals in the early stages of the disease, and is not consistently present in the nasal secretions or body fluids of sick animals (OIE 2012b). Serological tests are most commonly used to evaluate antibody response to vaccination, although carriers are reported to have high antibody levels (Bastianello & Henton 2004). Highly sensitive and specific surveillance results can be achieved using the indirect-ELISA with capsular antigens on sera or nasopharyngeal swabs (Afzal, Muneer & Akhtar 1992; Dawkins et al. 1990; Dziva et al. 2008; El-Jakee et al. 2016; Kharb 2015; Takada-Iwao et al. 2007).

### Prevention

Antibiotic treatment may be effective if started in the earliest phase of disease. When administered at a herd level, vaccination is effective at reducing the incidence of disease (Zamri-Saad & Annas 2016). Differentiating Infected from Vaccinated Animals (DIVA) markers and assays are in development for HS vaccines (Qureshi 2014; Qureshi & Saxena 2017).

Despite extensive work in cattle and bison, currently available vaccines have variable efficacy and duration (Ahrar et al. 2011; Qureshi & Saxena 2017; Tabatabaei et al. 2007). Live attenuated vaccines, when delivered as a two-dose regime, via the intramuscular route provide significant protection for up to a year (Ahmad et al. 2014; Hodgson et al. 2005; Tabatabaei et al. 2007). Recombinant vaccines may produce a stronger immunological response than conventional vaccines (Qureshi 2014). Live attenuated intranasal vaccines for B2 & E2 types are available and effective but may result in auto-vaccination of other in-contact animals (Khin et al. 2009; Rafidah et al. 2012). Sporadic outbreaks and virulence in young animals has been associated with the intranasal vaccine based off B3,4 (Zamri-Saad & Annas 2016).

Although calves of domestic cattle vaccinated twice with a live attenuated product displayed no clinical signs, post-mortem examination a week later revealed bacteria in upper respiratory tissues and lymph nodes (Dagleish et al. 2007). Other studies show that water buffalo vaccinated and surviving challenge with minimal signs also had varying degrees of pathology in the respiratory tissues (Rafidah et al. 2012). A limitation of vaccination-challenge studies is that they deliver the challenge dose via injection, circumventing the role of upper respiratory defences against the initial incursion, and also do not always incorporate the involvement of stress in promoting infection. The significance of these findings as it relates to a potential carrier status despite vaccination is unclear.

Vaccination of bison against HS results in a protective antibody response and may have played a role in halting an epidemic (Heddleston & Wessman 1973). Some vaccination regimes have been shown to be protective for young water buffalo (Saleem et al. 2014; Shah, Shah & de Graaf 1997). Vaccination is likely to confer similar protection in other non-domestic bovidae species given the susceptibility of buffalo and young animals to natural disease.

### 4.11.3 Current biosecurity measures

Australia's current biosecurity measures for HS in live elephants and fresh beef products include country freedom or premises freedom and vaccination. The OIE Code recommendations for cattle and buffaloes include country or zone freedom, testing and vaccination (OIE 2016b).

### 4.11.4 Risk review

HS is present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of HS in zoo bovidae:

- HS is endemic in tropical and subtropical regions including South-East Asia, India, the Middle East, regions of Africa, and southern and central Europe.
- HS most commonly affects cattle and water buffalo but may have a wide host range.
- Several species of bovids (primarily *Bos* and *Bubalis* spp.) are considered highly susceptible to HS and outbreaks have been reported in wild bovids.
- Transmission may occur through several methods including close exposure to infected animals, and exposure to infected fomites, feed, vectors and aerosols, however close contact between animals is generally required for transmission by ingestion or inhalation.
- HS infection is acute with high mortality and morbidity rates. Naïve herds experience significant mortality. An uncontrolled outbreak in Australia would cause severe production losses in the cattle industry and loss of export markets.
- A carrier state is known to exist and may persist for up to a year. Carriers that undergo sufficient stress become active shedders. The latent carrier state persists in spite of antibiotic and other treatment.
- Vaccination is known to be effective in several bovid species, as well as elephants, and is likely to confer similar protection in other bovidae species.
- There is no evidence that semen poses a risk for transmission of HS between hosts.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

### 4.11.5 Conclusion

Accordingly and based on the preceding information, risk management measures continue to be warranted. Specific biosecurity measures for semen are not warranted.

Australia's biosecurity measures for HS for zoo bovidae are:

#### Option ONE

- For 270 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of haemorrhagic septicaemia occurred during the previous 12 months before export and the disease is compulsorily notifiable.

## Option TWO

- For 270 days immediately before export the animal did not reside on any premises where clinical, epidemiological or other evidence of haemorrhagic septicaemia occurred during the previous 12 months before export and the disease is compulsorily notifiable.

AND

- Between 90 and 180 days immediately before export the animal was vaccinated against haemorrhagic septicaemia with an approved vaccine.

## 4.12 Heartwater

### 4.12.1 Background

Heartwater is a tick-borne, rickettsial disease of ruminants caused by *Ehrlichia ruminantium* (formerly *Cowdria ruminantium*), an aerobic, gram-negative obligate intracellular bacterium. Its distribution is confined to that of its *Amblyomma* tick vectors, with the disease currently restricted to Africa, Madagascar and some Indian and Atlantic Ocean and Caribbean islands (OIE 2015e). It is a disease of significant economic importance. In endemic areas, diagnosis of heartwater is often assumed, rather than confirmed by testing and formally reported, leading to underreporting of the disease (Allsopp 2010).

Heartwater is a multiple species OIE-listed disease (OIE 2018c). It is absent from Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016). Heartwater is a Category 4 disease in the EADRA. An outbreak of the disease would cause serious production losses and loss of export markets.

### 4.12.2 Technical information

#### Epidemiology

The primary method of transmission is via ticks and the disease can only exist where the vectors are present (Kasari et al. 2010). *Amblyomma variegatum* and *A. hebraeum* are the most important species involved in transmission. Field and experimental conditions have demonstrated disease transmission by several other *Amblyomma* species (Kasari et al. 2010). Three tick species native to the United States are capable vectors (Deem 2008). Several Australian species of *Amblyomma* are potential vectors (Animal Health Australia 2013).

Vectors of heartwater are three-host ticks and transmission is transstadial, although transovarial transmission is also reported (Bezuidenhout 1987). Ticks become infected within two to four days of feeding on acutely ill or subclinically infected hosts and retain their infectivity for life (Allsopp, Bezuidenhout & Prozesky 2004; Bath, van Wyk & Pettey 2005). Infected larvae or nymphs also remain infective for life (Allsopp 2010). Transmission of *E. ruminantium* to vertebrate hosts takes several days of feeding and only a portion of infected ticks will transmit the infection (Kasari et al. 2010). Translocation of infected *Amblyomma* spp. by migrating birds (e.g. African cattle egret, *Ardeola ibis ibis*) is thought to be possible, if not a major pathway of spread (Allsopp 2010).

Vertical transmission has been demonstrated from domestic cattle to their calves, thought to be due to ingestion of the organism within infected cells in colostrum (Allsopp 2010). This method of infection may occur in other ruminants, including non-domestic bovidae. Iatrogenic transmission is also possible but significant fomite transmission is unlikely (Spickler 2015c).

Many species of African ruminants have been shown to be susceptible to infection in the wild or in a captive zoo environment, including African buffalo (*Syncerus caffer*), black wildebeest (*Connochaetes gnou*), blesbok (*Damaliscus pygargus*), blue wildebeest (*Connochaetes taurinus*), eland (*Taurotragus oryx*), giraffe (*Giraffa camelopardalis*), greater kudu (*Tragelaphus strepsiceros*), sable antelope (*Hippotragus niger*), lechwe (*Kobus leche kafuensis*), sitatunga (*Tragelaphus spekii*), springbok (*Antidorcas marsupialis*) and steenbok (*Raphicerus campestris*) (Deem 2008; Peter, Burridge & Mahan 2002). Clinical disease has been reported in blesbok, eland, springbok, black wildebeest and African buffalo. Experimentally infected eland, giraffe, kudu and wildebeest have successfully transferred their infection to susceptible cattle via the *A. hebraeum* vector (Peter et al. 1998). Non-African bovid species have also been shown to be susceptible to infection with *E. ruminatum*. Heartwater probably evolved in southern Africa and it is likely that wild African ruminants are the original reservoir of the disease. The most important reservoir species in southern Africa are considered to be blesbok, black wildebeest, African buffalo and eland (Allsopp 2010). The disease is however able to maintain itself in the absence of a wild ruminant reservoir (e.g. in domestic stock in Madagascar). A role for non-ruminant vertebrates (e.g. leopard tortoise, *Geochelone pardalis*) as disease reservoirs has been proposed.

The incubation period varies from 9 to 29 days but is commonly around two weeks. Recovered animals, including non-domestic bovidae species, may remain carriers of the infection for extended periods of time (Kock et al. 1995). Clinically healthy ruminants may continue to be infective for a year in cattle and eleven months in goats (Allsopp, Bezuidenhout & Prozesky 2004; Andrew & Norval 1989). A carrier state of up to a year has also been demonstrated, in natural and experiment situations, in eight of the aforementioned wild ruminant species (Bezuidenhout 1987; Peter et al. 1999; Peter, Burridge & Mahan 2002; Spickler 2015c).

Williams (2003) did not locate any data on survivability in any animal-derived commodity, including germplasm. There is no clear evidence this obligate intracellular organism is transmissible in germplasm. *E. ruminantium* is very fragile and does not survive outside a living host for more than a few hours at room temperature (Williams 2003).

### Clinical signs

Heartwater occurs as an acute or peracute illness. The acute form is characterised by pyrexia, depression, inappetance and neurological signs. Mortality rates are high once clinical signs develop but vary significantly (<10 to 90 per cent) depending on species, breed and previous exposures (Spickler 2015c). A subacute form is characterized by pyrexia for 10 or more days and less pronounced clinical signs, though death is still possible.

The clinically inapparent form of heartwater may be difficult to detect with only a transient pyrexia, apathy and slight tachypnoea before recovery. Many wild ruminants can be asymptomatic carriers for heartwater (Kock et al. 1995).

### Diagnosis

Diagnosis of heartwater in live animals may be challenging. Clinical signs are not pathognomonic for the disease and its progression is rapid. Serology has limited diagnostic usefulness, due to high levels of cross-reactivity with other *Ehrlichia* spp. and none of these tests have been validated for wildlife (Deem 1998; Deem 2008; Peter, Burridge & Mahan 2002). Prior to the development of molecular techniques, diagnosis typically relied on xenodiagnosis and pathology of brain and lung tissue.

The pCS20 PCR assay is considered the most sensitive, reliable and best characterised test for *E. ruminantium* in ticks and blood from animals with clinical signs (Allsopp 2010; Deem 2008; OIE 2016c; Thomas 2016). Of molecular techniques, the only test shown able to detect subclinical *E. ruminantium* infection is the pCS20 PCR assay (Deem 2008), though the detection of carrier animals remains difficult and insensitive due to fluctuating, low levels of rickettsial organisms in the blood. Some PCR techniques become more sensitive when used on ticks that are allowed to feed on suspected carriers (OIE 2016c; Thomas 2016). If PCR is to be used to determine the infection status of asymptomatic animals it is advised that the blood of such animals is repeatedly tested by the pCS20 PCR assay, albeit still with the above caveats (OIE 2016c).

### **Treatment**

Tetracycline antimicrobials can be used to treat heartwater, especially during outbreaks in endemic countries, however to be effective treatment must start during the very early stages of infection.

### **Prevention**

Spread of heartwater may occur from the transport of infected ticks, infected animals, and subclinical carrier animals. Heartwater requires capable *Amblyomma* spp. ticks to exist in a region in order to persist. Therefore careful selection of disease free animals is required prior to transport to a region where capable *Amblyomma* spp. exist (Kasari et al. 2010). Inspection of imported animals for ticks, regular acaricide treatments and sourcing animals from heartwater free areas are useful strategies.

Within endemic areas, control focuses on four methods: tick control, farming with resistant stock, metaphylactic administration of antibiotics and vaccination (OIE 2016c). Eradication of capable *Amblyomma* spp. from an area is costly and difficult, if not impossible. An effective vaccine is yet to be developed.

## **4.12.3 Current biosecurity measures**

Australia's previous biosecurity measures for heartwater includes country freedom. The OIE Code recommendations include diagnostic testing and treatment for ticks for domestic and wild ruminants (OIE 2014).

## **4.12.4 Risk review**

Heartwater is not present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of heartwater in zoo bovidae:

- Heartwater is endemic in much of Africa and is likely underreported. It is also present in the Caribbean, Madagascar and some islands in the Indian and Atlantic Ocean.
- Heartwater is absent from approved countries.
- A carrier state of *E. ruminantium* exists in domestic and wild ruminants and may last up to a year. African ruminants are likely to be the original reservoir of the disease.
- *E. ruminantium* is transmitted to mammals from ticks of the genus *Amblyomma*. The organism can only persist where tick vectors are present.

- Vertical transmission is possible from dam to calf, probably via ingestion of colostrum, and iatrogenic spread.
- *E. ruminantium* is difficult, if not impossible, to eradicate once established in *Amblyomma* tick vectors, which themselves are unlikely to be eradicated.
- Australia has *Amblyomma* spp. that may be capable vectors.
- Heartwater is a disease of significant morbidity and economic consequence.
- Ante-mortem diagnostic techniques for identifying the carrier state of heartwater are not reliable. PCR may be used reliably to detect the organism in animals with clinical signs.
- There is no reliable vaccine.
- There is no evidence of transmission through germplasm.
- General zoo import conditions require physical inspection of animals for ticks and acaricide treatments.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs, are under veterinary supervision, and have physical barriers against sources of ticks (woodlands, etc.).

#### 4.12.5 Conclusion

Accordingly, based on the preceding information specific risk management measures for heartwater are warranted.

Australia's proposed biosecurity measures for heartwater for zoo bovids are:

- For 24 months prior to export the animal (and its dam, if the animal for export is under 12 months of age) has not resided in any country where clinical, epidemiological or other evidence of heartwater has occurred in any species during the previous 2 years and the disease is compulsorily notifiable.

AND

- Within two working days of entry to the pre-export quarantine facility the animal was examined thoroughly for ticks, found free of ticks and treated under the supervision of the Official Veterinarian, with a long acting parasiticide effective against ticks.

Specific risk management measures for zoo bovidae semen are not warranted.

## 4.13 Infectious bovine rhinotracheitis

### 4.13.1 Background

Infectious bovine rhinotracheitis (IBR) is a viral disease of cattle caused by bovine herpesvirus 1 (BoHV-1) from the genus *Varicellovirus*, subfamily *Alphaherpesvirinae*, family *Herpesviridae* (ref). It is a pathogen primarily of domestic cattle (*Bos* spp.). BoHV-1 occurs worldwide although several countries have eradicated the infection, including Switzerland, Denmark, Finland, Sweden and Austria. Evidence indicates that only low virulence strains of subtype 1.2b are present in Australia (Animal Health Australia 2016; Allan, Dennett & Johnson 1975; Gu & Kirkland 2003; Young 1993). Most infections in Australia are subclinical and pass unnoticed (Beveridge 1986).

IBR is an OIE listed disease (OIE 2018c). It is not a notifiable disease in Australia.

#### 4.13.2 Technical information

##### Epidemiology

BoHV-1 causes a complex of disease syndromes including infectious bovine rhinotracheitis (IBR) and infectious pustular vulvovaginitis (IPV). Other syndromes include eye infections, abortions, and a generalized infection of newborn calves. The disease is most commonly seen in cattle housed in close proximity, such as in feedlots (Babiuk, Van Drunen Littel-Van Den Hurk & Tikoo 2004).

BoHV-1 has three subtypes, BoHV-1.1, BoHV-1.2a and BoHV-1.2b. BoHV-1.1 is commonly associated with respiratory disease (IBR) and BoHV1.2a and BoHV1.2b, with genital disease (IPV) (OIE 2017b). Over the past 20 years, BoHV1.1 has been responsible for severe IBR outbreaks in Europe and North America (Raaperi, Orro & Viltrop 2014). Subtype 1.2b is less virulent than 1.1 and typically causes only subclinical infection or very mild clinical signs (Muylkens et al. 2007).

The incubation period is usually 2 to 4 days but may be as long as 20 days (Babiuk, Van Drunen Littel-Van Den Hurk & Tikoo 2004). The OIE Code prescribes a 21 day incubation period for BoHV-1 in cattle (OIE 2017b). Mortality is low but significant production losses can occur from morbidity (Muylkens et al. 2007).

BoHV-1 is excreted from vaginal and nasal secretions of infected cattle. Respiratory infections are generally acquired via aerosol, and genital infections are generally spread venerally. Direct nose-to-nose contact is the common mode of transmission of BoHV-1 (Muylkens et al. 2007). Airborne transmission by aerosol has been demonstrated experimentally over short distances (Mars et al. 2000). The virus is highly contagious and introduction of a new, infected animal to a herd can result in significant disease outbreaks, with up to 100 per cent of animals in a herd becoming infected with BoHV-1 (Hage et al. 1996; Radostits et al. 2007a). Once infected, animals maintain a lifelong latent infection. Shedding of the virus is intermittent and generally associated with periods of stress such as disease, transportation, cold or overcrowding (Babiuk, Van Drunen Littel-Van Den Hurk & Tikoo 2004).

The virus is sufficiently stable to persist in the environment for several days. It may spread via fomite contamination of feed and equipment during this time (Babiuk, Van Drunen Littel-Van Den Hurk & Tikoo 2004).

BoHV-1 infects domestic and wild cattle of all ages. Other ruminants such as goats, deer, water buffalo, Cape buffalo and wildebeest are susceptible to infection and may be potential reservoirs of the virus (Castro 2001; Hedger & Hamblin 1978; Karstad et al. 1974; St George & Philpott 1972). Wild ruminants seldom display clinical signs associated with BoHV-1 infection. BoHV-1 infection in water buffalo may cause both respiratory and genital disease (St George & Philpott 1972).

BoHV-1 belongs to the extensive subfamily of *Alphaherpesvirinae* that have a relatively wide host range (Muylkens et al. 2007). Antibodies to BoHV-1 have been found in a wide range of wild African bovidae including buffalo, kudu, eland, waterbuck, lechwe, kob, reedbuck, bushbuck, nyala, sable, roan antelope, topi, hartebeest, wildebeest and impala (Anderson & Rowe 1998b; Hedger & Hamblin 1978; Rampton & Jessett 1976). The high seroprevalence found in Cape

buffalo suggests this species may be an important reservoir of the virus in the wild. No clinical disease was seen in over 1000 animals sampled, and BoHV-1 was not able to be isolated from wild bovidae species (Hedger & Hamblin 1978). Serological evidence of exposure to BoHV-1 has not been demonstrated in wild bovidae in Europe (Gaffuri et al. 2006). Some of these earlier serology-based reports may represent BoHV-2 or other species-specific herpesviruses (Gu & Kirkland 2003; Kálmán & Egyed 2005; Raaperi, Orro & Viltrop 2014). BoHV-1 has been isolated from wildebeest, pronghorn, mink and ferrets (*Mustela putorius furo*) without clinical disease (Bhat, Manickam & Kumanan 1997; OGTR 2005). BoHV-1 has been detected, by PCR, in several sheep and deer species (Kálmán & Egyed 2005).

Serological evidence of BoHV-1 infection has been reported in a variety of North American ungulates, including a range of deer species, pronghorn, American bison (*Bison bison*) and Peninsular bighorn sheep (*Ovis canadensis cremnobates*) (Clark et al. 1993; Taylor et al. 1997). Infection may result in clinical disease in *Ovis canadensis cremnobates* (Clark et al. 1993). BoHV-1 is rarely reported in zoo bovids (Citino 2003; Doyle & Heuschele 1983b; Mahmoud 2015; Probst 2008; Yeşilbağ, Alpay & Karakuzulu 2011).

BoVH-1 is the most common viral pathogen of bull semen. Infection can be transmitted via semen, excretions and secretions, and foetal fluids, and indirectly by personnel (Autrup & Bitsch 1977; Babiuk, Van Drunen Littel-Van Den Hurk & Tikoo 2004; Deas & Johnston 1973; Raaperi, Orro & Viltrop 2014). Not all bulls with respiratory BoHV-1 infections have infected semen, and some may only sporadically shed BoHV-1 in semen (De Gee, Wagter & Hage 1996; Van Oirschot 1995). Bulls shedding BoHV-1 in semen may be seronegative (Hage et al. 1998). Semen can be contaminated, by a primary preputial infection, prior to antibody response (Deka, Maiti & Oberoi 2005).

### **Clinical signs**

Most infections in domestic bovids are subclinical. Clinical cases last 5 to 20 days. Signs include pustular vulvovaginitis, balanoposthitis, purulent nasal discharge, muzzle hyperaemia, conjunctivitis and sometimes coughing, fever, depression, inappetance, abortions, and reduced milk yield (Muylkens et al. 2007). Animals may develop secondary bacterial infections that lead to a more severe respiratory disease presentation. Abortion is a feature of BoHV-1.2a infection. BoHV-1.2b can cause IBR and IPV but it is relatively benign and is not foetopathic or abortogenic (Van Oirschot 1995).

Clinical disease as a result of BoHV-1 infection is generally not recognised in African ruminants, however deliberate immunosuppression of temporarily captive wildebeest resulted in clinical vulvovaginitis and seroconversion to BoHV-1 (Karstad et al. 1974).

### **Diagnosis**

Diagnosis is via nasal or genital swabs (taken during the acute phase of infection) using virus isolation or real time PCR. Serological tests such as VNT and ELISA are used for antibody detection and prescribed by the OIE for international trade, but do not always identify latently infected animals. Detection of latently infected animals relies on post-mortem detection of the virus in tissues such as the trigeminal ganglia (Puentes et al. 2016). Antibody-positive animals should be classified as infected with BoHV-1, unless the serological response was induced by vaccination with an inactivated vaccine or due to the presence of colostral antibodies (OIE 2017b). Differentiation between subtypes of BHV-1 may be accomplished by PCR or other laboratory techniques.

Semen can be tested by a virus isolation test or PCR. PCR is generally more sensitive. Because semen of infected bulls is infected sporadically or has very low titres of BoHV, it is essential that at least three straws from each batch be transported frozen or chilled to the laboratory and tested (OIE 2017b). Because the seminal fluid contains enzymes and other factors that are toxic to the cells and inhibit viral replication, it is necessary to treat the semen to remove the toxic factors prior to testing (OIE 2017b; Van Oirschot 1995).

### **Prevention**

Live attenuated and killed vaccines are available to provide protection against clinical disease and reduce viral shedding after infection (Platt et al, 2006; Xue et al, 2010), but do not completely prevent infection (OIE 2017b). Modified live vaccines containing BoHV-1 are widely used in breeder cattle in the United States and Canada. However, BoHV-1 strains, including vaccine strains, continue to be isolated from diseased animals or foetuses after vaccination of the dam, indicating latent infection (Fulton et al 2015).

The virus is sensitive to many chemical disinfectants, especially solvents (Straub 1990).

### **4.13.3 Current biosecurity measures**

There are no previous biosecurity measures for BoHV-1 for live animals. The OIE Code recommendations for cattle include herd freedom, testing and vaccination. Biosecurity measures for bull semen include herd freedom certified to OIE standards, serological testing, and molecular testing.

### **4.13.4 Risk review**

BoHV-1.1 and BoHV-1.2a are present in approved countries. Only BoHV-1.2b is present in Australia.

The following key points are relevant to the biosecurity risk of IBR in non-domestic zoo bovidae:

- BoHV-1 is a disease of low mortality but can cause serious production losses. BoHV-1.2b infections generally pass unnoticed. BoHV-1.1 and BoHV-1.2a can cause serious outbreaks.
- BoHV-1 has been eradicated in several countries.
- The primary modes of transmission are aerosol or venereal routes. Aerosol transmission is generally limited to short distances.
- After acute infection an animal becomes a lifelong, latent carrier of BoHV-1. Stress may reactivate a latent carrier to actively shed virus.
- BoHV-1 is primarily a disease of domestic cattle. A range of non-domestic bovidae species may be infected, though it is not known to be a common disease in zoos in approved countries.
- BoHV-1 is rarely reported in zoo bovids.
- Differentiation between subtypes requires isolation of viral DNA and subsequent PCR or other laboratory techniques.
- BoHV-1 can infect, and be transmitted via bovine semen.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.

- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.13.5 Conclusion

Accordingly and based on the preceding information, specific risk management measures for the live zoo bovid species and their semen are warranted.

Australia's biosecurity measures for BoHV-1 for zoo bovids are:

##### Option ONE

- For 180 days immediately before export the animal was continuously resident on premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- In the 30 days immediately before export the animal was tested for BoHV-1 twice at an interval of no less than 21 days, on separate blood samples drawn during this time. The test was of a type approved by the department. The test results were negative.

##### Option TWO

- For 180 days immediately before export the animals was continuously resident on any premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- For 180 days immediately before export the animals for export were part of a collection subject to a documented BoHV-1 screening program. The screening program must include:
  - Diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department.
  - The collection must have been a 'closed-herd'<sup>4</sup> during that time.
  - The collection must contain at least four zoo bovids.

AND

- During the 180 days immediately before export the animal was tested as part of the screening program.

Australia's biosecurity measures for BOHV-1 for zoo bovid semen are:

##### Option ONE

- The semen was tested for BoHV-1 by RT-PCR in accordance with the OIE prescribed preparation and testing regime for semen (OIE 2017b).

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<sup>4</sup> A 'closed-herd' in this context means that new animals susceptible to BoHV-1 were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

## Option TWO

- For 180 days immediately before the semen collection period the donor animal was continuously resident on any premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- For 180 days immediately before the semen collection period the donor animal was part of a zoo collection subject to a documented BoHV-1 screening program. The screening program must include:
  - Diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department.
  - The collection must have been a 'closed-herd' during that time.
  - The collection must contain at least four zoo bovids.

AND

- The donor animal was tested by the screening program during this time.

## 4.14 Lumpy skin disease

### 4.14.1 Background

Lumpy skin disease (LSD) is caused by lumpy skin disease virus (LSDV) in the genus *Capripoxvirus*, family Poxviridae. The three members of the *Capripoxvirus* (LSDV, sheep pox virus and goat pox virus) are closely related and clinically and morphologically indistinguishable.

LSD is considered endemic in many African and Middle Eastern countries (OIE 2017d). In late 2015 outbreaks of LSD occurred in Greece and in 2016 the disease continued to spread through the Eastern European countries of Bulgaria, Republic of Macedonia, Serbia, Kosovo, Albania and Montenegro. The disease has historically demonstrated a high risk of becoming endemic in countries it spreads to (FAO 2017).

LSD is considered a transboundary disease. Patterns of LSD spread are erratic and difficult to predict, even if movement control and quarantine processes have been enacted. The 1989 and 2006 outbreaks of LSD in Israel are suggested to have started from windborne translocation of vectors from distant Egyptian ports (Klausner 2015).

LSD is an OIE-listed disease (OIE 2018c). LSD is not present in Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016). LSD is a Category 3 disease under the EADRA and an outbreak would result in severe consequences to Australia.

### 4.14.2 Technical information

#### Epidemiology

Capripoxviruses are fairly host-specific with LSDV primarily causing disease in cattle and water buffalo. Other species such as oryx, impala and giraffe are able to be infected experimentally but are not known to have a significant role during natural infection (FAO 2017).

Morbidity rates vary dramatically between outbreaks, reported as being between 5 to 45 per cent and sometimes approaching 100 per cent (Tuppurainen et al. 2015b). Mortality is generally

low (1-3 per cent) but sometimes reaches 40 per cent (Coetzer 2004). Variations between host immune status and mechanical arthropod vector abundance are reasons proposed for such variation. Animals affected by Capripoxviruses eventually clear the infection, establish lifelong immunity and do not become carriers. Morbidity from LSD has a significant economic impact, causing abortions and temporary or permanent infertility, reduced growth of animals, a marked reduction in milk production and permanent damage of skins and hides as a result of deep scars (Green 1959; OIE 2017d; Tuppurainen et al. 2015b).

Many aspects of LSDV transmission pathways are unclear. Studies have revealed that the mode of LSD spread has links to infected windborne vectors and the movement (generally illegal, from an affected country) of infected animals or products (Klausner, Fattal & Klement 2015) (Abutarbush et al. 2016; Sevik & Dogan 2016; Tasioudi et al. 2016). Vector-borne transmission of LSDV is known to be mechanical rather than biological (Klausner, Fattal & Klement 2015), however the range of haematophagous insect vectors is not fully known. *Culicoides punctatus* has recently been suggested to have a role in transmission (Sevik & Dogan 2016). The role of ticks in trans-ovarial, mechanical or intra-stadial, and trans-stadial transmission is increasingly recognised (Lubinga et al. 2014; Lubinga et al. 2013; Tuppurainen et al. 2015a; Tuppurainen et al. 2013).

Transmission from infected milk or teat skin lesions to suckling calves is rare, but possible (Weiss 1968). Transmission via a shared drinking trough has been demonstrated in cattle (Haig 1957), but other attempts to demonstrate direct transmission, by housing uninfected with infected animals, were not successful (Carn & Kitching 1995). Direct transmission is considered an inefficient method of transfer of LSDV (Carn & Kitching 1995; Weiss 1968). Cattle may be viraemic, and capable of transmitting virus, in the absence of skin nodules or other clinical signs.

Virus can be found in salivary, nasal, and ocular secretions and in milk. LSDV is known to persist in the male genital tract, with viral DNA detected in semen 5 months after infection in cattle (Annandale et al. 2010; Irons, Tuppurainen & Venter 2005). Intrauterine transmission of LSD has been demonstrated (Rouby & Aboulsoud 2016), as has seminal transmission under experimental conditions (Annandale et al. 2014). LSDV is extremely stable in the environment and is able to survive in favourable conditions on fomites (e.g. hay and straw) for up to 6 months. Viral DNA has been detected in skin lesions in cattle up to 92 days post infection (Tuppurainen, Venter & Coetzer 2005). The persistence of virus in skin nodules and scab material may constitute an important factor in maintenance of the disease in the field, providing biting insects with sufficient levels of virus to enable propagation to new hosts (Diallo & Viljoen 2007).

Clinical disease from natural infection with LSDV is seen in cattle (*Bos taurus* and *B. indicus*) and water buffalo (*Bubalus bubalis*). A variety of species such as giraffe, springbok and impala are known to be susceptible to LSDV (Davies 1982; Fagbo, Coetzer & Venter 2014), and LSDV-specific antibodies have been demonstrated in various wild ruminants such as eland and greater kudu (Barnard 1997; Tuppurainen et al. 2015b).

The significance of wildlife in transmission or maintenance of LSDV is unknown. Virologically confirmed cases of clinical LSD have not been detected in African wildlife. A giraffe and impala died from experimental infection with LSDV, however experimentally infected buffalo and black wildebeest did not seroconvert nor develop signs of disease (Young, Basson & Weiss 1970).

Clinical disease has been reported in both wild and captive giraffes (Muneza et al. 2016). Clinical disease through natural infection was reported in a single captive Arabian oryx and a serological survey of the herd showed a low prevalence (2 per cent) of infection (Greth et al. 1992). Clinical disease suspected to be LSD has been described in water buffalo in Egypt (Ali et al. 1990), springbok in Namibia, and oryx in South Africa (Coetzer 2004). LSDV nucleic acid has been reported in springbok skin samples (*Antidorcas marsupialis*) collected in South Africa (Lamien et al. 2011).

The presence of LSDV antibodies in a host species indicates species susceptibility to the virus and potential involvement in the epidemiology of the disease but is not proof that the host can amplify or transmit the virus (Barnard 1997; Tuppurainen & Oura 2012a). Immune response to capripoxviruses is known to be predominantly cell-mediated and the serological response to infection or vaccination may be below detectable limits (Diallo & Viljoen 2007; Tuppurainen & Oura 2012a).

Although attempts have been made to identify a wildlife reservoir for LSDV in Africa, there is no evidence that non-domestic mammals are involved in the epidemiology of the disease. Serosurveys have found antibodies to LSDV in a number of free ranging wild ungulate species in Africa (African buffalo, greater kudu, waterbuck, reedbuck, impala, eland, springbok, black wildebeest, blue wildebeest and giraffe) (Barnard 1997; Fagbo, Coetzer & Venter 2014; Hedger & Hamblin 1983). In one study, antibody titres in giraffe and reedbuck samples were reported to be as high as in convalescent cattle (Hedger & Hamblin 1983). Although several authors have suggested a role for African buffalo in LSDV epidemiology (Fagbo, Coetzer & Venter 2014; Hedger & Hamblin 1983; Hunter & Wallace 2001; Tuppurainen & Oura 2012b), authors have concluded that the role of wildlife is either unclear or insignificant (Fagbo, Coetzer & Venter 2014; Hedger & Hamblin 1983; Tuppurainen & Oura 2012b).

Immunity from natural infection with Capripoxviruses is thought to be lifelong and no carrier state exists (Coetzer 2004).

The incubation period in natural outbreaks is unknown but is estimated to be between 1-4 weeks (Coetzer 2004).

The OIE Code considers the incubation period for LSD to be 28 days for epidemiological purposes (OIE 2017c).

### **Clinical signs**

Infection in cattle causes mild to serious systemic disease and the presentation can be variable even within a herd. Clinical disease is characterized by fever, skin nodules, lethargy, inappetance, lymphadenitis, salivation, ocular and nasal discharges, pox lesions in ocular, nasal and oral mucous membranes and the surface of internal organs, and sometimes death (Gari et al. 2015; Tuppurainen et al. 2013). A classic sign of LSD infection is the progression of skin nodules to a centrally cavitated, necrotic 'sit-fast' appearance. Scabs from skin lesions eventually detach. Skin lesions gradually resolve over several months but maintain high levels of virus during this time. Infection leads to a prolonged period of debility, and recovery from severe infection may be slow (OIE 2017d).

"Silent" infections (without skin lesions) commonly occur (Tuppurainen et al. 2015b). Experimental infection of cattle with LSDV demonstrated that only a third to one half of infected

individuals develop clinical disease and yet are all viraemic (Osuagwuh et al. 2007; Tuppurainen, Venter & Coetzer 2005).

### Diagnosis

A tentative diagnosis of LSD can be made in animals with a classical presentation of generalised skin nodules. Rapid confirmation of a diagnosis is most commonly performed by conventional or real-time PCR on blood, semen, tissue culture or biopsy samples (OIE 2017d). Current PCR techniques are unable to distinguish between LSDV and sheep and goat pox viruses.

Serial PCRs have been employed as a method of importation surveillance for trade purposes within the European Union (Borrello 2016). Viraemia is detectable by PCR (conventional or real-time) on blood samples during the acute phase of infection, usually between days 4 to 14.

The virus neutralization assay is the most widely used test for detecting capripoxvirus antibodies in domestic species. Other available tests include ELISA, indirect fluorescent antibody test and Western blot (Diallo & Viljoen 2007). Immunity is primarily cell mediated and the level of antibody response may not be indicative of the individual's ability to resist challenge with virulent virus (Coetzer 2004). There is no ability to differentiate naturally infected individuals from those that have received a LSDV vaccine, though a method to distinguish field virus from vaccine virus by PCR exists (FAO 2017).

Virus isolation on cell culture is available. A sandwich ELISA is available that can detect capripoxvirus antigen. Fluorescent antibody tests may also be performed but are subject to nonspecific reactions (OIE 2017d). Electron microscopy of tissues has also been used (Diallo & Viljoen 2007).

### Prevention

In domestic ruminants, cross-immunity occurs between the three members of the *Capripoxvirus* genus (LSDV, sheep pox virus, goat pox virus). There are several vaccines against LSDV, based on strains of LSDV (e.g. Neethling, SIS) or sheep and goat pox virus strains (e.g. RM-65, Gorgon™). Only live attenuated vaccines are currently available. The use of goat and sheep pox strain live attenuated vaccines is not recommended for use in regions free from these diseases due to the risk of introducing these diseases (OIE 2017d). There is no vaccine with a Differentiation of Infected from Vaccinated Animals (DIVA) component.

Vaccination is used to help control outbreaks and, in countries where the disease is endemic, in reducing overall incidence of disease, but none of the available vaccines confer complete protection for a herd (Tuppurainen et al. 2015b). Whilst necessary for disease control, vaccination also complicates disease surveillance; vaccination may mask clinical signs in animals, a problem compounded by the high proportion of animals that may not display clinical signs of infection yet be viraemic and capable of transmitting the disease (Tuppurainen et al 2015; Tuppurainen et al 2005; Annandale et al 2010); the live attenuated vaccines may cause clinical signs mimicking LSD in a proportion of vaccinated cattle, in addition to shedding vaccine virus (Abutarbush 2014; Bedekovic et al 2017; Tasioudi et al. 2016).

Studies on vaccination against LSDV in species other than cattle were not identified in the literature review.

**Treatment**

Treatment is symptomatic only. Animals may be subject to stamping out or modified stamping out to control outbreaks of LSD depending on the competent authority's plan for control.

**4.14.3 Current biosecurity measures**

There are no previous biosecurity measures for live animals. Country freedom is used for risk commodities such as dairy. The OIE Code recommendations include country freedom or isolation (OIE 2017c).

**4.14.4 Risk review**

LSD is present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of LSD in zoo bovidae:

- LSD is endemic to most of Africa and the Middle East. It has recently spread to eastern European countries previously free from the disease.
- LSD is an emerging, transboundary disease that is highly contagious and difficult to control. It is transmitted predominantly by biting insects but other modes of transmission are possible.
- An outbreak of LSD would result in severe consequences to Australian industry and export markets.
- There is a risk of LSD becoming endemic with permanent impacts on Australia.
- Serological evidence of LSD has been detected in a wide range of African ruminant species. There is no clear evidence of a role for wildlife in the epidemiology of the disease.
- Capripoxviruses like LSD are fairly host-specific. However, natural and experimental LSD or LSD viral material has been reported in a range of species other than cattle.
- Immunity after natural infection is lifelong and there is no known carrier state of LSD in animals.
- Accurate assessment of a region's disease status is difficult in vaccinated populations.
- Vaccination does not provide full or lasting immunity for individual animals, and can hinder detection of disease incursions.
- There is no vaccine which would allow differentiation of infected from vaccinated individuals.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.
- The quarantine facilities used in zoo pre-export quarantine and post-arrival quarantine are not typically vector proof.

**4.14.5 Conclusion**

Based on the preceding information specific risk management measures are warranted.

Australia's proposed risk management procedures for LSD for zoo bovidae are:

### **Option ONE**

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of LSDV has occurred during the previous 3 years, the disease is compulsorily notifiable and vaccination against LSDV has not occurred in the previous 3 years.

AND

- The animal showed no clinical signs of LSD during pre-export quarantine (PEQ).

AND

- The animal has not been vaccinated against capripoxviruses in the previous 3 years (LSDV or Sheep or Goat Pox strain vaccines).

### **Option TWO**

The department will consider applications to import live bovids from approved countries that do not meet the requirements of Option ONE on a case-by-case basis, with respect to the epidemiological situation at the time. As a guide, minimum requirements will consist of all of the following:

- The animal has not been vaccinated against capripoxviruses in the previous 3 years (LSDV or Sheep or Goat Pox strain vaccines).
- The animal showed no clinical signs of LSD during pre-export quarantine (PEQ).
- During the final 7 days of pre-export quarantine, each animal was tested for LSD with a PCR test and returned negative results.

Australia's proposed risk management procedures for LSD for zoo bovidae semen are:

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

## **4.15 Malignant catarrhal fever (wildebeest associated)**

### **4.15.1 Background**

Malignant catarrhal fever (MCF) is an acute, generalised and usually fatal disease affecting many species of Artiodactyla. The disease has been most often described as affecting species of the subfamily Bovinae and family Cervidae, but is also recognised in domestic pigs as well as giraffe and species of antelope belonging to the subfamily Tragelaphinae (OIE 2013a).

Sheep-associated MCF (SA-MCF) is the predominant form outside Africa. It is a particular problem in farmed species such as bison, deer and Bali cattle, although it occasionally affects relatively resistant hosts such as pigs and European breeds of cattle.

Wildebeest associated MCF (WA-MCF) is an important disease among cattle in Africa, while zoo ruminants can be affected by either of these two forms, as well as by less common MCF viruses

carried in other species of non-domestic ruminants. Malignant catarrhal fever is difficult to control, as infection is widespread and asymptomatic in the reservoir species, and the incubation period can be long in susceptible animals. The only reliable methods of control are to separate susceptible species from carriers or to breed virus-free reservoir herds (Spickler 2016b).

There are currently no wildebeest (*Connochaetes* spp.) in Australian zoos and the genus was last held in captivity in Australia in the 1970s (A Reiss pers. comm. ZAA, May 2017).

MCF is not an OIE-listed disease (OIE 2018c) but WA-MCF is included in the OIE's Working Group for Wildlife Diseases list of non-OIE listed pathogens found in wildlife (OIE 2014e). Malignant catarrhal fever caused by ovine herpesvirus-2 (OvHV-2) is present in Australia and is not a nationally notifiable disease. The status of other recently identified MCF-like herpesviruses in Australia is unknown. Other than WA-MCF, the other MCF viruses are either not nationally notifiable, are not known to cause disease in spill over hosts, or are severely restricted in information and so are not considered further in this review. This review is primarily concerned with information, risks and risk management of alcelaphine herpesvirus-1 (AIHV-1), the causative agent of WA-MCF.

WA-MCF is not present in Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016).

#### 4.15.2 Technical information

##### Epidemiology

Malignant catarrhal fever is caused by a group of herpesvirus in the family *Herpesviridae*, subfamily *Gammaherpesvirinae*, genus *Macavirus* (OIE 2013a). The viruses cause little to no clinical disease in their host species, but result in serious and often fatal systemic disease when non host species become infected. Malignant catarrhal fever may result in serious disease in many species of *Artiodactyla*.

There are ten identified members with the MCF subgroup of viruses, of which six have been associated with clinical disease.

- Wildebeest-associated MCF, caused by alcelaphine herpesvirus 1, is endemic in wildebeest populations worldwide, both captive and free-living.
- Sheep-associated MCF, caused by ovine herpesvirus 2 (OvHV-2), is endemic in most sheep populations worldwide, including in Australia.
- Caprine-associated MCF, caused by caprine herpesvirus 2 (CpHV-2), is endemic in most goat populations worldwide, including Australia. It also causes MCF disease in cervids.
- MCF in white tailed deer (MCFV-WTD) is caused by a virus whose origins are currently unknown.
- Ibex MCFV (MCFV-ibex) is carried by Nubian ibex (*Capra nubiana*) and has caused fatal clinical disease in bongo (Gasper et al. 2012; Li et al. 2003; Okeson et al. 2007).
- Alcelaphine herpesvirus-2 (AIHV-2) is carried by hartebeest (*Alcelaphus buselaphus*) and topi (*Damaliscus lunatus*). There are sporadic reports of AIHV-2 spill over to cervidae hosts and mortality, but other than experimental inoculation the disease is not recognised as

overtly pathogenic (Flach et al. 2002; Miller & Fowler 2014; Taus et al. 2014). An AIHV-2-like virus, carried by a subspecies of hartebeest (*Alcelaphus buselaphus*), has caused serious disease in Barbary red deer (*Cervus elaphus barbarous*) (Klieforth et al. 2002).

- The remaining four viruses carried by roan antelope (hippobovine herpesvirus 1) (Reid & Bridgen 1991), oryx, muskox and aoudad have not been associated with disease in ruminants (Li et al. 2003; Li et al. 2005), however hippobovine HV-1 has been reported to experimentally cause disease in rabbits (Russell, Stewart & Haig 2009).

The natural hosts of alcelaphine herpesvirus-1 are both species of wildebeest (*C. gnou* and *C. taurinus*). Infection with AIHV-1 occurs perinatally in all wildebeest calves in natural herds and all wildebeest calves are considered to become infected within the first few weeks of life. Infection does not result in detectable clinical disease in wildebeest but individuals remain carriers of the virus for life. The principle source of free virus in wildebeest is tears and nasal secretions (Russell, Stewart & Haig 2009). One to two month old wildebeest calves have the highest incidence of free viral shedding (Wambua et al. 2016; Whitaker et al. 2007). Free virus is only excreted by wildebeest and not by other infected species. The minimum distance to prevent aerosol transmission has been suggested as 100 metres though a few cases have been reported in animals separated by more than this distance (Spickler 2016b; Whitaker et al. 2007).

Transmission of the virus occurs when susceptible hosts, including other wild or zoo ruminants and cattle, come into direct or indirect contact with wildebeest excreting the virus (Wambua et al. 2016; Whitaker et al. 2007). The susceptibility of various species to MCF infection and disease varies significantly. Banteng, American and European bison, water buffalo, yak and certain deer species are considered highly susceptible to infection (Gasper et al. 2012; Pfitzer et al. 2015; Russell, Stewart & Haig 2009). In both field and experimental conditions, animals that exhibit signs have mortality rates approaching 100 per cent (Spickler 2016b; Wambua et al. 2016).

Susceptible animals are generally infected when they are exposed to parturient wildebeest or young wildebeest calves, or to pasture contaminated by them (Reid & Van Vuuren 2004). Transmission of AIHV-1 in Africa is generally associated with livestock coming into direct or indirect contact with calving wildebeest (Pretorius, Oosthuizen & Van Vuuren 2008). In the zoo setting, AIHV-1 has been transmitted to clinically susceptible species despite segregation from infective sources (i.e. calving wildebeest or young wildebeest calves) (Meteyer et al. 1989; Okeson et al. 2007). Persistent infection may be induced in cattle under experimental conditions, however, non-*Connochaetes* spp. with clinical disease are considered to be dead end hosts (Berezowski 2003; Horner & Tham 2003; Reid & Van Vuuren 2004; Spickler 2016b; Vorster & Mapham 2013; Wambua et al. 2016).

AIHV-1 has not been found in wild ungulates or livestock outside Africa (Kálmán & Egyed 2005; Staric et al. 2015; Zarnke, Li & Crawford 2002). Outside Africa, AIHV-1 has only been detected in zoo collections which contain *Connochaetus* spp. OvHV-2 may also be the cause of MCF in zoos, including in areas (such as Australia) where AIHV-1 does not occur (Matzat, Eulenberger & Mueller 2015; Meteyer et al. 1989; Modesto et al. 2015).

The incubation period varies but is generally accepted to be around three weeks. Incubation periods as long as nine months have been reported (Whitaker et al. 2007).

Specific information on the presence of AIHV-1 in semen was not located. However, as the reservoir species (wildebeest) are primarily infected in the first few months of life or during the

last stage of gestation, it is unlikely that semen constitutes a transmission risk. Zoo bovidae other than *Connochaetus* spp. are considered dead-end hosts and semen collection from a healthy donor represents negligible or zero transmission risk.

### **Clinical signs**

The disease may present with a wide spectrum of clinical manifestations ranging from the acute form, where minimal changes are observed prior to death, to more florid cases. MCF usually appears sporadically and affects few animals, though the virus can give rise to epidemics. MCF is characterised by high fever, bilateral corneal opacity, profuse catarrhal discharges from the eye and nostrils, necrosis of the muzzle and oral erosions. Death may be peracute or acute, with a high fatality rate. Death can occur within a few days or up to several weeks after onset of clinical signs (Russell, Stewart & Haig 2009).

### **Diagnosis**

Diagnosis is via histopathology, PCR or serology (ELISA, VN or IMPO). Cross-reactivity between AIHV-1 and OvHV-2 has been an issue with both serological and PCR tests but accurate discrimination may be possible by more recent PCR methods (Bremer et al. 2005; Kálmán & Egyed 2005; Wambua et al. 2016).

### **Prevention**

No vaccine is commercially available (OIE 2013a), however efforts continue to develop a vaccine for WA-MCF (Wambua et al. 2016).

The only currently effective prevention strategy is segregation of susceptible animals from the reservoir host (*Connochaetes* spp.) and prevention of indirect transmission pathways (Spickler 2016b; Vorster & Mapham 2013). Creation and maintenance of a virus-free *Connochaetes* herd would entail significant long-term efforts to identify uninfected animals.

## **4.15.3 Current biosecurity measures**

There are no previous biosecurity measures for live animals and MCF. There are no recommendations in the OIE Code.

## **4.15.4 Risk review**

WA-MCF (AIHV-1) is present in some approved countries. It is not present in Australia and is a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of WA-MCF in non-domestic zoo bovidae:

- WA-MCF has the potential to cause adverse effects to exposed wildlife and livestock if introduced to Australia.
- All wildebeest (*Connochaetes* spp.), whether captive or free-ranging (unless from a specific virus-free herd), can be considered to have acquired AIHV-1 in the early months of their life.
- Infection is persistent and infected wildebeest can be considered to carry, and potentially shed, the virus throughout their lives.
- The virus is only excreted by the natural host, wildebeest.
- Other domestic and wild species are dead-end hosts.

- Transmission to susceptible animals is via direct or indirect contact with wildebeest, e.g. via contaminated pasture.
- Aerosol transmission may occur at distances over 100 metres.
- Susceptibility to WA-MCF may vary between bovidae species but if clinical disease develops the morbidity and mortality rates are invariably high.
- The presence or transmission of the virus in semen is unlikely, especially in non-*Connochaetes* spp.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.15.5 Conclusion

Accordingly and based on the preceding information, risk management measures are warranted.

Australia's proposed risk management procedures for live zoo bovidae are:

- No live animals of the genus *Connochaetes* may be imported into Australia.

Australia's proposed biosecurity measures for zoo bovidae semen are:

- The animals from which the semen for export was collected are not members of the genus *Connochaetes*.

Importation of live animals or semen from the genus *Connochaetes* may be considered if they are sourced from herds with demonstrated freedom from WA-MCF. The herd source and evidence of disease freedom must be assessed and approved by the department on a case-by-case basis.

## 4.16 Nairobi sheep disease

### 4.16.1 Background

Nairobi sheep disease (NSD) is a serious haemorrhagic disease of small ruminants caused by Nairobi sheep disease virus group (NSDV), within the genus *Nairovirus*, family *Bunyaviridae* (Davies & Terpstra 2004). A closely related agent, Ganjam virus, is considered to be a different isolate of the same virus. Dugbe virus is also represented within the Nairobi sheep disease group (Marczinke & Nichol 2002).

NSD causes severe disease in sheep and goats and primarily occurs in Central and Eastern Africa, with serological evidence of infection reported from Kenya, Ethiopia, Somalia, Botswana and Mozambique. Ganjam virus has been reported in sheep and goats in India and Sri Lanka. (Marczinke & Nichol 2002). Evidence of NSDV has recently be reported from ticks in China (Gong et al. 2015).

Both NSDV and Ganjam virus have been reported to cause disease in humans, including flu like symptoms in humans.

NSD is an OIE listed disease (OIE 2018c). It is absent from Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016).

## 4.16.2 Technical information

### Epidemiology

NSD is a serious disease of small ruminants, primarily of sheep, although goats may also be clinically affected. Both sheep and goats are considered natural hosts of the virus (Davies & Terpstra 2004). Ganjam virus is considered to be less pathogenic than NSDV both in goats, sheep and humans.

NSD is primarily transmitted by the bite of ticks, although midges may also be implicated in transmission. NSDV in Africa is primarily transmitted by the Ixodid tick *Rhipicephalus appendiculatus* whereas Ganjam virus is primarily transmitted by *Hamaphysalis intermedia* and *H. alis* (Marczinke & Nichol 2002; Perera et al. 1996). The virus is maintained in tick populations by transovarial and transstadial transmission and can persist in infected ticks for more than two years.

NSDV is not considered to be directly contagious between animals, although virus can be shed in urine and faeces (Animal Health Australia 2013). Infection in humans is acquired through tick bite or needle stick.

The incubation period in sheep is generally 4-7 days, with a range of 1-15 days. Sheep and goats are the only known vertebrate reservoirs and amplifying hosts of NSD (Marczinke & Nichol 2002). Amongst domesticated animals, only sheep and goats are readily infected (Animal Health Australia 2013; Spickler 2016c). Susceptibility varies amongst breeds and strains of sheep and goats (OIE 2014c).

Antibodies to NSDV have been found in some species of wild ruminants in Kenya at low prevalence (Davies 1978b). Attempts to isolate the virus from a range of wild ruminants were not successful (Davies 1978a). Blue duiker (*Cephalophus monticola*) are reported to have developed natural infection, both in the wild and in an African zoo, with some cases resulting in mortality (CABI 2016; Haddow 1958). However, serological studies have not demonstrated evidence of a significant role for duiker in the cycle of NDSV (Davies & Terpstra 2004). Waterbuck (*Kobus ellipsiprymnus*) and other wild ruminants found to be heavily infested with *Rhipicephalus appendiculatus* have not demonstrated high prevalence of NSDV antibody (Davies & Terpstra 2004). In comparison, most sheep and goats in endemic areas have antibody to the virus, and the geographic prevalence of antibodies is closely linked to presence of *Rhipicephalus appendiculatus*. Other than these reports, disease associated with NDSV has never been identified in wild ruminants and there is no evidence to suggest that wild or zoo bovidae play a role in the epidemiology of this disease (CABI 2016; Davies 1978a; Davies 1978b; Spickler 2016c).

A review of the scientific literature found no evidence for shedding of NSDV in germplasm (Williams 2003). The virus is noted to be fragile outside its optimal pH range and not known to persist outside the host or ticks.

### Clinical signs

NSDV causes acute haemorrhagic gastroenteritis in sheep and goats, with a high rate of morbidity. Clinical signs include fever, leukopenia, anorexia, rapid respiration, foetid diarrhoea, depression, bloody nasal discharge, swollen lymph nodes and abortion. The case fatality rate may be as high as 90 per cent in susceptible populations of these species (Davies & Terpstra

2004; Marczinke & Nichol 2002). Death may occur quickly (within 12 hours of onset of signs) or around 3-7 days into the disease (OIE 2014c).

### **Diagnosis**

Diagnosis of NSD may be made by identification of the virus from diagnostic materials or by detection of antibodies. Serological tests include the indirect fluorescent antibody test (FAT), which is considered the most suitable. Other tests include the ELISA and an RNA probe (OIE 2014c).

### **Control and management**

Animals bred in endemic areas are protected by maternal antibodies when young and then develop additional immunity through natural exposure. Naïve animals which are brought into endemic areas are most at risk of disease. The disease is very difficult to eradicate once established in vector populations. Control focuses on movement control of susceptible hosts and control of tick populations. There is no safe vaccine commercially available, however experimental vaccines have been developed in some endemic areas (Spickler 2016c).

## **4.16.3 Current biosecurity measures**

There are no previous biosecurity measures for NSD. There are no recommendations in the OIE Code (2014c).

## **4.16.4 Risk review**

NSD is not present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of NSDV in zoo bovidae:

- NSDV is an OIE-listed disease and is nationally notifiable in Australia.
- NSD infection may result in severe disease in naïve sheep and goats and mild disease in humans. Sheep and goats are the only known vertebrate reservoirs and amplifying hosts of NSD.
- Natural transmission of NSDV between animals is only considered to occur via tick bite; no direct transmission has been reported.
- The virus is maintained in tick populations in endemic areas and may survive two years in a tick without a blood meal.
- General zoo import conditions include examination of animals for parasites and acaricide treatment.
- There is no evidence that NSDV is shed in semen of infected animals.
- There is no evidence to suggest that any of the zoo bovidae covered by this policy play a role in the epidemiology of this disease.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.16.5 Conclusion

Based on the preceding information, specific risk management measures for NSD for live animals and semen are not warranted.

### 4.17 Peste des petits ruminants

#### 4.17.1 Background

Peste des petits ruminants (PPR) is a highly contagious disease of sheep and goats caused by a virus in the genus *Morbillivirus*, family *Paramyxoviridae* (Taylor 1984). It is related to, and almost certainly evolved from, rinderpest virus (Geering, Forman & Nunn 1995). PPR occurs in most countries of central, west and North Africa as well as the Middle East, central, south and south-east Asia. The disease appears to be spreading geographically into Tibet, Mongolia, Kazakhstan, China, Morocco and East Africa (Banyard, Wang & Parida 2014; Bao et al. 2011; Kock et al. 2017; OIE 2013c).

PPR is an OIE listed disease (OIE 2018c).

PPR is not present in Australia and is a nationally notifiable disease (Department of Agriculture and Water Resources 2016). PPR is a Category 2 disease under the EADRA and has the potential for rapid spread with serious impacts on production, mortality and trade.

#### 4.17.2 Technical information

##### Epidemiology

In domestic livestock, PPR is primarily a disease of goats, in which it is most severe, and sheep. The incubation period in sheep and goats is usually 4 - 5 days, but may range from 3 to 10 days (Couacy-Hymann et al. 2007; Geering, Forman & Nunn 1995). Mild forms of the disease may allow infected animals to convalesce rapidly (within two weeks) (Parida et al. 2015), however most infections result in severe clinical effects. The mortality rate may be as high as 90 per cent in naïve goat and sheep flocks, and similarly high in vulnerable non-domestic species (Kock et al. 2017). The virus is shed by infected animals in expired air and in all secretions and excretions during the acute stage. Viral shedding usually begins one to two days before onset of clinical signs (Couacy-Hymann et al. 2007).

Transmission of the virus is by direct contact between animals, including movement of infected animals into naïve populations (Geering, Forman & Nunn 1995).

Animals that survive the disease generally undergo a long convalescent period. There is no recognised chronic carrier state in recovered goats and sheep (Geering, Forman & Nunn 1995) and recovered animals are immune to reinfection (ESFA 2015; Rossiter et al. 2008). The virus does not survive long outside the host and indirect transmission of the virus is considered very unlikely (Munir 2013; Rossiter et al. 2008). PPR has been reported to cause clinical disease, similar to that seen in sheep and goats in a range of non-domestic bovidae species. Reports are most commonly from antelopes and wild goat species, and most often in a semi-free range situation, although outbreaks in captive bovidae are reported (Parida et al. 2015). It is considered to have the potential to cause disease in some, if not all, of the bovidae family, as well as other non-domestic ruminants. Species in the genus *Gazella* are considered to be some of the most susceptible of the wild ungulates (Munir 2013). In 2016-17 an outbreak of PPR (believed to originate from domestic livestock) resulted in deaths of thousands of saiga antelope in the Gobi Altai of Western Mongolia (Kock et al. 2017).

Fatal PPR infection has also been confirmed in a wide range of other non-domestic bovidae species, with many species reports occurring for the first time in recent years. Fatal infection has been reported in gemsbok, Dorcas gazelle, Nubian ibex (*Capra ibex nubiana*), Laristan mouflon (*Ovis orientalis laristanica*), Thompson's gazelle, bushbuck (*Tragelaphus scriptus*), impala (*Aepyceros melampus*), goitered gazelle (*Gazella subgutturosa*), Sibirian Ibex (*Capra sibirica*), Argali sheep (*Ovis ammon*), Rheem gazelle (*Gazella subgutturosa marica*), Arabian gazelle (*Gazella gazella*), Arabian mountain gazelle (*Gazella cora*), springbok (*Antidorcas marsupialis*), Barbary sheep (*Ammotragus lervia*), Afghan Marhor goat (*Capra falconeri*), water buffalo, Sindh ibex (*Capra aegagrus blythi*) and wild goats (*Capra aegagrus*) (Abubakar et al. 2011; Elzein et al. 2004; Furley, Taylor & Obi 1987; Govindarajan et al. 1997; Hoffmann et al. 2012; Kinne et al. 2010; Kock 2017).

Clinical disease has been reported in bharal (*Pseudois nayaur*) (Bao et al. 2011) and subclinical disease or serological evidence for PPR infection has been reported in nilgai (*Boselaphus tragocermalus*), grey duiker (*Sylvicapra grimmia*), Bubal hartebeest (*Alcelaphus buselaphus*), African buffalo (*Syncerus caffer*), waterbuck (*Kobus ellipsiprymnus*), kob (*Kobus kob*) and goitered gazelle (*Gazella subgutturosa subgutturosa*) (Couacy-Hymann et al. 2005; Furley, Taylor & Obi 1987; Gür & Albayrak 2010; Ogunsanmi et al. 2003). Other non-domestic ungulates such as white tailed deer and camels (*Camelus dromedaries*), may be clinically affected by PPR (Hamdy & Dardiri 1976; Khalafalla et al. 2010).

The epidemiological role of wild ungulates in PPR is not well understood and there is limited information about the disease in these species (Munir 2013; Parida et al. 2015). A recent report recommends further investigation of the potential for PPR to spread via wildlife (ESFA 2015).

Outbreaks of PPR in wild ruminants generally occur in concurrence with outbreaks in nearby livestock. Although wild ruminant species have demonstrated the ability to transmit PPR to domestic stock when habitats overlap, there is no evidence that PPR circulates in wild animals or that wildlife act as reservoirs for the virus (Bello et al. 2016; Munir 2013; OIE 2013c). It is most likely that PPR infection is not self-sustaining in wildlife populations and most of the reported outbreaks in wildlife originate from nearby infected sheep and goats, as was the case with rinderpest (Couacy-Hymann et al. 2005; Munir 2013). Most studies in wildlife have concluded that PPR is not sustainable outside the domestic animal cycle (Mahapatra et al. 2015; Munir 2015). For example, an outbreak of PPR in free-ranging Sindh ibex (*Capra aegagrus blythi*) within a wildlife park was controlled by vaccination of the domestic sheep and goats in surrounding villages (Abubakar et al. 2011). In contrast to the above, a more direct role for wildlife in the epidemiology of PPR is suggested by some authors (Boshra et al. 2015; Kinne et al. 2010).

Minimal information is available regarding PPR presence and transmission in semen. Virus is present in all secretions and excretions from infected animals for approximately 10 days after the onset of fever (Animal Health Australia 2009). Williams (2003) reviewed the persistence of peste des petits ruminants virus (PPRV) in biological materials and concluded (from the limited evidence) that semen from infected animals may contain the virus. Extrapolation from rinderpest data indicates that there may be a risk of transmission for PPR, and that in chilled or frozen conditions (e.g. stored semen) may promote survival (Spickler 2015d). Data on the period of shedding in semen or whether the virus persists in the reproductive tract was not located. Hosts can shed viral antigens in faeces for 11 weeks post-recovery and it is believed that

virus is present in milk for up to 7 weeks post-recovery (Munir, Zohari & Berg 2012). The OIE Code prescribes vaccination or testing and 21 days zone freedom for semen donors based on the incubation period (OIE 2018c).

The OIE Code considers the incubation period to be 21 days for epidemiological purposes (OIE 2018c).

#### **Clinical signs**

Clinical signs of disease in non-domestic ruminants are similar to those seen in goats and sheep and can include severe pyrexia, mucopurulent nasal and ocular discharges, cough, dyspnoea, necrotic stomatitis and diarrhoea. Painful sores in the oral mucous membranes prevent the animal from eating. Clinical signs may be milder in some wildlife species (Kinne et al. 2010).

#### **Diagnosis**

A multitude of tests exist, including virus isolation, molecular testing (e.g. RT-PCR) and detection of antibodies. Virus neutralisation is considered the 'gold standard' and is the prescribed test for international trade (OIE 2018c).

#### **Prevention**

Country freedom and animal movement control are the most effective preventative measures against PPR. The propagation of PPR typically requires an ongoing fresh supply of susceptible hosts, so early detection of disease and strict control of animal movements, combined with isolation, slaughtering-out and disinfection, can significantly limit or halt an outbreak.

Attenuated and recombinant homologous vaccines exist and, whilst effective in small domestic ruminants, their primary use is in countries endemic with PPR, to control overall disease burden. Vaccination has been used in Arabian oryx, but efficacy has not been tested (Sa et al. 2013). No vaccine with a DIVA component exists.

### **4.17.3 Current biosecurity measures**

Australia's current biosecurity measures for PPR for dairy products and germplasm include country freedom. The OIE Code recommendations include country freedom or zone freedom and testing (OIE 2018c).

#### **4.17.4 Risk review**

PPR is not present in approved countries. It is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of PPR in zoo bovidae:

- PPR is an OIE-listed disease and is nationally notifiable in Australia.
- PPR is not present in Europe, the United States, Canada or Singapore.
- PPRV has no known reservoir or carrier state. Immunity after infection in domestic stock is lifelong.
- PPR has the potential for rapid spread with serious impacts on production, mortality, and trade.
- Zoo bovidae species may be infected with PPRV and develop clinical disease.

- Infection in zoo bovidae species has similar clinical signs and course as domestic species.
- Zoo bovidae species may contribute to transmission of PPRV but there is no evidence of a reservoir or carrier state after infection has run its course.
- PPR is very unlikely to spread through indirect methods.
- Limited information exists on the presence and transmission of PPR in semen, although the virus may be shed in body fluids for 10 days after viraemia begins and for prolonged periods in milk and faeces after recovery.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.17.5 Conclusion

Based on the preceding information and in accordance with recommendations in the OIE Code (2014f), risk management measures for zoo bovidae are warranted.

Australia's recommended risk management procedures for PPR in zoo bovidae are:

- Since birth the animal was continuously resident in a country where no clinical, epidemiological or other evidence of PPR has occurred during the previous 2 years and the disease is compulsorily notifiable.

Australia's proposed biosecurity measures for PPR for zoo bovidae semen are:

- Since birth the donor was continuously resident in a country where no clinical, epidemiological or other evidence of PPR has occurred during the previous 2 years and the disease is compulsorily notifiable.

## 4.18 Rabies

### 4.18.1 Background

Rabies virus is a member of the *Lyssavirus* genus of the family *Rhabdoviridae* (Tordo et al. 2005). There are currently 14 species in the *Lyssavirus* genus including the type species rabies virus (ICTV 2014). Rabies virus causes a progressively fatal encephalitis that can affect all species of mammals, including ruminants. Rabies is seen predominantly in domestic dogs, with other species of the order *Carnivora* (particularly canids) and *Chiroptera* (bats) recognised as wildlife reservoirs (ARMCANZ 1996; World Health Organisation 2005). Rabies is present virtually worldwide and is common on all continents except Australia and Antarctica. Many island countries, territories and states are also free of rabies.

Australia is free of classical rabies. However, a lyssavirus has been isolated from bats that causes disease in bats, humans (Field, McCall & Barrett 1999; Gould et al. 1998; Greene & Rupprecht 2006) and horses (Shinwari et al. 2014).

Rabies is an OIE-listed disease (OIE 2018c).

Rabies is not present in Australia and it is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016). Rabies is a zoonosis and a nationally notifiable disease of public health concern (Health 2015).

#### 4.18.2 Technical information

##### Epidemiology

The dog is the chief source of infection but a wide variety of species of the orders *Carnivora* and *Chiroptera* can act as reservoirs (Geering & Forman 1987; World Health Organisation 2005). Transmission is normally through biting when virus in the infected animal's saliva enters the new host through broken skin. In dogs, virus may be present in the infected animal's saliva up to 14 days before the onset of clinical signs. Rabies virus may be transmitted between species, resulting in either a dead-end infection (where there is no further transmission of the virus), or transmission of the virus by the new host. Spill over infections can cause sporadic cases of rabies without further transmission due to there being no other species to interact with, low salivary shedding of virus or failure of infection to induce biting behaviour (Bingham 2005).

The incubation period in all species is variable and may be prolonged. It is influenced by the quantity of virus introduced, proximity of the bite site to the head, the sensory innervation at the bite site, the age of the animal and the biotype of the rabies virus involved (Kaplan 1969; Niezgoda, Hanlon & Rupprecht 2002). In canids, incubation is normally four to eight weeks, but can be as short as four days or greater than a year in very rare cases (Kaplan 1969; Swanepoel et al. 1993). The incubation period in experimentally infected cattle and sheep averaged 15 and 11 days, respectively (Hudson et al. 1996). On rare occasions, circumstantial evidence has pointed towards incubation periods in humans as long as several years (Geering & Forman 1987; Smith et al. 1991).

Domestic and wild herbivores such as bovids are considered dead-end hosts because they usually succumb to disease and die without further transmission (Rubenstein et al. 2012).

Horizontal transmission of rabies infection between ruminant hosts is extremely rare. However, a unique epidemiological situation exists in Namibia where horizontally transmitted epidemic cycles of rabies occur in greater kudu (*Tragelaphus strepsiceros*), reported from the 1970s onwards. It is estimated that over 50,000 kudu died in the 1970s and 1980s as a result. A second major epizootic occurred between 2003 and 2011, with an estimate of up to 70 per cent of the Namibian kudu population on game ranches lost. The mode of transmission between these animals has not been determined, although social browsing and/or mutual grooming have been proposed (Barnard & Hassel 1981; Scott, Hassel & Nel 2012; Scott et al. 2013; Swanepoel et al. 1993). Suggested risk factors include the high susceptibility of kudu to acquiring rabies infection through unbroken mucous membranes, the high concentration of rabies virus in the saliva of infected individuals and the very high density of kudu in the years prior to the epidemic (Barnard et al. 1982; Scott, Hassel & Nel 2012). An increase in jackal rabies was noted prior to the epidemic which may have been the source of spill over. Infection may have been subsequently communicated to other herbivores such as cattle and eland antelope (*Taurotragus oryx*) (Swanepoel 2004).

Kudu are commonly held species in zoos globally, including in countries where rabies is endemic. There are no reports to indicate that a horizontal transmission of rabies occurs in zoo kudu, nor that zoo kudu are abnormally susceptible to rabies compared to other ungulates. There are no other reports of bovids acting as reservoirs of rabies virus.

There are no reports of rabies persistence or transmission in semen of any bovid species and it is not considered further.

OIE recommendations are based on an incubation period of six months (OIE 2018c).

### **Clinical signs**

The initial clinical signs of rabies are non-specific and may include inappetance, malaise and pyrexia. Hydrophobia, paresis and paralysis are common. Death usually results from cardiac or respiratory failure (Rupprecht 1999). In domestic cattle, sheep and goats, infected animals typically show neurological disease with paralytic (dumb) or aggressive (furious) signs. Clinical signs described in ruminants include aggression, restlessness, salivation, abnormal bellowing, incoordination and other signs of neurological disease (Hudson et al. 1996; Swanepoel 2004). The furious form of rabies was seen in 70 per cent of the experimentally infected cattle and in 80 per cent of sheep (Hudson et al. 1996). The most frequently observed signs in the 1977 to 1985 kudu epidemic in Namibia were salivation, docility and paresis or paralysis. In South Africa small antelope such as duikers were reported to sometimes show increased aggression (Swanepoel 2004).

### **Diagnosis**

No test reliably detects rabies in the incubation or prodromal stages of the disease. In animals, diagnosis is confirmed at post-mortem. The most widely used test for rabies diagnosis is the indirect fluorescent antibody test, recommended by World Health Organization (WHO), Center for Disease Control and Prevention (CDC) and OIE. This test may be used direct on a brain smear and takes only a few hours to perform. Other post-mortem tests include nucleic acid detection assays such as PCR assays and viral cultures such as the rabies tissue culture infection test (OIE 2016e).

### **Prevention**

A range of highly effective, safe and thermostable inactivated veterinary vaccines exist for use in domestic carnivores and herbivores. Duration of protective immunity varies between one to three years depending on the antigen content of the specific vaccine and the rate of challenge. Recombinant and DNA vaccines against rabies continue to be developed.

## **4.18.3 Current biosecurity measures**

Australia's current biosecurity measures for rabies include premises and country freedom. The OIE Code recommendations include country or premises freedom (OIE 2018c).

## **4.18.4 Risk review**

Rabies is present in exporting countries. It is not present in Australia and is a nationally notifiable animal disease.

The following key points are relevant to the biosecurity risk of rabies in zoo bovids:

- Rabies is endemic in most countries across Africa, Asia and the Americas.
- Rabies is a nationally notifiable animal disease and a nationally notifiable disease of public health concern.
- Rabies is a multiple species OIE-listed disease.
- The incubation period in all species is variable and may be prolonged.
- Rabies has been reported in both wild and captive bovid species.

- Rabies transmission to humans via exposure from infected bovids is considered extremely unlikely.
- Rabies occurrence in zoo bovids is very rarely reported.
- Bovid (other than greater kudu in Namibia) are considered dead-end hosts for rabies virus. Horizontal transmission of rabies has never been reported in kudu (or other bovids) held in zoos, and is considered extremely unlikely to occur.
- There are no reports of transmission of rabies in semen of any bovidae species.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.18.5 Conclusion

Accordingly and based on the preceding information, risk management for live zoo bovids is warranted. Risk management measures for semen are not warranted.

Australia's proposed biosecurity measures for rabies for zoo bovids are:

- For 180 days immediately before export the animal did not reside on any premises where clinical, epidemiological or other evidence of rabies occurred during the previous twelve months before export and the disease is compulsorily notifiable.

### 4.19 Rift Valley fever

#### 4.19.1 Background

Rift Valley fever (RVF) is a transboundary disease caused by the arthropod-borne Rift Valley fever virus (RVFV), family Bunyaviridae, genus Phlebovirus (Nichol et al. 2005). RVF is a zoonotic disease of ruminants considered endemic in sub-Saharan Africa and Madagascar (Arishi et al. 2000; Gould & Higgs 2009). In recent decades RVF has extended its geographic range to northern African countries, the Arabian Peninsula and the Indian Ocean archipelagos of Mayotte and Comoros. Commercial movement of infected animals and windborne movement of infected mosquitoes are reasons suggested for the detection of epizootics outside the usual RVFV endemic regions (Ikegami 2012; Mansfield 2015).

RVFV affects a large number of species, including camels, monkeys, rodents and ruminants. Serological or clinical evidence of RVF infection has been reported in a wide variety of African bovidae species, including impala (*Aepyceros melampus*), topi (*Damaliscus korrigum*), red-fronted gazelle (*Eudorcas rufifrons*), Thompson's gazelle (*Eudorcas thomsonii*), dorcas gazelle (*Gazella dorcas*), sable (*Hippotragus niger*), waterbuck (*Kobus ellipsiprymnus*), gerenuk (*Litocranius walleri*), dama gazelle (*Nanger dama*), scimitar-horned oryx (*Oryx dammah*), reedbuck (*Redunca redunca*), African buffalo (*Syncerus caffer*) and greater kudu (*Tragelaphus strepsiceros*) (Anderson & Rowe 1998b; Evans et al. 2008; LaBeaud et al. 2011; Maurice 1966; Ringot et al. 2004; Swanepoel & Coetzer 2004). Ruminants are highly susceptible to RVF and are considered the major amplifying hosts.

Outbreaks of RVF may cause significant disease burden with large numbers of livestock affected and severe economic consequences from the loss of neonates and young animals, disease control

costs and lengthy trade restrictions (Garcia-Bocanegra et al. 2016). Countries with a history of infection in live animals are very likely to remain infected with RVF virus (Gerdes 2004).

RVF is a zoonosis but is not a nationally notifiable disease of public health concern in Australia.

RVF is an OIE-listed (OIE 2018c).

RVF is not present in Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016).

## **4.19.2 Technical information**

### **Epidemiology**

The epidemiology of RVF is complex with important links to ecological and climatic conditions which may vary between geographic regions (e.g. East Africa vs West Africa). A characteristic feature of RVF is its occurrence as cyclical epidemics separated by quiescent periods of 5-15 years or longer (Gerdes 2002). Epidemics are strongly linked to persistent heavy rain, raising of the water table, and flooding, which triggers the emergence of large numbers of floodwater breeding mosquitoes (Gerdes 2002; Manore & Beechler 2015). The intensification of a variety of vectors involved in both biological and mechanical transmission results in a RVF epidemic.

Maintenance of RVFV through the irregular inter-epidemic periods is uncertain. Two main theories exist: long-term survival in mosquito eggs infected via vertical transmission; and cryptic cycling in as yet undetermined hosts (Beechler et al. 2015; Chevalier et al. 2010). The two hypotheses are not mutually exclusive, with some authors proposing maintenance occurs due to a combination of many factors (Manore & Beechler 2015; Olive, Goodman & Reynes 2012a). Low-level inter-epidemic cycling of the disease is known to occur, both as sporadic clinical cases and subclinical circulation, arising predominantly from mosquito bites (Chevalier et al. 2010; Pepin et al. 2010). Serological studies have demonstrated sub-clinical circulation of RVFV between ruminants and humans (Cetre-Sossah et al. 2009; Chevalier et al. 2011; Fafetine et al. 2013; Lernout et al. 2013; Lichoti et al. 2014; Sumaye et al. 2013).

Infected ruminants display high levels of viraemia (Manore & Beechler 2015). Vectors for transmission of RVFV are haematophagous insects, mainly mosquitoes. Dissemination of RVFV is generally over short distances but may spread to distant regions with translocation of infected animals or mosquitoes (Pepin et al. 2010).

Several mosquito species present in Australia are considered to be competent vectors for RVFV (Turell & Kay 1998).

A maintenance host in African wildlife has been suggested but the role of mammals in the maintenance of the virus, including the existence of a wildlife maintenance host, remains largely unclear (Evans et al. 2008; Olive, Goodman & Reynes 2012a; Nakounne, Selekon & Morvan 2000; Pourrut et al. 2010). Seroconversion of African buffalo outside of epidemic periods suggests this species may act as a reservoir host (Beechler et al. 2015; LaBeaud et al. 2011). Beechler et al. (2015) concluded that there is likely undetected inter-epidemic cycling of RVF within African buffalo populations at a very low rate. Olive, Goodman and Reynes (2012a) concluded that wild ruminants, especially African buffalo, and some domestic ruminants may be involved in the maintenance of RVFV however, during the inter-epidemic period, circulation of RVFV among these animals probably leads to dead-end infection. A recent review considered a rodent or

chiropteran reservoir as a maintenance host (Olive, Goodman & Reynes 2012b). No long-term mammalian carriers of RVFV are known and a definitive mammalian reservoir is yet to be found (Chevalier et al. 2010; LaBeaud et al. 2011; Manore & Beechler 2015; Olive, Goodman & Reynes 2012a).

RVF virus can be found in aborted fetuses, faeces, milk, and other secretions (Beechler et al. 2015; Radostits et al. 2007). Exposure to infected tissues and secretions may infect animals, however this is only of epidemiological relevance in transmission to humans (Archer et al. 2013; Chevalier et al. 2010; Mansfield 2015; Olive, Goodman & Reynes 2012a; Pepin et al. 2010).

The reported incubation period in domestic livestock, primates and rodents ranges from a few hours to a few days, dependant on multiple factors including the inoculation dose and route, virus strain, and age and species of animal (Pepin et al. 2010). Viraemia is typically brief, only 2 to 7 days.

RVF virus is excreted in saliva, nasal, rectal and vaginal discharges, and possibly milk, during the viraemic phase (Saber et al. 1984; Swanepoel & Coetzer 2004). The shedding of RVFV in semen has not been demonstrated in domestic livestock (Radostits et al. 2007; Swanepoel & Coetzer 2004), however inflammatory cells and leukocytes, which potentially can be infected with RVF virus, may be secreted in seminal fluids for the viraemic period or longer. Semen is considered a risk for infection and transmission (Animal Health Australia 2016b; Thibier & Guerin 2000). RVFV can be transmitted vertically in sheep in the absence of detectable maternal viraemia (Antonis et al. 2013).

The OIE Code defines the incubation period for RVF in animals as 14 days for epidemiological purposes (OIE 2018c).

### **Clinical signs**

Clinical signs of RVF are variable. Some animals, especially in endemic areas, may be asymptomatic whilst others are affected by severe clinical disease including abortions and mortalities. Clinically affected animals may show a range of nonspecific signs that may include fever, conjunctivitis, nasal discharge, weakness, lymphadenitis, anorexia, decreased milk yields, malformed newborns, abortion and mortalities. A hallmark of RVF epidemics is a large number of abortions among ruminants concurrent with human disease (primarily flu-like symptoms) (Pepin et al. 2010). Newborn and young ruminants are the most susceptible with mortalities reaching 100 per cent (Chevalier et al. 2010). Adult mortalities may reach up to 30 per cent (Ganter 2015; Gerdes 2004). There are limited descriptions of clinical disease in wild ruminants however signs appear to be similar to those seen in domestic livestock (Olive, Goodman & Reynes 2012a).

### **Diagnosis**

Diagnosis of RVF is based on isolation of virus, demonstration of viral antigens and serological methods and should involve a combination of different diagnostic approaches (OIE 2013d; Pepin et al. 2010).

Serological diagnosis is most commonly by the Virus Neutralisation Assay (VNA) or enzyme-linked immunosorbent assays (ELISA). The VNA is the gold standard for international trade and is generally used for vaccine potency determination (OIE 2013d). ELISAs are also in wide use by OIE reference laboratories and are available commercially. Limitation of ELISAs include the

transient nature of IgM antibodies and, that while detection of IgM suggests a current or recent infection, the detection of IgG cannot distinguish between past and current infection unless paired serum samples are analysed (Pepin et al. 2010).

Molecular diagnostics can detect viral RNA in blood during the brief, acute (febrile) phase of the disease when high levels of viraemia occur in animals and humans (Ikegami 2012; Mansfield 2015). Recent studies have shown consistently high sensitivity and specificity for the diagnosis of RVFV using the quantitative RT-PCR protocol (Escadafal et al. 2013; Odendaal et al. 2014).

Virus culture and isolation can be performed on whole blood or serum from the acute stage of disease or on tissues collected at post-mortem such as spleen or abortion products (Anderson & Rowe 1998b; OIE 2013d). Histopathology and immune-histochemical labelling have also been used (Odendaal et al. 2014).

#### **4.19.3 Current biosecurity measures**

Australia's current biosecurity measures for RVF include country freedom. The OIE Code recommendations include country or zone freedom and vaccination (OIE 2018c).

#### **4.19.4 Risk review**

RVF is not present in approved countries and it is not present in Australia.

The following key points are relevant to the biosecurity risk of RVF in zoo bovidae:

- RVF has a wide host range including ruminants, rodents, humans and other primates.
- RVF occurs in cyclical outbreaks at approximately 5-15 year intervals in endemic areas and may cause significant disease, with fatalities in livestock and humans.
- Outbreaks of RVF may cause significant disease burden with large numbers of livestock affected and severe economic consequences, disease control costs and lengthy trade restrictions.
- Countries with a history of infection in live animals are very likely to remain infected with RVF virus.
- Mosquitoes are considered the main vectors; infection may also occur through contact with infected materials.
- Maintenance of RVFV during inter-epidemic cycles is not fully understood. It is likely that the virus survives in mosquitoes. A role for ruminants and other mammalian wildlife in the maintenance of RVF during inter-epidemic periods has been suggested but remains unproven. Whilst a maintenance role for African buffalo has been suggested, this zoo bovidae policy specifically excludes species from the tribe *Bovini* (which includes buffalo species).
- No RVF carrier-state has been identified in mammals.
- The viraemic period is short in both animals and humans.
- Countries with a history of infection in live animals typically remain infected with RVF virus.
- Australia has competent mosquito vectors for RVF transmission and should RVF enter, spread and establish in Australia, eradication is likely to be challenging.

- There is a risk of transmission via both live animals and semen.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.19.5 Conclusion

Based on the preceding information and in accordance with recommendations in the OIE Code (2016f), risk management measures for zoo bovidae are warranted.

Australia's proposed biosecurity measures for RVF for live zoo bovidae are:

- For 90 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of RVF has occurred during the previous ten years and the disease is compulsorily notifiable.

Australia's proposed biosecurity measures for RVF for zoo bovidae semen are:

- For 90 days immediately prior to collection the animal was continuously resident in a country where no clinical, epidemiological or other evidence of RVF has occurred during the previous ten years and the disease is compulsorily notifiable.

## 4.20 Schmallenberg virus

### 4.20.1 Background

Schmallenberg virus (SBV) is a virus from the family *Bunyaviridae*, genus *Orthobunyavirus* and falls within the Simbu antigenic group (Hubálek, Rudolf & Nowotny 2014). It is closely related to Shamonda, Aino and Akabane viruses. Disease caused by SBV was first recognised in dairy cows in Europe in 2011 and spread rapidly throughout Europe thereafter (Wuethrich et al. 2016). The emergence of SBV in Europe resulted in international trade restrictions of significant cost (Stuchin, Machalaba & Karesh 2016). After the initial epidemic, case numbers reduced for a time and now seem to occur in cyclical outbreaks, similar to other arboviruses (Gache et al. 2017; Kameke et al. 2016). SBV affects domestic ruminants, and evidence of infection has also been found in free-ranging and captive non-domestic ruminants in Europe.

No zoonotic risk from SBV has been identified (Ducomble et al. 2012; European Food Safety 2014).

SBV is not an OIE-listed disease. SBV is not present in Australia and is not a nationally notifiable disease (Department of Agriculture and Water Resources 2016).

### 4.20.2 Technical information

#### Epidemiology

The Simbu serogroup is composed of several arthropod-borne viruses transmitted by biting midges (*Culicoides* spp.) and mosquitos (Elbers et al. 2012). Infection with these viruses is usually clinically inapparent in non-pregnant ruminants; infection in pregnant dams can lead to central nervous system and musculoskeletal malformations in the foetus. The epidemiology of SBV is considered similar to Akabane virus (AKAV) (a Simbu virus) and bluetongue virus (BTV).

In SBV, infection occurs through the bites of midges (*Culicoides* spp.); subsequent transplacental transmission can cause teratogenic effects on foetuses (Beer, Conraths & Van der Poel 2013;

Esteves et al. 2016; Sedda & Rogers 2013). Studies suggest that SBV replicates within *C. obsoletus* group midges, which are the most important vector (Esteves et al. 2016; Rasmussen 2012). Although there is no evidence that other insect genera are involved in transmission of SBV, mechanical transmission might occur through the bites of other haematophagous insects. There is no evidence of horizontal transmission between infected animals (Claine et al. 2015; Wernike et al. 2012). Reservoir hosts may exist outside of the commonly affected domestic livestock species (Mouchantat et al. 2015), however, a carrier state has not been identified for SBV and ruminants do not become long-term carriers of the closely related AKAV (Spickler 2016a). Australia has many *Culicoides* spp. and there is a risk of establishment and spread of SBV. Introduction of SBV would likely result in a viral distribution similar to AKAV, Aino or bluetongue viruses.

Transmission of SBV is dependent on vector abundance and the availability of susceptible hosts, and is influenced by climatic conditions, wind patterns and vector feeding preferences (Bessell et al. 2014; Sedda & Rogers 2013). Where conditions are favourable, SBV spreads rapidly, producing seroprevalences of over 90 per cent in the majority of cattle herds after large outbreaks (Elbers et al. 2012; Steukers et al. 2012; Wuethrich et al. 2016). As seroprevalence in the host population increases, the risk of outbreak decreases significantly (Veldhuis et al. 2017). Over time, herd immunity declines and the risk of new outbreaks increases (Collins et al. 2016; Méroc et al. 2013; Veldhuis et al. 2016). The re-circulation of SBV in Belgium (2012) and Germany (2014), suggests the disease is consistently active at a low level even when herd seropositivity is high (Tarlinton, Daly & Kydd 2013). In this situation, outbreaks and epidemics are only expected after alteration to the endemic cycle. Most transmission occurs during warmer weather, however transmission may occur during any season if vector activity is present (Wernike et al. 2013a). SBV is known to overwinter without the active vector, possibly due to a sylvatic cycle, long term survival of virus in vector populations, or transovarial transmission (Collins et al. 2016; Poskin et al. 2016).

Movement of infected livestock may result in new foci of infection and outbreaks in distant locations. In Poland, two separate outbreaks coincided with the introduction of French bulls (Larska, Kesik-Maliszewska & Kuta 2014). Animal transport or a failure to detect new infections in intermediary regions may explain the appearance of SBV at locations in Italy, Spain and Finland, 500 to 1000 kilometres from known outbreak locations (Afonso & Conraths 2014).

The incubation and viraemic periods are short (one to five days and five to six days respectively) in adult animals (Hoffmann et al. 2012), although there is a report of viraemia lasting up to 14 days in natural infection (Claine et al. 2015). Seroconversion occurs 10 to 14 days post-infection (Hechinger, Wernike & Beer 2013; Poskin et al. 2015). SBV antibodies are considered protective and thus disease is only expected in naïve animals or those that have not sero-converted. Maternal antibodies in neonatal livestock may last 4 to 6 months (Claine et al. 2015; Elbers, Stockhofe-Zurwieden & van der Poel 2014). SBV has been detected in tissues (but not blood) of clinically affected newborn calves, kids and lambs (European Food Safety Authority 2014). Natural infection of non-pregnant animals is considered advantageous as these animals develop a strong immunity to SBV that will protect an ensuing pregnancy and provide the neonate with its own maternal antibodies.

Immunity developed after natural exposure is typically stronger than that from vaccination (Wernike et al. 2013b). In cattle, protective immunity following natural SBV infection has been

reported to persist for at least 2 years (Claine et al. 2015; Elbers, Stockhofe-Zurwieden & van der Poel 2014; Meroc et al. 2015) and in sheep for at least 15 months (Poskin et al. 2015). In zoo ruminants there is evidence for antibody persistence of at least one year in a sable antelope (*Hippotragus niger niger*) and a bharal (*Pseudois nayaur*) (Laloy et al. 2016). Molenaar (2015) documents persistence of high antibody levels of between 6 to 12 months in several zoo species including a greater kudu and a scimitar-horned oryx. The long term protection of antibodies from infection by the related AKAV is well documented (Poskin et al. 2015).

The susceptibility of wild ruminants to SBV is expected to be similar to that of domestic livestock, based on the behaviour of related viruses of the Simbu serogroup (where a number of wildlife and livestock reservoirs exist). Evidence of SBV exposure has been found in both free-ranging and captive non-domestic bovidae in Europe. SBV antibodies have been detected in free-ranging European bison, chamois and mouflon (Larska et al. 2014; Mouchantat et al. 2015; Rossi et al. 2015) as well as a range of cervid species and other taxa. Within European zoos, serological evidence of SBV infection has been detected in the following bovidae species: yak, roan antelope, greater kudu, scimitar-horned oryx, gemsbok (*Oryx gazelle gazelle*), European bison (*Bison bonasus*), sitatunga (*Tragelaphus spekii*), blackbuck and gaur (*Bos gaurus*). Serological exposure has also been detected in a range of other zoo herbivores including deer, camelids and elephants (Molenaar et al. 2015).

There is no evidence of a significant sylvatic cycle involving wild ruminants. The virus has primarily been recovered from domestic cattle, sheep and goats by either PCR or virus isolation but isolated reports of recovery exist from bison, roe deer, mouflon and a dog (Kauffold, Vahlenkamp & Hoopsa 2014; OIE 2017z). Any role of wildlife and zoo bovidae is likely to be determined by multiple factors including density of host and insect vector, proximity to livestock and climatic conditions, rather than species-specific factors (Tarlinton, Daly & Kydd 2013). Seroprevalence in wild ruminants in Poland was found to be lower in regions with low domestic stock density, despite wild ruminant populations being more numerous in this region (Larska et al. 2014). The effects of infection in wildlife have been difficult to investigate. The seroprevalence among wildlife ruminant species is relatively high (>30-50 per cent across multiple species), but significantly lower than that in livestock (Diaz et al. 2015; Lievaart-Peterson et al. 2015; Rossi et al. 2015).

Infection and seroconversion of zoo collections may follow a similar pattern to that of livestock (Molenaar 2015). Molenaar (2015) notes that all their positive cases in the study were in animals housed near buildings or that shelter under bushes or trees; most species in the open-range, wide open space with limited housing and vegetation were seronegative. Foetal malformations have not been reported in European wild ruminants or boar (Barlow et al. 2013; Linden et al. 2012; Mouchantat et al. 2015). The potential role of wild ruminants in the epidemiology of the disease remains under investigation.

Although serological evidence of exposure to SBV has been documented in a range of bovidae in European zoos, there was no confirmed disease associated with seroconversion in these animals (Laloy et al. 2016; Molenaar et al. 2015). Viraemia was detected in a blue wildebeest at a Netherlands zoo (Laloy et al. 2016). These studies suggest that exposure to SBV may be expected by captive zoo animals in both urban and rural areas.

SBV may be shed in bull semen for at least three months post-infection (Hoffmann, Schulz & Beer 2014; Pawaiya & Gupta 2013; Ponsart et al. 2014). SBV RNA can be detected for several weeks in different tissues such as lymphatic organs (especially mesenteric lymph nodes), and the spleen (Hechinger, Wernike & Beer 2013; Poskin et al. 2015; Van der Poel et al. 2014). The infectivity via inoculation of SBV-containing semen has been demonstrated in several experimental studies and semen may remain infectious after the host has seroconverted (Doceul et al. 2013; Ponsart et al. 2014). However, the venereal transmission of SBV and actual risk of this pathway itself has not been demonstrated. It has been recommended that semen batches from infected bulls are tested using PCR, unless produced by an animal that remains SBV-specific antibody negative at least 28 days after collection (Schulz et al. 2014; Van der Poel et al. 2014). The method of PCR and its preparation can significantly alter test sensitivity (Schulz et al. 2015).

### **Clinical signs**

In many cases, adult ruminants display no clinical signs. When present, signs in cattle include fever, anorexia, diarrhoea and reduced milk yield. Clinical signs are generally absent in goats and sheep (Claine et al. 2015). When the dam is infected during early to mid-pregnancy, SBV is often associated with aborted and stillborn offspring, and congenital musculoskeletal and neural malformations leading to death shortly after birth (European Food Safety Authority 2014).

Clinical signs relating to SBV have only been observed in domestic ruminants (cattle, sheep, and goats) and bison but have not been reported in other non-domestic or zoo bovidae (Claine et al. 2015; OIE 2017z).

### **Diagnosis**

Diagnosis of SBV infection is via PCR (during the short viraemic period) and serological tests including virus neutralisation and indirect and competitive ELISA (Bréard et al. 2013; Claine et al. 2015). The ELISA may cross-react with other viruses from the Simbu serogroup (Claine et al. 2015). The competitive ELISA was found to be a reliable test for non-domestic and zoo bovidae (Bréard et al. 2013; Molenaar et al. 2015; Mouchantat et al. 2015), but may have lower sensitivity and specificity than the VNT (Claine et al. 2015; Laloy et al. 2014; Poskin et al. 2015). At lower prevalence levels, the VNT may be used in series with the ELISA, due to the lower positive predictive value of the ELISA in this scenario (Laloy et al. 2014) and the high sensitivity and specificity of the VNT (Stokes, Baylis & Duncan 2016).

### **Prevention**

There are two commercially available, inactivated SBV vaccines registered in Europe for use in domestic bovidae: Bovilis SBV (MSD Animal Health) and SBVvax (Merial). Vaccination induces protective immunity in cattle and sheep, although the duration is yet to be fully quantified (Claine et al. 2015; Poskin et al. 2015; Wernike et al. 2013b). Sheep require a single injection but cattle require two injections, four weeks apart. Vaccination against other Simbu group viruses does not provide cross-protection for SBV (Hechinger, Wernike & Beer 2013). Vaccination has played a major role in controlling other insect-transmitted viruses in Europe, such as BTV and lumpy skin disease. The use of SBV vaccines in zoo bovidae has not been reported.

Risk management may include translocation of animals during colder months when *Culicoides* spp. activity is reduced or absent and thus transmission events lower. This is similar to the principle of designating areas 'seasonally free' from BTV based on epidemiological surveillance.

Direct management of *Culicoides* spp. vectors, for example, by use of insect traps, may form part of a control strategy, although the effectiveness of such methods may be difficult to assess.

#### 4.20.3 Current biosecurity measures

There are no current biosecurity measures for SBV in live animals. Current measures for cattle semen include country freedom and selection of donors based on their serological status.

#### 4.20.4 Risk review

SBV is present in approved countries. It is not present in Australia and is not a nationally notifiable animal disease. If introduced to Australia it could cause adverse effects.

The following key points are relevant to the biosecurity risk of SBV in non-domestic zoo bovidae:

- First recognised in 2011, SBV is a newly emerged disease that has affected livestock throughout Europe. It appears to have similar epidemiology to closely related viruses such as AKAV and Aino virus. Its emergence resulted in significant economic losses.
- *Culicoides* spp. are the most important insect vectors, although other haematophagous insects could have a mechanical role in transmission. Vertical transmission may occur via the placenta.
- Other methods of transmission have not been identified for SBV and are not known to occur in related viruses.
- The viraemic period in livestock is short.
- Antibodies to SBV are considered protective. The duration of antibody persistence in domestic livestock ranges from one to two years. There is limited information for other bovidae, but persistence, where reported, is similar to domestic livestock.
- Antibodies have been detected in a wide range of bovidae species, both in the wild and in European zoos, however clinical signs attributed to SBV have not been documented in these species.
- Viraemia has been reported in a single zoo bovid.
- The epidemiological role of non-domestic bovids is unknown and is still under study. Zoo collections generally have low stocking densities which reduces the opportunity for vector-based disease spread.
- Important factors for the spread of SBV and related arboviruses are the density of insect vectors, presence of both viraemic and naïve hosts, and favourable climatic conditions for vectors.
- Given the presence of competent *Culicoides* spp. in parts of Australia, there is a risk of entry, establishment and spread of SBV. SBV would be likely to establish permanently in regions of Australia and cause an ongoing disease burden to livestock holdings.
- Commercial vaccinations induce a protective immunity in sheep and cattle. The same vaccination preparations are used on both species.
- The use of vaccination has not been reported in zoo bovids. However, a full protective effect across a *Caprinae* subfamily species and *Bovini* tribe species, in addition to the protective

effect of antibodies themselves suggests its use may be effective in other *Bovidae* family species.

- SBV may be shed in bull semen for at least three months post-infection.
- PCR testing of semen from donors is recommended unless the donor remains SBV-antibody negative for at least 28 days after collection.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### **4.20.5 Conclusion**

Accordingly and based on the preceding information, risk management measures are warranted.

Australia's proposed biosecurity measures for SBV for live zoo bovids are:

##### **Option ONE**

- For 30 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of SBV has previously occurred and the disease is compulsorily notifiable.

##### **Option TWO**

- The animal tested seropositive to SBV between 10 and 90 days immediately prior to export, by a virus neutralisation test or approved ELISA.

##### **Option THREE**

- The animal was vaccinated against SBV using an approved, commercially available inactivated vaccine during the 180 days immediately prior to export. Vaccination must be completed before entering pre-export quarantine (PEQ). The vaccination regime must follow the commercial regime for cattle (two doses given 4 weeks apart).

Australia's proposed biosecurity measures for SBV for zoo bovid semen are:

##### **Option ONE**

- For 30 days immediately prior to collection the donor was continuously resident and in a country where no clinical, epidemiological or other evidence of SBV has previously occurred and the disease is compulsorily notifiable.

##### **Option TWO**

- The animal tested seronegative to SBV between 28 and 90 days after semen collection, by an approved virus neutralisation test or approved ELISA.

##### **Option THREE**

- The semen for export was tested by an approved qRT-PCR method, with negative results.

##### **Option FOUR**

- The semen was collected before 1 June 2011.

## 4.21 Screwworm fly myiasis

### 4.21.1 General information and epidemiology

Two species of flies cause screwworm fly myiasis—New World screwworm, *Cochliomyia hominivorax* and Old World screwworm, *Chrysomya bezziana*. Both species are members of the family *Calliphoridae*, subfamily *Chrysomyinae*. Screwworms are the larvae of flies that feed on living flesh. ‘New World’ refers to the Americas and ‘Old World’ to Africa, Asia and Europe. *C. hominivorax* has never been reported in Canada and was eradicated from the United States in the 1980s (Hall 1991). An incursion of New World screwworm occurred in late 2016 in Key deer (*Odocoileus virginianus clavium*) located in Florida, United States. The United States declared freedom again in March 2017 after a successful eradication program (USDA 2017). An incursion of Old World screwworm occurred in 2017 in Singapore, found in zoo animals (OIE 2017y). *C. bezziana* has not been reported in European countries or New Zealand. However, *C. bezziana* is endemic in Malaysia (OIE 2013b). In Hong Kong, *C. bezziana* myiasis is present, and was thought to have been introduced from southern China (Chemonges-Nielsen 2003; FEHD 2011).

New World screwworm and Old World screwworm are multiple species OIE-listed diseases (OIE 2018c). They are absent from Australia and are nationally notifiable animal diseases (Department of Agriculture and Water Resources 2016).

Both species of flies can affect all warm-blooded animals, including humans. Infections in birds are rare (Spickler 2012). *C. hominivorax* and *C. bezziana* have similar climatic requirements. Australia is the only continent with a suitable climate where screwworm fly has not established.

Screwworm flies lay eggs in the open wounds or orifices of warm blooded mammals. The larvae feed on the wounds causing extensive tissue damage and can result in high morbidity and mortality rates within the host population (Allan 2001). The flies prefer warm, moist conditions with temperatures of 16-30 °C and larvae usually spend four to seven days on the host before dropping off to pupate within the soil (Rodriguez & Raphael 2008). At tropical temperatures maggots may hatch from the eggs within 24 hours of being laid. The life span of a male fly is up to 14 days. A ten day lifespan is common for a female but some may live up to 30 days or more. The life cycle of a single fly may vary with temperature; at tropical temperatures it may be less than 21 days whilst at low temperatures maturation may take two to three months (Spickler 2016d).

Semen is not a risk material for screwworm fly.

Identification of adult flies confirms the presence of screwworm fly in a region, but identification of larvae from clinical cases is required to confirm individual animal infection.

An incursion of screwworm fly is considered most likely to occur on the north coast, eastern seaboard or south-east coast of Australia. Modelling suggests that climatic conditions would limit screwworm fly survival to the northern areas of Australia should it be introduced and become established (Fruean & East 2014).

*C. hominivorax* was eradicated from the southern United States and Mexico by treating wounds of all infected animals with insecticidal smears and releasing billions of sterile flies in a program known as the Sterile Insect technique (SIT). Trials have shown that SIT can also be used to control *C. bezziana*, which poses the greater risk to Australia (Spradbery et al. 1989). As per the

AUSVETPLAN manual, Australia's response to screwworm fly incursion would include initial suppression of screwworm fly populations through a variety of management strategies, followed by SIT, the only proven method of eradication (Animal Health Australia 2007). Treatment of individual animals for screwworm include physical debridement, application of topical insecticide to kill remaining larvae and prophylactic treatment with long-acting insecticides.

Australia's previous biosecurity measures for screwworm fly myiasis in other zoo species include country freedom. The OIE Code recommendations include country freedom or inspection for external parasites, treatment of infested wounds and prophylactic treatment for domestic and wild mammals (OIE 2018c).

#### 4.21.2 Risk review

Screwworm fly is present in Singapore but not other approved countries. It is absent from Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of screwworm fly in non-domestic zoo bovidae:

- All warm blooded mammals may be hosts for screwworm.
- High morbidity and mortality may occur in host populations.
- The full life cycle of a fly may vary with temperature. In hotter temperatures it may be less than 21 days.
- Screw-worm-flies lay eggs in the wounds or orifices of warm-blooded animals. Larvae eat living tissue for four to seven days before dropping off the host to pupate in the soil or substrate.
- Semen is not a risk material.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.21.3 Conclusion

*C. hominivorax* and *C. bezziana* are not present in approved countries (other than Singapore) and are not present in Australia. Accordingly and based on the preceding information, risk management measures are warranted for live animals. Specific risk management measures are not warranted for zoo bovid semen.

Australia's proposed biosecurity measures for screwworm fly myiasis for live zoo bovidae are:

- For 60 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of screw-worm-fly (*Cochliomyia hominivorax* or *Chrysomya bezziana*) myiasis occurred during the previous 12 months and the disease is compulsorily notifiable.

## 4.22 Surra

### 4.22.1 Background

Surra is a blood-born protozoan parasitic disease caused by the flagellate protozoan *Trypanosoma evansi*. Its principal hosts vary by geographic region, with camels being the most important in Africa, capybara (*Hydrochoerus hydrochaeris*) and coati (*Nasua* spp. and *Nasuella* spp.) in South America, and buffalo, cattle and pigs in Asia (Eyob & Matios 2013). It can cause disease in many domesticated mammals, wild animals and some zoo species (Desquesnes et al. 2013b). Surra is most severe in donkeys, mules, deer, camels, llamas, cats and dogs (Geering, Forman & Nunn 1995). Surra has been described in domestic cattle, sheep, goats and water buffalo, but there is limited information in other non-domestic bovidae (Desquesnes et al. 2013a).

*T. evansi* is the most widely distributed pathogenic trypanosome and is found in tropical and sub-tropical parts of the world including in Africa north of the tsetse fly belt, Asia, Central and South America and the Middle East (Desquesnes et al. 2013b; Radostits et al. 2007a).

Only a handful of confirmed cases have been reported in humans, despite hundreds of millions of exposures to the infective agent and the disease is not considered zoonotic (Animal Health Australia 2005b; Spickler 2009b).

Surra is an OIE-listed disease (OIE 2018c). It is absent from Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016). Surra is a Category 4 disease in the EADRA and an uncontrolled outbreak would cause production losses in the beef and dairy industries and ongoing costs in the horse industry.

### 4.22.2 Technical information

#### Epidemiology

Although almost all mammals are considered susceptible to infection by *T. evansi*, only some are considered to be significant in the epidemiology of the disease. The principal mammalian hosts vary from one geographical region to another (Desquesnes et al. 2013b). In the domestic bovidae, *T. evansi* is considered pathogenic in cattle and can also infect sheep and goats. It is considered non-pathogenic in the African buffalo.

In Africa, camels are the main host for *T. evansi*, whereas in South America capybara and coati are considered important reservoirs. The significance of other wild species as reservoirs is unknown (Animal Health Australia 2005b). In Asia, *T. evansi* is a major parasite for water buffalo (*Bubalus bubalis*) and in some countries is also considered an economically important disease in cattle, pigs, and goats (Desquesnes et al. 2013a). Cattle and buffalo in Asia are more susceptible to development of disease from *T. evansi* infection than in Africa or Latin America, with surveys reporting figures up to 50 per cent prevalence in Asian populations (Manuel, Mikami & Hirumi 1998; Thu et al. 1998; Tuntasuvan et al. 1998). *T. evansi* has been found in the saiga antelope (*Saiga tatarica*) and wild sheep (*Ovis ammon*) (Desquesnes et al. 2013b). There is no indication that these species are important in the epidemiology of the disease.

*T. evansi* is transmitted mechanically, primarily by the horse fly (*Tabanus* spp.) and to a lesser degree by the stable fly (*Stomoxys* spp.) (Geering, Forman & Nunn 1995). The organism does not survive long on biting fly mouthparts and is very unlikely to be present after 24 hours, especially if successive feeds have occurred (Animal Health Australia 2005b). Replication occurs in the

mammalian host with no intermediate stage. Transmission by other biting insects (e.g. Culicidae mosquitoes, Ceratopogonidae midges) has been reported or suspected and may contribute to local spread (Spickler 2009b). Transmission via consumption of a fresh carcass or infected milk and by the venereal route may also be possible (Spickler 2009b; Williams 2003).

The incubation period of surra in equids is usually one to two weeks, but can be up to 60 days (Geering, Forman & Nunn 1995). Little is known about the progression of surra in bovidae, however in cattle and buffalo, infection is frequently chronic in nature (Geering, Forman & Nunn 1995; Muraleedharan 2015). Stressors including translocation, restraint and physical effort can trigger clinical signs, with resulting morbidity, mortality or disease propagation (Manuel, Mikami & Hirumi 1998; Thu et al. 1998). Introduction of the parasite to naïve geographic regions is generally characterised by a high prevalence of infection, morbidity and mortality (Spickler 2009b; Eyob & Matios 2013).

Potential tabanid vectors and reservoir hosts for trypanosomes are present in Australia (Animal Health Australia 2005b; Reid 2002). Australia contains several wild species, including camels, feral pigs, and dingoes, that would likely be suitable reservoirs (Desquesnes et al. 2013b). Experimental studies have shown that two species of wallaby, the agile wallaby (*Macropus agilis*) and the dusky pademelon (*Thylogale brunii*) are susceptible to *T. evansi* (Reid et al. 2001). The only known incursion of *T. evansi* into Australia was in 1907, in camels imported from India. The incursion was eradicated by rapid identification and slaughter of infected animals (Reid 2002). France (2006) and Spain (2008) have also experienced and subsequently eradicated outbreaks of surra.

*T. evansi* is a fragile organism with poor environmental survival (Geering, Forman & Nunn 1995), however it is routinely found in cryopreserved samples in laboratories and is considered highly likely to survive in frozen semen (Holland et al. 2001). Transplacental transmission has been demonstrated in ruminants and donkeys (Spickler 2009b). The Williams report (2003) references a study reporting sexual transmission of *T. evansi* and concluded that semen was a risk and should not be used. However, whilst some studies demonstrate deleterious effects on fertility, venereal transmission is not demonstrated and not recognised in the epidemiology of surra (Dargantes et al. 2005; Ogundele et al. 2016; Wada et al 2016a; Wada et al 2016b).

### **Clinical signs**

Infection may be subclinical or result in signs ranging from chronic wasting to acute death. Clinical signs of acute disease include pyrexia, depression, weakness and oedema. Death occurs within a few weeks. Chronic surra is characterised by intermittent episodes of pyrexia, anaemia, dependent oedema and emaciation (OIE 2012c).

### **Diagnosis**

A definitive diagnosis requires laboratory methods to detect the parasite. When parasitaemia is high, examination of blood films or lymph node materials may reveal the trypanosomes. Blood films from peripheral veins such as the ear or tail are recommended over jugular samples (OIE 2012c). In more chronic cases and in hosts where parasitaemia is usually low, methods of parasite concentration are required, such as the Haematocrit centrifuge technique (HCT, Wood Method) (OIE 2012c; OIE 2018b). Direct inoculation of rodents may also be utilised if necessary (Manuel, Mikami & Hirumi 1998). PCR testing is less sensitive in host species which develop low levels of parasitaemia, such as bovidae. A range of serological tests are available, although most have not been validated for bovidae species other than cattle and water buffalo (Desquesnes et

al. 2001; Geysen, Delespaulx & Geerts 2003; Holland et al. 2001; OIE 2012c; Reid & Copeman 2002; Reid, Husein & Copeman 2001; Verloo et al. 2000).

### Prevention

Geographical spread of *T. evansi* is related to the movements of infected animals and dissemination of infection by mechanical vectors (Desquesnes et al. 2013a). Prevention relies on sourcing from populations known to be free of the disease, or otherwise excluded by quarantine and testing.

### 4.22.3 Current biosecurity measures

Australia's biosecurity measures for surra in zoo perissodactyls include country freedom. There are no recommendations in the OIE Code. A risk assessment was undertaken for the horse IRA and risk management measures for equids were recommended including country freedom, or premises freedom, PEQ, diagnostic testing, preventative treatment against biting flies and PAQ (Biosecurity Australia 2010).

### 4.22.4 Risk review

Surra is not present in approved countries. Surra is not present in Australia and is a nationally notifiable disease.

The following key points are relevant to the biosecurity risk of Surra in zoo bovidae:

- Transmission of *T. evansi* is mechanical, primarily by biting tabanid flies.
- Incubation is commonly one to two weeks in equids, though may be as long as 60 days. Incubation periods are unknown in bovidae, however, in buffalo and domestic cattle, infection is often chronic.
- A wide range of mammalian species may be infected, however certain species are recognised as more epidemiologically important than others and this varies by geographic region.
- *T. evansi* has a wide host range and has been detected in several of the bovidae species covered by this policy. However, there is no evidence they are epidemiologically important.
- Chronic subclinical infection is documented in several species of the *bovini* tribe, but the species are not included in this policy.
- Stress may cause recrudescence of disease in carriers, resulting in possible morbidity and/or mortality.
- Introduction of the parasite into naïve populations is generally characterised by high levels of infection, morbidity and mortality.
- Australia has multiple potential tabanid vectors and wild animal reservoirs that could allow establishment of *T. evansi*.
- An uncontrolled outbreak would cause production losses in the beef and dairy industries and ongoing costs in the horse industry.
- Venereal transmission of surra is not known to be a risk.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.

- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.22.5 Conclusion

Accordingly and based on the preceding information, risk management measures for surra in live animals are warranted. Risk management measures for semen are not warranted.

Australia's proposed biosecurity measures for surra for live zoo bovidae are:

##### Option ONE

- Since birth, the animal was continuously resident in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

##### Option TWO

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

AND

- During pre-export quarantine a blood sample was drawn from *a peripheral vein* of the animal and tested using the Haematocrit centrifuge technique (OIE 2018b)<sup>5</sup>. The test was negative for trypanosomes.

## 4.23 Transmissible spongiform encephalopathies

### 4.23.1 Background

Transmissible spongiform encephalopathies (TSEs) are fatal degenerative neurological diseases affecting a variety of mammalian species. TSEs are caused by prions, which are proteinaceous infectious particles which lack nucleic acid (Prusiner 1982). TSEs behave like infectious diseases but are slow in development once the host is infected. There is no treatment available for TSEs and all are invariably fatal (Wisniewski, Chabalgoity & Goni 2007).

Australia is free from TSEs in animals, and is recognised as meeting OIE requirements for a Bovine Spongiform Encephalopathy (BSE) *Negligible Risk* and scrapie free country. Historically, TSE has been diagnosed in two cheetah (*Acinonyx jubatus jubatus*) and one Asiatic golden cat (*Catopuma temminckii*) housed in Australian zoos. All three cats were born in zoos overseas, in BSE-infected countries during the European epidemic (Peet & Curran 1992; Young & Slocombe 2003). Given the implications of Australia reporting a TSE, this remains an important disease for zoo importations (Animal Health Australia 2015a).

A zoonotic risk for BSE is recognised. The BSE variant affecting humans (Creutzfeldt-Jakob Disease) is nationally notifiable (Health 2009).

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<sup>5</sup> Method detailed by: OIE 2018b, 'Trypanosomosis (tsetse-transmitted)', in *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 2018*, World Organisation for Animal Health, Paris. Available at: [http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.04.17\\_TRYPANOSOMOSIS.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.04.17_TRYPANOSOMOSIS.pdf)

BSE and scrapie are OIE-listed diseases (OIE 2018c). Transmissible Spongiform Encephalopathies are nationally notifiable animal diseases (Department of Agriculture and Water Resources 2016). BSE (any strain) is included as a Category 2 disease in EADRA where a confirmed case of any strain of BSE in Australia could result in serious economic loss, export market disruption, and domestic market disruption.

#### 4.23.2 Technical information

##### Epidemiology

TSEs in animals include scrapie in domestic sheep and goats, transmissible mink encephalopathy in mink (*Mustela vison*); chronic wasting disease (CWD) in wild and farmed cervidae; bovine spongiform encephalopathy (BSE) and related diseases in exotic ungulates and carnivores; and feline spongiform encephalopathy (FSE) in domestic cats. TSEs in humans include kuru, Creutzfeldt-Jakob disease (CJD), Gerstmannn-Staussler-Scheinker syndrome and fatal familial insomnia (Wisniewski, Chabalgoity & Goni 2007). Molecular and biological investigations have supported an etiological link between BSE, FSE, exotic ungulate spongiform encephalopathy and variant Creutzfeldt-Jakob disease (vCJD) of humans; they are assumed to be caused by the same prion (Cook, Richards & Middleton 2010).

Scrapie occurs in ovine and caprine species globally, with the exception of Australia and New Zealand. A disease very similar (and perhaps identical) to scrapie has been reported in two different herds of mouflon (*Ovis mosimon*) in the United Kingdom (Wood, Lund & Done 1992).

Chronic wasting disease (CWD) has been diagnosed in cervids across North America, with rare reports in Asia and Europe (Detwiler & Baylis 2003; Williams & Miller 2002). The CWD strain is different from the prion strain that causes BSE and there is no relationship known between CWD and any other TSE of animals or humans. The origin of CWD is not known, but the disease has emerged significantly in the past 25 years and has been diagnosed in both captive and free-ranging cervids in 24 American states and two Canadian provinces (USGS National Wildlife Health Center 2016). CWD occurs commonly in white-tailed deer (*Odocoileus virginianus*) and mule deer (*O. hemionus*) and less commonly in wapiti (or elk, *Cervus elaphus nelsonii*). A single natural case has been reported in a moose (*Alces alces*) (Baeten et al. 2007). Until recently, reports of CWD were confined to northern America with isolated cases in South Korea, following importation of elk from Canada (Lee et al. 2013). In 2016, CWD emerged in free-ranging reindeer in Norway (Benestad et al. 2016). CWD has not been reported to occur outside cervidae species and no naturally occurring cases have been reported in bovidae.

BSE emerged as a new disease in the United Kingdom (UK) in 1986 following the feeding of cattle with prion-contaminated meat and bone meal products (possibly containing scrapie). The epidemic in the UK arose from the recycling of processed waste from infected cattle as feed to other cattle. Variant CJD (vCJD) developed following entry of very large numbers of BSE-infected cattle into the human food chain. In Europe, including the UK, over 185,000 cases of BSE were reported and it is estimated that four times this number entered the food chain, with up to three million cattle infected. In European countries other than the UK, the combined total of BSE cases was relatively low (Wisniewski, Chabalgoity & Goni 2007). The epidemic peaked in 1992 and numbers of affected animals have declined significantly since the implementation of ruminant feed bans. Although the vast majority of cases were seen in the UK, cattle and feedstuffs exported from the UK resulted in smaller epidemics in other European countries (see OIE website <http://www.oie.int/animal-health-in-the-world/bse-specific-data/>).

Some forms of TSE occur spontaneously, or due to inherited conditions. Occasionally, BSE occurs as an atypical presentation, which is thought to develop spontaneously in older animals (OIE 2016h). It is not known if atypical BSE is transmissible (OIE 2016h).

TSEs are believed to gain entry to the host via ingestion of prions from contaminated tissues and there are no reports of natural transmission other than by ingestion. Exposure to infected tissues or materials by direct inoculation or splashing of mucous membranes are also considered to be risks, albeit low. There is no evidence of horizontal transmission of BSE and little data supporting a role for maternal transmission (Cook, Richards & Middleton 2010; Kirkwood et al. 1992). The ability of prions to transmit across host species is limited, however, once this inherent barrier has been overcome, a novel, stable and distinct pattern of infection can develop in the new host species (Wisniewski, Chabalgoity & Goni 2007). Prions are not uniformly distributed in the tissues of affected individuals and infectivity depends upon the stage of incubation. In general, the highest concentration of prions are seen in neural tissues (including the eye), spinal fluid and lymphoid tissues late in the incubation period and during clinical disease (Animal Health Australia 2015d).

TSEs have been reported in non-domestic bovidae ('exotic ungulate encephalopathy') and occur as a result of infection with the BSE agent (Cook, Richards & Middleton 2010; Cunningham et al. 2004). Non-domestic bovidae species may vary in their susceptibility to BSE. TSEs have been reported in seven species of non-domestic bovidae, captive or born in zoos in the UK, including eland, greater kudu, nyala, scimitar horned oryx, gemsbok, Arabian oryx and bison (Jeffrey & Wells 1988; Williams, Kirkwood & Miller 2001). The cases in zoo ungulates followed exposure of individuals to feeds containing ruminant-derived protein. The number of new cases in zoo ungulates declined once ruminant feed bans were introduced in July 1988. Greater kudu appear to be the most susceptible of the non-domestic bovidae species exposed to BSE (Cunningham et al. 2004; Sigurdson & Miller 2003). The high incidence of BSE in a small herd of kudu held at a zoo in the UK during the endemic BSE period has been reported to be consistent with either a particularly high species susceptibility and / or direct transmission between kudu, however no further cases have occurred in captive kudu since 1992 (Williams, Kirkwood & Miller 2001), reducing the likelihood that direct transmission was responsible. Studies have shown that prions are more widely distributed in infected kudu tissues than in cattle infected with BSE and it has been suggested that infectivity in kudu may differ to that in cattle (Cunningham et al. 2004).

The apparent maximum incubation period of BSE following exposure in cattle is considered to be 28 to 48 months. There are limitations in the ability to determine the incubation period in zoo ungulates, due in part to small sample sizes, but it is believed to be similar to that seen in cattle (Williams, Kirkwood & Miller 2001).

Prions are extremely resistant to autolysis, environmental degradation, and common chemical and physical decontamination methods including autoclaving at conventional times and temperatures (that is, 121°C for 15 min).

The risk of transmission of BSE and scrapie via semen and embryos was assessed during the British BSE outbreak as small or non-existent. This was supported by review of cattle mating records during the British BSE outbreak and experimental observations (Wrathall 1997). There is no evidence of transmission of BSE and related TSEs in semen (Greenlee & Greenlee 2015).

**Clinical signs**

Clinical signs of BSE in zoo ungulates are similar to those seen in affected cattle. They include central nervous system dysfunction such as ataxia, abnormal head and ear posture, tremors, changes in mentation and weight loss. Clinical disease typically progresses over several weeks once signs are evident, but in some cases a more rapid progression over days has been reported (Cook, Richards & Middleton 2010; Williams, Kirkwood & Miller 2001).

**Diagnosis**

Ante mortem diagnosis of BSE and related TSEs is not possible. There may be a high index of suspicion when typical neurological signs are seen in susceptible species during an endemic BSE period, such as that of the early 1990s in the UK. Diagnosis relies on detection of characteristic post mortem changes to neurological tissue (Williams, Kirkwood & Miller 2001). The pattern of brain lesions and the extent of prion amyloid deposition vary within and between affected species (Wisniewski, Chabalgoity & Goni 2007).

**Prevention**

Prevention, control and elimination of BSE focuses on preventing tissues that have the highest risk of causing infection from entering the human or animal food chain. In cattle and other bovidae, this is largely achieved by ensuring that no ruminant carcass material is included in ruminant feeds (Greenlee & Greenlee 2015). In other host species, this may mean ensuring that high-risk carcasses or high-risk cuts of meat are not fed to carnivores, or humans. TSE prions are extremely resistant to inactivation and destruction so complete avoidance of risk material is required.

**4.23.3 Current biosecurity measures**

Australia's current biosecurity measures for TSEs in zoo animals include protocols for post-mortem investigation and the appropriate disposal of zoo animal carcasses. The protocol is detailed by Animal Health Australia (2015d).

Australia operates a nationally integrated program of active surveillance for TSEs, known as the National TSE Surveillance Program (NTSESP), to demonstrate Australia's on-going freedom from BSE and scrapie, and to provide early detection of these diseases if they occur. There is a national protocol for management of risk-animals (imported individuals from susceptible species, which lived in BSE positive countries prior to the introduction of ruminant feed bans). This includes policy for a response to a potential positive TSE diagnosis in an animal within the Australian zoo population (Animal Health Australia 2015d). TSEs potentially affecting zoo animals are not considered contagious, provided affected animals are kept out of the human and animal food chain. There is considered to be negligible risk of spread to in-contact animals and cohorts, or contamination of the environment. Specific management measures are not required for progeny, in-contact animals or animal enclosures, but national guidelines have been developed to minimise any potential risk to humans associated with conducting a post mortem examination (Animal Health Australia 2015d).

The OIE Code recommendations for BSE vary according to the BSE-assessed risk of the exporting country, zone or compartment and include permanent identification of the individual and assurances that the animal has been subjected to a ruminant feed ban its entire life.

**4.23.4 Risk review**

TSEs are present in approved countries and are not present in Australia.

The following key points are relevant to the biosecurity risk of TSEs in zoo bovidae:

- TSEs are nationally notifiable animal diseases.
- Australia is free from TSEs in animals, and is recognised as meeting OIE requirements for a BSE *Negligible Risk* and scrapie free country.
- Countries approved in this policy consist only of nations with Negligible or Controlled BSE risk status (OIE 2018).
- Chronic wasting disease has been reported in cervidae in North America and northern Europe, but has never been reported to occur naturally in bovids.
- Scrapie is present in domestic goats and sheep globally, other than Australia and New Zealand, but has never been reported in the species of bovidae covered by this policy.
- TSEs derived from BSE have been reported in a range of captive zoo bovid species during and following the outbreak of BSE in the UK during the 1990s.
- TSEs are believed to gain entry to the host via ingestion of prions from contaminated tissues, primarily neural tissues in the case of BSE and related TSEs.
- Horizontal transmission is not a feature of BSE. Scant evidence exists for the possibility of vertical transmission.
- TSEs result in 100 per cent fatality and treatment is not possible.
- Diagnosis of TSEs is only possible post mortem.
- TSEs potentially affecting zoo bovidae are not considered contagious, provided affected animals are kept out of the human and animal food chain. There is considered to be negligible risk of spread to in-contact animals and cohorts, or contamination of the environment.
- There is no evidence of transmission of BSE or related TSEs via semen.
- A confirmed case of any strain of BSE in Australia could result in serious economic loss, export market disruption and domestic market disruption.
- There is an established national protocol for the management of potential 'risk' zoo animals.
- Zoo bovids in Australia do not enter the human food chain.
- It is illegal in Australia to feed restricted animal material (which includes zoo animal carcasses or carcass parts) to any ruminant (including zoo animals and domestic livestock).
- Australian protocols exist regarding the management and disposal of the carcasses of imported, potential 'risk' zoo animals.

#### 4.23.5 Conclusion

Accordingly and based on the preceding information, risk management measures are warranted for BSE in live bovidae. Risk management measures for semen, or for other TSE agents in live bovidae are not required.

Australia's proposed risk management measures for BSE for live zoo bovidae are:

- Since birth, the animals for import have only lived in a country (or countries) listed as having a negligible or controlled BSE status by the OIE (for the period of that residency). The countries and periods of residency must be listed on the veterinary health certificate.

## 4.24 Trypanosomosis (tsetse fly associated)

### 4.24.1 Background

Trypanosomes are blood-borne protozoan parasites that cause diseases of livestock and humans and are transmitted by haematophagous arthropods. *Trypanosoma brucei*, *T. congolense*, *T. simiae* and *T. vivax* cause trypanosomiasis, also known as nagana, which results in anaemia, loss of body condition and emaciation in livestock.

Trypanosomes are found in regions of Africa wherever the tsetse fly is endemic between latitude 15 °N and 29 °S, from the southern edge of the Sahara desert to Zimbabwe, Angola and Mozambique. *T. vivax* has spread beyond the 'tsetse fly belt' through mechanical transmission by biting flies and is found in South and Central America and the Caribbean (Spickler 2009a). Tsetse flies are not present in Australia; however, mechanical transmission is possible by biting flies in Australia as there are suitable vectors in the genera *Stomoxys* and *Tabanus* in some regions.

Trypanosomiasis (tsetse-transmitted) is an OIE-listed disease of cattle (OIE 2018c). It is absent from Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016).

### 4.24.2 Technical information

#### Epidemiology

Trypanosomes in Africa that cause disease in livestock (*T. brucei*, *T. congolense* and *T. simiae*) require development in tsetse flies. The parasites are present in the saliva of an infected tsetse fly and are transmitted when the fly bites an animal (Radostits et al. 2007a). *T. vivax* does not require tsetse flies to develop and is found in parts of Africa free or cleared of tsetse flies, and parts of Central and South America (OIE 2018b). In this scenario *T. vivax* replicates in vertebrate hosts only and is mechanically transmitted by biting flies. The level of host parasitaemia, density of suitable biting vectors and time elapsed since vector uptake of sufficient parasites, amongst other factors, determine whether transmission occurs (Osório et al. 2008). Once infected by tsetse associated trypanosomes, a tsetse fly remains infected for life and forms a reservoir of infection.

Trypanosomes occur in the blood of a wide range of wild and domestic hosts. More than 30 species of wild animals, including a wide range of non-domestic bovidae, can become carriers of pathogenic trypanosomes, acting as reservoirs of infection for vectors and livestock (Connor & Van den Bossche 2004). Antelope species shown to be carriers of trypanosomes include, but are not limited to, kob, roan antelope, reedbuck, puku, impala, greater kudu, bushbuck, buffalo, waterbuck, wildebeest and kongoni (Anderson et al. 2011; Kupper, Wolters & Scharf 1983; Truc et al. 1997). These parasites are considered to be well-adapted to wild bovidae and other wild species, and these species are considered highly tolerant to infection (Connor & Van den Bossche 2004).

Tolerant animals usually establish an equilibrium between host immunity and the parasite, though self-cure is sometimes possible. Factors that disrupt host immunity (e.g. malnutrition, concurrent infection) may lead to an episode of clinical signs. A blood-born parasite,

trypanosomes can also be spread by fomites and mechanical vectors including surgical instruments, needles, syringes and various biting flies, though the epidemiological importance of this is unclear (Spickler 2009d; Connor & Van den Bossche 2004). In the absence of the tsetse vector, the translocation of an infected animal into a naïve population is very unlikely to result in even temporary spread of the trypanosomes other than *T. vivax*.

Morbidity and mortality vary with species, breed, trypanosome and other factors, but in general a high morbidity rate occurs; some cattle herds may reach up to 100 per cent mortality within a few months of exposure (Spickler 2009d).

Trypanosomiasis results in serious debilitating effects on male and female fertility that may be long-lasting, including degeneration of reproductive tissues, poor sperm quality and destruction of endocrine function (Anosa & Kaneko 1984; Okubanjo et al. 2015). Transplacental transmission of trypanosomes has been reported in lambs and calves and results in debilitation or death of neonates (Osório et al. 2008; Sekoni 1994). There is experimental evidence of sexual transmission of *T. brucei gambiense* in mice (Biteau et al. 2016). Information on seminal transmission of tsetse-associated trypanosomes was not located in this review.

### **Clinical signs**

Clinical signs of tsetse-transmitted trypanosomiasis include fever, oedema, abortion, decreased fertility, emaciation and, frequently, anaemia (OIE 2018b). The incubation period for trypanosomiasis ranges from four to 20 days in most livestock species (Radostits et al. 2007a) but may be as long as eight weeks (Spickler 2009a). Infections with more virulent isolates have a shorter incubation period (Spickler 2009a). Clinical disease may range from peracute to chronic and may persist for years in cattle (Connor & Van den Bossche 2004).

### **Diagnosis**

Trypanosomiasis can be diagnosed using microscopic examination of blood, with best results obtained using a parasite concentration technique, such as the Haematocrit centrifuge technique (HCT, Woo Method; OIE 2018b). Serological tests include an indirect fluorescent antibody test and an antibody-detection ELISA. PCR may also be used, however it may be necessary to run multiple PCR tests to investigate each species or subspecies of trypanosome potentially involved (OIE 2018b).

### **Prevention**

Other than *T. vivax*, the tsetse-associated trypanosomes are unable to spread or establish without their specific vector. Measures such as eradication of tsetse fly and prophylactic or metaphylactic administration of trypanocidal drugs are used in countries where the disease is endemic or at risk of spread (Spickler 2009d).

## **4.24.3 Current biosecurity measures**

Australia's biosecurity measures for trypanosomiasis (*T. vivax*) in zoo perissodactyls include country freedom. There are no recommendations in the OIE Code.

## **4.24.4 Risk review**

Tsetse-transmitted trypanosomes (*T. brucei*, *T. congolense*, *T. simiae* and *T. vivax*) are not present in approved countries and are not present in Australia.

The following key points are relevant to relevant to the biosecurity risk of tsetse fly-associated trypanosomes in zoo bovidae:

- The disease has a wide host range and causes serious peracute to chronic disease with a poor cure rate. High morbidity is generally expected in susceptible animals and significant mortality rates can occur over time.
- Many African species of zoo bovidae are tolerant carriers and are considered the natural hosts for the parasite. They may succumb to clinical disease and high levels of parasitaemia, particularly if under stress.
- Tsetse flies are not present in Australia, however other competent vectors for *T. vivax* are present.
- The spread of tsetse-associated trypanosomes (other than *T. vivax*) to new hosts is not considered a risk in the absence of the tsetse fly.
- *T. vivax* can spread via competent mechanical vectors but requires replication and sufficient levels of parasitaemia within host animals. Mechanical spread can only occur for short periods.
- Tsetse-associated trypanosomiasis results in debilitating effects on fertility and sexual organs. Placental transmission of tsetse-associated trypanosomes is possible but information on seminal transmission was not located and not known to be a risk.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.

#### 4.24.5 Conclusion

Accordingly and based on the preceding information, risk management measures for *T. vivax* in live animals are warranted. Risk management measures for semen are not warranted.

Australia's proposed biosecurity measures for *T. vivax* for live zoo bovidae are:

##### Option ONE

- Since birth the animal was continuously resident in a country where no clinical, epidemiological or other evidence of trypanosomiasis due to *T. vivax* infection has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

##### Option TWO

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of trypanosomiasis due to *T. vivax* infection has occurred in any species during the previous 2 years and the disease is compulsorily notifiable.

AND

- During pre-export quarantine a blood sample was drawn from a *peripheral vein* of the animal and tested using the Haematocrit centrifuge technique (OIE 2018b). The test was negative for trypanosomes.

## 4.25 Vesicular stomatitis

### 4.25.1 Background

Vesicular stomatitis (VS) is caused by four viruses in the family *Rhabdoviridae*, genus *Vesiculovirus* (New Jersey virus; vesicular stomatitis Indiana virus; vesicular stomatitis Alagoas virus; Cocal virus) (Tordo et al. 2005; Spickler 2016e) and cause disease characterised by vesicular lesions on the tongue, oral mucous membranes, mammary glands, external genitalia and coronary bands (McCluskey & Mumford 2000). Except for its occurrence in horses, vesicular stomatitis is clinically indistinguishable from foot and mouth disease (FMD) (Letchworth, Rodriguez & Del Cbarrera 1999). Any vesicular hoof disease in cloven-hoofed animals should be regarded as suspicious of FMD until proven otherwise. VS and FMD may infect an animal concurrently. The report of any FMD-like disease in Australia, including in a zoo setting, may have an immediate impact on Australia's livestock industry.

The most commonly affected mammalian species include equidae, cattle and pigs. Sheep and goats are more resistant and less often affected by VS (Mare & Mead 2004; Reis et al. 2009). The disease is limited to the Americas (OIE 2015h). Outbreaks in the United States mainly affect horses and cattle, although VS viruses have caused disease in other species including other equids, pigs, llamas and humans. Serological evidence of infection has been found in a wide range of warm-blooded species.

Vesicular stomatitis is zoonotic and can cause an influenza-like illness in humans (Letchworth, Rodriguez & Del Cbarrera 1999).

VS is not an OIE-listed disease (OIE 2018c). It has never been reported in Australia and is a nationally notifiable animal disease (Department of Agriculture and Water Resources 2016). VS is a Category 2 disease under the EADRA with important implications for international trade and of itself can significantly affect production in cattle and performance in horses. Prompt diagnosis is also important to prevent major livestock diseases such as FMD from spreading undetected (Spickler 2016e).

### 4.25.2 Technical information

#### Epidemiology

The epidemiology of VS is not well understood. Viral reservoirs, amplification hosts and natural modes of transmission remain unclear despite extensive study (Cornish et al. 2001; Smith et al. 2011; Trujillo et al. 2010). The role of vertebrates as amplifying and maintenance hosts for VS is not clear, and in most cases, vertebrate amplifying host species have not been identified. During an outbreak, affected animals produce large quantities of saliva, containing high viral loads. Contamination of the animals' environment, including pasture, with saliva facilitates spread of the virus between animals in the herd (Stallknecht et al. 1999). Infective virus may survive in the environment for up to a week (Spickler 2016e).

Insects have been implicated as both mechanical vectors and reservoirs for the virus and have been suggested to play an important role in initial introduction of the virus into a herd, or transmission of the virus across large distances (Drolet, Stuart & Derner 2009; Letchworth 1996). Studies suggest grasshoppers may play a role as amplifying hosts, as well as acting as potential mechanical vectors (Drolet, Stuart & Derner 2009; Nunamaker et al. 2003). Infected insects bite susceptible livestock on the mouth, nostrils or coronary band area and vesicular lesions develop (Mead et al. 2009; Scherer et al. 2007; Smith et al. 2012). By contrast, insect

feeding (and viral inoculation) at the flank, neck, ear and peri-ocular areas does not cause the formation of vesicles but results in development of low levels of neutralising antibody (Mead et al. 2009; Smith et al. 2012). Infection of susceptible hosts appears to be enhanced by minor trauma to skin or mucosal surfaces when compared to inoculation of unbroken surfaces (Howerth et al. 2006).

Direct and indirect contact with saliva and vesicular fluids are considered to be important methods of transmission, as is mechanical transfer via flying insects (Clarke, Stallknecht & Howerth 1996; McCluskey & Mumford 2000; Stallknecht et al. 2001). The virus has been isolated from many insect species such as black flies, *Culicoides*, house flies, eye gnats, mosquitoes and sand flies (Rodriguez 2002). However a role in transmission of the virus has not been confirmed for most of these species. Biological and transovarial transmission has been demonstrated in a limited number of insect vectors (Mead et al. 2009; Mead et al. 2004). Black flies are known to transmit VSV to other black flies during co-feeding on a host. Microscopic vesiculo-pustules containing low levels of virus develop soon after the feeding of infected flies on a mammalian host, which may explain how flies become infected in the absence of visible lesions on the host (Smith et al. 2011).

Serological evidence of infection has been found in many domestic animals and in wildlife species native to the Americas, including dogs, goats, antelope, deer, elk, pronghorn, bighorn sheep, coyote, raccoon, turkey, duck, cotton rats, deer mice and wood rats (McCluskey & Mumford 2000). Deer mice have recently been identified as a potential amplifying host and reservoir (Mesquita et al. 2017). An epidemiological role for non-domestic bovidae, distinct from other cloven-hoof species, is not recognised.

The incubation period is generally short (one to seven days). Virus shedding from an active lesion is thought to continue for up to seven days after formation. Persistent shedding of infective VSV from recovered animals is not known to occur (McCluskey & Mumford 2000). The virus is restricted in distribution to lesions in the skin, anterior alimentary tract mucosa and associated lymph nodes. Large amounts of virus are shed in the copious saliva produced by clinically affected animals. Viral spread appears to stop at draining lymph nodes and there is no development of viraemia in the vast majority of domestic and wild hosts (Mesquita et al. 2017; Reis et al. 2009; Yuill et al. 2001). Morbidity rates vary widely between outbreaks, from 5 per cent of animals to over 90 per cent. Death is occasionally reported but rare (Reis et al. 2009; Spickler 2016e).

Within a herd, disease spreads through direct contact with clinically affected animals or contact with saliva-contaminated fomites (for example, feed, water troughs) (Leder et al. 1983). Visible vesicular lesions are generally considered necessary for efficient animal-to-animal transmission (Reis et al. 2009). Flying insect vectors may acquire infection by feeding on pasture contaminated with VSV-infected saliva. Viral amplification has been detected in some herbivorous insects (Drolet, Stuart & Derner 2009; Nunamaker et al. 2003).

Williams (2003) did not uncover any reports of VSV isolated from, or transmitted by, semen. AQIS previously concluded that risk of transmission of VSV in semen is very low, although equipment such as straws could be contaminated (AQIS 1999). No reports of VSV transmission via semen were located in the literature.

**Clinical signs**

Clinical signs of VS include mild fever, with blister-like lesions on the inside of the mouth, lips, nose, hooves and udder. The blisters break, leaving raw, sore areas. Affected animals often salivate profusely and are unwilling to eat or drink. VS is rarely fatal but mastitis, anorexia, dehydration and weight loss result in significant production losses in cattle (Bridges et al. 1997). Complete clinical recovery typically occurs within 2-3 weeks.

Subclinical infection is common in livestock and reported in many wildlife species (Mare & Mead 2004; Reis et al. 2009).

**Diagnosis**

VS is not easily clinically distinguished from other vesicular diseases, particularly FMD, Seneca valley virus and swine vesicular disease, so laboratory confirmation of diagnosis is crucial. The preferred immunological methods for identifying viral antigens are the ELISA, the CFT and fluorescent antibody staining (OIE 2018c). These tests may not be validated in non-domestic bovidae. Real time-PCR may be more sensitive than virus isolation or CFT (Letchworth 1996).

For serology, the prescribed tests for international trade described in the Manual are C-ELISA, virus neutralisation test and CFT. Antibody can usually be detected between five and eight days post-infection (OIE 2015h). The C-ELISA can detect antibodies five to six days post infection and VNT one to three days later (McCluskey & Mumford 2000).

**Control and prevention**

During an outbreak of VS in livestock, management measures include movement controls, herd isolation and strict hygiene and biosecurity management of all equipment, products and personnel (Spickler 2016e). Stabling animals at night time has been shown to limit spread (Mare & Mead 2004). Numerous disinfectants such as 40 to 70 per cent ethanol and aldehydes are effective against the virus (Spickler 2016e). The control of biting insects and potential migratory grasshopper vectors has been recommended (Drolet, Stuart & Derner 2009).

**4.25.3 Current biosecurity measures**

Australia's biosecurity measures for VS in zoo perissodactyls include premises and regional freedom. The OIE Code previously recommended country freedom or premises freedom and testing for live animals (OIE 2018c). A risk assessment was undertaken for the 2010 Horse IRA and risk management measures were recommended including country freedom or premises freedom, PAQ and diagnostic testing (Biosecurity Australia 2010). Australia's biosecurity measures for semen include country or premises freedom.

**4.25.4 Risk review**

Vesicular stomatitis is present in approved countries. It has never been reported in Australia.

The following key points are relevant to the biosecurity risk of VS in non-domestic zoo bovids:

- VS occurs only in the Americas.
- Clinically, in cloven-hoofed species, VS closely resembles FMD.
- VS has important implications for international trade and of itself can significantly affect production in cattle and performance in horses.

- Efficient animal-to-animal transmission requires the presence of vesicular lesions on the host. The majority of VS cases in wildlife are considered to subclinical.
- Mechanical insect vectors may be involved spreading disease between herds and across distances. Fomite transmission is also a risk pathway.
- There is no evidence of VSV isolation or transmission in semen, however contamination of equipment or semen samples from ruptured vesicles is possible.
- A significant epidemiological role for non-domestic bovidae has not been demonstrated.
- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.

#### 4.25.5 Conclusion

Accordingly, based on the preceding information risk management measures for live animals and their semen are warranted.

Australia's proposed biosecurity measures for VS for live zoo bovidae are:

##### Option ONE

- For 60 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

##### Option TWO

- For 60 days immediately before export the animal was continuously resident on premises in the country of export where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous 90 days and the disease is compulsorily notifiable.

AND

- The animal was held in PEQ for at least 30 days immediately before export.

AND

- The health monitoring program of the exporting zoo includes vesicular stomatitis.

Australia's proposed biosecurity measures for VS for zoo bovid semen are:

- During the 30 days immediately prior to collection of the semen there were no clinical signs or other evidence of vesicular stomatitis at the premises of origin and the disease is compulsorily notifiable.

## 5 Biosecurity measures for the importation of zoo bovids and their semen

The biosecurity measures described in this draft risk review are proposed for the importation of zoo bovids and their semen from approved countries.

There are general risk management measures common to most current import policies for zoo animals, including:

- 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 approved by the department. The residency requirement may be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.
- 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 the disease of biosecurity concern identified in this review (e.g. post-mortem records for deceased animal; disease testing programs; 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 policy.
  - The required outcome of a *Health monitoring program* 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. This underpins official certification.
- The animal must be held in pre-export quarantine for at least 30 days and isolated from all other animals not eligible for export to Australia, during which it is inspected at least daily for signs of disease, treated effectively for internal and external parasites, and tested for diseases in accordance with Australian entry requirements.
- The animal must be transported to a quarantine approved premises in Australia 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 of at least 30 days.
- The pre-export quarantine facility has documented standards of how it will meet all Australian requirements. This may include standard operating procedures, staff training manuals, etc.

- The receiving institution must be approved under relevant Australian state or territory legislation to hold the species being imported.

General risk measures relevant to zoo semen are:

- The donor 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 collection, unless otherwise approved by the department. The residency requirement may be achieved in more than one approved country or holding institution if specifically authorised by the department and the conditions for each country of residence and holding institution must be met.
- 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 the disease of biosecurity concern identified in this review (e.g. post-mortem records for deceased animal; disease testing programs; 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 policy.
  - The required outcome of a *Health monitoring program* 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. This underpins official certification.
- The animal was not under quarantine restriction for the collection period or the 90 days immediately prior.
- A semen collection period (or ‘collection’) starts on the first day semen is collected from the donor and finishes on the last day semen is collected, up to a maximum of 30 days. (A new collection period may begin the day after and is required to meet conditions applicable to that new time frame).
- The receiving institution must be approved under relevant Australian state or territory legislation to hold the imported semen.
- The donor animal(s) showed no signs of infectious or contagious disease *during* the collection period and for the 30 days immediately *after*.

Additional assumptions for zoo bovids and their semen this policy is predicated on are:

- Zoo bovids are sourced from and maintained in facilities that have health monitoring programs including post mortem investigation of deceased animals and are under veterinary supervision.
- Zoo bovids do not mix with open herds of domestic livestock in Australia.
- For the 30 days immediately before export the animal showed no clinical signs or other evidence of the diseases retained for risk review in this policy.

The period of clinical health may be longer under some disease's risk management requirements if used as a specific risk management measure.

The OIE Code recommends periods of premises residency and periods in which premises should remain free from certain diseases ranging from less than 30 days up to two or more years. This applies to the time period before an animal enters pre-export isolation, if applicable.

For disease agents of biosecurity concern that have no recommendations in the OIE Code for the periods of premises residency and/or disease freedom, the periods are based on the epidemiology and information detailed in the relevant sections in Chapter 4.

The biosecurity measures for the importation of zoo bovids and their semen are in Section 5.1. The residency periods and timing of tests in Section 5.1 are based on recommendations in the OIE Code (where applicable) and are amended for consistency and clarity of certification.

The operational and quarantine facilities requirements apply to all zoo bovids and their semen.

### **Explanatory notes**

Where the term 'approved' appears in conditions (e.g. 'an approved test') this refers to the approval of the Department of Agriculture and Water Resources. Where possible, examples have been given of approved tests but importers may need to contact the department to check if other tests have been assessed as 'approved'.

The terminology for quarantine periods has changed since the release of the 2015 Biosecurity Act. The legislation uses the phrase '**pre-arrival quarantine**' for the period in the exporting country (i.e. off-shore) and the phrase '**post-entry quarantine**' for the period in Australia. *However*, for consistency with other zoo policies the traditional terms '**pre-export quarantine**' (PEQ) and '**post-arrival quarantine**' (PAQ) are used in these conditions.

### **Equivalence**

In accordance with Australia's international obligations under the Application of Sanitary and Phytosanitary Measures Agreement 16, the principle of equivalence applies to these biosecurity measures. Where the Competent Authority of an exporting country can objectively demonstrate that alternative biosecurity measure(s) to those required by the Department of Agriculture and Water Resources would provide an equivalent level of sanitary protection, the Department will consider relevant submissions.

Submissions for equivalence must present evidence that the overall biosecurity risk management outcome can be met. The outcomes required for each disease may vary depending on its epidemiology, sensitivity and specificity of available tests, environmental factors in both the exporting country and Australia, and any other relevant matters. For example, a positive test result may not necessarily prevent import of an animal for a disease where a carrier state does not exist and where evidence can be provided that the animal is not currently infected.

Evidence to support a proposal for equivalence may include peer-reviewed literature (e.g. a new diagnostic test), health management records and records of post-mortems examinations that demonstrate freedom from a specific disease.

Proposals for equivalence require assessment on a case by case basis and divert the department's staffing resources from other projects. Therefore, they are usually examined after

the permit-application stage. The time and resources required to assess a proposal for equivalence will vary depending on how far the proposal deviates from general policy. As a guide, the time frame for assessment of equivalence is expected to be several weeks. Large deviations from policy (e.g. where country-freedom has been proposed as the sole risk management measure) may take a prolonged amount of time or may not be possible to assess.

## 5.1 Proposed biosecurity measures for the importation of live zoo bovids from approved countries

### 5.1.1 Documentation

Each zoo bovid must travel with an original international veterinary health certificate that conforms to Article 5.10.2. of the OIE Code, signed by the Official Veterinarian of the country of export.

These biosecurity requirements apply to live zoo bovids.

An **Official Veterinarian** means a veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the OIE Code.

The veterinary health certificate must:

- be written in English and a language understood by the Official Veterinarian of the country of export
- meet the requirements of the '5.1.3 certification' section and state that all the pre-export quarantine requirements have been met
- provide unique identification for each zoo bovid including the International Standards Organisation (ISO) microchip number, a physical description, species, sex and age
- include the name and address of the zoological or wildlife park of origin
- include the name and address of the exporter and importer and identify the import permit against which it was issued.
- include the dates of isolation of each zoo bovid
- include the dates of sampling for any tests required, the type of test used and the test results.

The Official Veterinarian must:

- scan and confirm as well as document the microchip number of each zoo bovid during the pre-export quarantine period and restate the relevant microchip number for each separate veterinary health certificate
- provide a veterinary health certificate that is specific to the group of zoo bovids it covers
- sign, date and stamp (with the stamp of the Veterinary Authority (Official Veterinarian stamp)) each page of the veterinary health certificate and all attached documents (e.g. laboratory reports) that form part of the extended veterinary certification
- endorse each page of copies of supporting documents with date, signature and Official Veterinarian stamp
- record their name, signature and official contact details on the veterinary health certificate.

### 5.1.2 Pre-export quarantine requirements

#### Pre-export quarantine

Any variation from the **pre-export quarantine requirements** must have been specifically authorised by the Department of Agriculture and Water Resources.

#### Location

The pre-export quarantine facility must be located within a government registered, licensed zoological institution or wildlife park that is under veterinary supervision and in which the animals held in the premises are subject to a health monitoring program that is capable of addressing Australia's biosecurity requirements.

- 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 a *Health monitoring program* 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. This underpins official certification.

#### Facilities

- 1) The pre-export quarantine facility must meet the country and premises requirements specified in the certification before export section.
- 2) The entire pre-export quarantine facility must be surrounded by physical and procedural barriers that provide sufficient security to isolate the zoo bovids in pre-export quarantine from all other animals except those that meet all the conditions in these biosecurity measures.
  - a) The required outcome is that quarantined animals are protected from disease transmission, which includes direct contact, direct and indirect aerosol transfer, and fomite transfer (e.g. footwear, feed, water).
- 3) The pre-export quarantine facility must be constructed so that it can be cleaned and disinfectant applied effectively and must be maintained in good order.
- 4) The institution where the pre-export quarantine facility is located must utilise a separate area for the cleaning and disinfection of vehicles for transporting zoo bovids, and facilities for the safe loading and unloading of zoo bovids.
- 5) The institution where the pre-export quarantine facility is located must have facilities for veterinary examination and collection of samples as needed, and must manage biosecurity requirements should it be necessary to utilise these facilities for animals in pre-export quarantine.

#### Operation

- 1) The pre-export quarantine facility must have current approval from the Department of Agriculture and Water Resources and the Veterinary Authority of the exporting country before commencement of pre-export quarantine.
- 2) The Department of Agriculture and Water Resources may audit the approved pre-export quarantine facility.

- 3) All pre-export quarantine operations and procedures must be detailed in Standard Operating Procedures (SOPs), consistent with a risk-based approach and approved by the Department of Agriculture and Water Resources.
- 4) The Official Veterinarian must inspect the pre-export quarantine facility within 72 hours before commencement of pre-export quarantine and must ensure that the facility was cleaned and disinfectant applied to his/her satisfaction.
- 5) Pre-export quarantine must be under the supervision of the Official Veterinarian.
- 6) The pre-export quarantine period commences from the time the last zoo bovid in the export consignment has entered the pre-export quarantine facility and all zoo bovids have been examined by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian.
- 7) All equipment used in feeding, handling and treating zoo bovids in pre-export quarantine must be new or cleaned and disinfected before entry, and must be used only in the facility during pre-export quarantine.
- 8) During pre-export quarantine, the facility must be occupied only by animals of the export consignment.
  - a) If other animals are present then all animals must be able to demonstrate equivalent health status to the export consignment. This includes testing and including the results of these tests to the department to demonstrate this.
- 9) Only personnel specifically authorised by the Official Veterinarian are permitted entry to the pre-export quarantine facility. Details of all visitor entries must be recorded.
- 10) All veterinary visits, health problems, tests, test results, treatments and reasons for removal from the pre-export quarantine facility of any animal, must be reported to the Official Veterinarian within 24 hours, and to the Department of Agriculture and Water Resources within 48 hours. The sole exceptions to this are inspections, visits and treatments required for certification.
- 11) A detailed health record must be kept for each zoo bovid and be available to the Official Veterinarian and to the Department of Agriculture and Water Resources on request.
- 12) Zoo bovids that leave the facility during pre-export quarantine for any reason not authorised by the Department of Agriculture and Water Resources cannot re-join the consignment during pre-export quarantine.

### **5.1.3 Certification**

The Official Veterinarian must certify:

- 1) During pre-export quarantine:
  - a) the zoo bovid(s) was/were not vaccinated
  - b) all zoo bovids in the pre-export quarantine facility remained free from evidence of infectious or contagious disease
  - c) all samples for testing were taken by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian

- d) all testing was conducted in a laboratory approved by the Veterinary Authority in the country of export.

2) All of the following risk management measures apply:

#### ANTHRAX

- For 20 days immediately before export the animals have not resided on any premises in which clinical, epidemiological or other evidence of anthrax has occurred in any species during the previous 20 days, and the disease is compulsorily notifiable.

#### BESNOITIOSIS

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of *B. besnoiti* has occurred in any species and the disease is compulsorily notifiable.

#### BLUETONGUE

##### **Option ONE**

- The animal was not vaccinated against BTV in the 60 days prior to export.

AND

- In the 7 days immediately before export, a blood sample was taken from the animal and tested by an approved PCR method for bluetongue virus. The test result was negative.

AND

- Immediately on arrival into Australia, during transit to post-arrival quarantine site, and during post-arrival quarantine, the animal was protected from vector attacks. Details of how this will be accomplished should be detailed in the travel plan for approval.

AND

- At least 7 days post-arrival into Australia, a blood sample was taken from the animal in PEQ and tested by an approved PCR method for bluetongue virus.
  - If the test result is negative, vector protection may cease.
  - If the test result is positive, vector protection is maintained and the Department of Agriculture and Water Resources is contacted as soon as possible (and no later than 48 hours) following the result for further direction.

##### **Option TWO**

- The animal was kept in a country free or seasonally free from BTV as recognised by Australia\* at least 60 days prior to export (and within the period the country is considered free from BTV).

\*Countries recognised as free from BTV or having seasonally free periods from BTV:

- Canada (not including the Okanagan Valley of British Columbia)—country seasonally free between 1 January and 15 May.
- New Zealand – country free from BTV.

AND

- The animal:
  - was not vaccinatedOR
  - was vaccinated, and the vaccine was: inactivated, approved by the competent authority in the exporting country, and administered more than 60 days prior to semen collection.

[The veterinary health certificate must indicate the option that applies].

### BOVINE TUBERCULOSIS

#### **Option ONE**

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals for export were each subject to a test for bovine tuberculosis performed between 210 and 72 days immediately before export, with negative results. The test must be:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative results.OR
  - Performed on a blood sample taken during pre-export quarantine and tested using *either* a gamma interferon assay approved by the department *or* a serological test approved by the department, with negative results.

AND

- The animals for export were each subject to a tuberculin skin test (TST) *or* comparative tuberculin skin test (CTST) performed in the 30 days immediately before export. The test was read 72 hours post-inoculation, with negative results.

AND

- The animals for export were each subject to *either* a gamma interferon assay approved by the department *or* a serological test approved by the department, with negative results. The test was performed in the 30 days immediately before export on blood taken during this period.

#### **Option TWO**

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals for export each received three separate skin tests for tuberculosis in the 210 days prior to export. The tests were performed a minimum of 42 days apart from each other

and one was *during* pre-export quarantine. Each test was *either* a TST *or* CTST. Each test was read 72 hours post-inoculation, with negative results.

### Option THREE

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - Diagnostic testing of all zoo bovids in the collection, performed at least annually, with negative results. The diagnostic tests must be of a type approved by the department (e.g. TST, CTST, approved gamma interferon, approved serological test).
  - That the collection must have been a ‘closed-herd’<sup>6</sup> during that time.
  - That the collection must contain at least four zoo bovids.
  - That full post mortem investigations were conducted on any dead ungulate species to determine the cause of death.

AND

- The animals for export were each tested for bovine tuberculosis between 210 and 72 days immediately before export. If the test for the collection screening program occurs during this time, it will fulfil this requirement. The test must be either:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative results.OR
  - 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.

AND

- The animals for export were each tested with a TST *or* CTST performed during the 30 days immediately before export. The test was read 72 hours post-inoculation, with negative results.

### Option FOUR

- For 12 months immediately before export the animals have not resided on any premises where clinical, epidemiological or other evidence of bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

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<sup>6</sup> A ‘closed-herd’ in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - diagnostic testing of all zoo bovids in the collection, performed at least annually, with negative results. The diagnostic tests must be of a type approved by the department (e.g. TST, CTST, approved gamma interferon, approved serological test)
  - that the collection must have been a ‘closed-herd’<sup>7</sup> during that time
  - that the collection must contain at least four zoo bovids
  - that full post mortem investigations were conducted on any dead ungulate species to determine the cause of death.

AND

- The animals for export were each tested via a TST *or* CTST, performed between 210 and 72 days immediately before export. The test was read 72 hours post-inoculation, with negative results. If the test for the herd screening program occurs during this time, it will fulfil this requirement.

AND

- The animals for export were each tested for bovine tuberculosis, performed during the 30 days immediately before export. The test must be either:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative results.OR
  - 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.

#### BOVINE VIRAL DIARRHOEA (Type 2)

##### **Option ONE**

- Since birth the animal was continuously resident in a country free from BVD. The disease must be compulsorily notifiable.

##### **Option TWO**

- During pre-export quarantine, a blood sample was drawn from the animal and tested by an RT-PCR test approved by the department. The test was negative to BVD.

AND

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<sup>7</sup> A ‘closed-herd’ in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

- During pre-export quarantine, a haired skin sample was taken from the animal (ear notch or caudal tail fold) and tested using an antigen ELISA test approved by the department. The test was negative to BVD.

### Option THREE

- During pre-export quarantine, a blood sample was drawn from the animal and tested by:
    - a RT-PCR test or antigen ELISA test approved by the department. The test was negative to BVD
- AND
- an antibody ELISA test or VNT approved by the department. The test was negative to BVD.

### Option FOUR

- For 180 days immediately before export the animal for export was part of a zoo collection subject to a documented BVD screening program. The screening program must include:
  - diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department. The testing regime must establish that neither infection nor persistently infected animals are present
  - that the collection must have been a ‘closed-herd<sup>8</sup>’ during that time
  - that the collection must contain at least four zoo bovids.

AND

- The animal was tested by the screening program during this time.

### BRUCELLOSIS (*B. abortus* and *B. melitensis*)

### Option ONE

- For 12 months immediately before export the animal was continuously resident in a country *and* premises where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* and *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable.

### Option TWO

- For 12 months immediately before export or since birth the animal has only resided on premises where no clinical, epidemiological or other evidence of brucellosis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

AND

- In the 30 days immediately prior to export the animal was subjected to an approved ELISA or BBAT test for brucellosis (*B. abortus* and *B. melitensis*). In the case of post-parturient

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<sup>8</sup> A ‘closed-herd’ in this context means that new animals susceptible to BVD were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

females, the test was carried out at least 30 days after giving birth. The test result was negative.

#### CONTAGIOUS CAPRINE PLEUROPNEUMONIA

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of CCPP has occurred during the previous 12 months and the disease is compulsorily notifiable.

#### FOOT AND MOUTH DISEASE

- For 270 days immediately before export the animal was continuously resident in a country on the department's FMD-free approved country list.

#### HAEMORRHAGIC SEPTICAEMIA

##### **Option ONE**

- For 270 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of haemorrhagic septicaemia occurred during the previous 12 months before export and the disease is compulsorily notifiable.

##### **Option TWO**

- For 270 days immediately before export the animal did not reside on any premises where clinical, epidemiological or other evidence of haemorrhagic septicaemia occurred during the previous 12 months before export and the disease is compulsorily notifiable.

AND

- Between 90 and 180 days immediately before export the animal was vaccinated against haemorrhagic septicaemia with an approved vaccine.

#### HEARTWATER

- For 24 months prior to export the animal (and its dam, if the animal for export is under 12 months of age) has not resided in any country where clinical, epidemiological or other evidence of heartwater has occurred in any species during the previous 2 years and the disease is compulsorily notifiable.

AND

- Within two working days of entry to the pre-export quarantine facility the animal was examined thoroughly for ticks, found free of ticks and treated under the supervision of the Official Veterinarian, with a long acting parasiticide effective against ticks.

#### INFECTIOUS BOVINE RHINOTRACHEITIS

##### **Option ONE**

- For 180 days immediately before export the animal was continuously resident on premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- In the 30 days immediately before export the animal was tested for BoHV-1 twice, at an interval of no less than 21 days, on separate blood samples drawn during this time. The test was of a type approved by the department. The test results were negative.

**Option TWO**

- For 180 days immediately before export the animals was continuously resident on any premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- For 180 days immediately before export the animals for export were part of a collection subject to a documented BoHV-1 screening program. The screening program must include:
  - diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department
  - that the collection must have been a 'closed-herd' during that time
  - that the collection must contain at least four zoo bovids.

AND

- During the 180 days immediately before export the animal was tested as part of the screening program.

**LUMPY SKIN DISEASE****Option ONE**

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of LSDV has occurred during the previous 3 years, the disease is compulsorily notifiable and vaccination against LSDV has not occurred in the previous 3 years.

AND

- The animal showed no clinical signs of LSD during pre-export quarantine (PEQ).

AND

- The animal has not been vaccinated against capripoxviruses in the previous 3 years (LSDV or Sheep or Goat Pox strain vaccines).

**Option TWO**

The department will consider applications to import live bovids from approved countries that do not meet the requirements of Option ONE on a case-by-case basis, with respect to the epidemiological situation at the time. As a guide, minimum requirements will consist of all of the following:

- The animal has not been vaccinated against capripoxviruses in the previous 3 years (LSDV or sheep or goat pox strain vaccines).
- The animal showed no clinical signs of LSD during pre-export quarantine (PEQ).

- During the final 7 days of pre-export quarantine, each animal was tested for LSD with a PCR test and returned negative results.

#### MALIGNANT CATARRHAL FEVER (WILDEBEEST ASSOCIATED)

- No live animals of the genus *Connochaetes* may be imported into Australia.

#### PESTE DES PETITS RUMINANTS

- Since birth the animal was continuously resident in a country where no clinical, epidemiological or evidence of PPR has occurred during the previous 2 years and the disease is compulsorily notifiable.

#### RABIES

- For 180 days immediately before export the animal did not reside on any premises where clinical, epidemiological or other evidence of rabies occurred during the previous twelve months before export and the disease is compulsorily notifiable.

#### RIFT VALLEY FEVER

- For 90 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of RVF has occurred during the previous ten years and the disease is compulsorily notifiable.

#### SCHMALLEMBERG VIRUS

##### **Option ONE**

- For 30 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of SBV has previously occurred and the disease is compulsorily notifiable.

##### **Option TWO**

- The animal tested seropositive to SBV between 10 and 90 days immediately prior to export, by a virus neutralisation test or approved ELISA.

##### **Option THREE**

- The animal was vaccinated against SBV using an approved, commercially available inactivated vaccine during the 180 days immediately prior to export. Vaccination must be completed before entering pre-export quarantine (PEQ). The vaccination regime must follow the commercial regime for cattle (two doses given four weeks apart).

#### SCREW-WORM FLY MYIASIS

- For 60 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of screw-worm-fly (*Cochliomyia hominivorax* or *Chrysomya bezziana*) myiasis occurred during the previous 12 months and the disease is compulsorily notifiable.

## SURRA

### **Option ONE**

- Since birth, the animal was continuously resident in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

### **Option TWO**

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

AND

- During pre-export quarantine a blood sample was drawn from *a peripheral vein* of the animal and tested using the Haematocrit centrifuge technique<sup>9</sup>. The test was negative for trypanosomes.

## TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

- Since birth, the animals for import have only lived in a country (or countries) listed as having a negligible or controlled BSE status by the OIE (for the period of that residency). The countries and periods of residency must be listed on the veterinary health certificate.

## TRYPANOSOMOSIS (tsetse fly associated)

### **Option ONE**

- Since birth the animal was continuously resident in a country where no clinical, epidemiological or other evidence of trypanosomiasis due to *T. vivax* infection has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

### **Option TWO**

- For 180 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of trypanosomiasis due to *T. vivax* infection has occurred in any species during the previous 2 years and the disease is compulsorily notifiable.

AND

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<sup>9</sup> Method detailed by: OIE 2018b, 'Trypanosomosis (tsetse-transmitted)', in *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 2018*, World Organisation for Animal Health, Paris. Available at: [http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.04.17\\_TRYPANOSOMOSIS.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.04.17_TRYPANOSOMOSIS.pdf)

- During pre-export quarantine a blood sample was drawn from a *peripheral vein* of the animal and tested using the Haematocrit centrifuge technique<sup>10</sup>. The test was negative for trypanosomes.

### VESICULAR STOMATITIS

#### **Option ONE**

- For 60 days immediately before export the animal was continuously resident in a country where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

#### **Option TWO**

- For 60 days immediately before export the animal was continuously resident on premises in the country of export where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous 90 days and the disease is compulsorily notifiable.

AND

- The animal was held in PEQ for at least 30 days immediately before export.

AND

- The health monitoring program of the exporting zoo includes vesicular stomatitis.
- During the first seven days of PEQ, the animal was treated with a broad spectrum anthelmintic (or combination of anthelmintics) effective against nematodes and cestodes, and tested by faecal flotation 14 days later. The animal was re-treated if there was evidence of parasites on testing (active ingredient/s, dose and date/s of treatment stated on the veterinary health certificate), then retested again 14 days later and returned a negative result.
- During the 30 days immediately before export, each bovid was treated on two occasions (at least 21 days apart), with a long acting external parasiticide effective against ticks to provide continual protection against tick infestation beyond the day of export. The final treatment must occur within seven days of export (active ingredient/s, dose and date/s of treatment stated on the veterinary health certificate).
- The zoo bovid was examined by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian within 72 hours before leaving the pre-export quarantine facility for the port of export and was found to be:
  - free from evidence of infectious or contagious disease
  - visibly free of external parasites
  - healthy and fit to travel.

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<sup>10</sup> Method detailed by: OIE 2018b, 'Trypanosomosis (tsetse-transmitted)', in *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals, 2018*, World Organisation for Animal Health, Paris. Available at: [http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.04.17\\_TRYPANOSOMOSIS.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.04.17_TRYPANOSOMOSIS.pdf)

- Vehicles and transport containers used for transporting zoo bovids from the pre-export quarantine facility to the port of export, and to Australia, were new or were cleaned and disinfected to the satisfaction of the Official Veterinarian before entering the pre-export quarantine facility to load the zoo bovids.
- The zoo bovid was sealed in its travel container with tamper-evident seals before leaving the pre-export quarantine facility for the port of export. The seal number is recorded on the certificate.
- Arrangements are in place to ensure that the zoo bovid had no contact with other animals prior to departure of the vessel or aircraft except those that meet all the conditions in these biosecurity measures.

#### **5.1.4 Transport**

- 1) Exporters or their agents must have detailed plans to cover procedures including contingency plans, for transporting the animal from pre-export quarantine until arrival in Australia.
- 2) Animals must be consigned to Australia by a route approved by the Department of Agriculture and Water Resources.
- 3) Animals must travel in a container recommended for that particular species under the International Air Transport Association (IATA) Live Animal Regulations.
- 4) The use of hay or straw as bedding during transport is not permitted. Treated wood shavings, sterilised peat and soft board can be used.
- 5) Animals must remain isolated from all animals except those that meet all the conditions described in these biosecurity measures, during transport from the pre-export quarantine facility until arrival in Australia.
- 6) Insect netting must be carried on the flight at all times for contingencies. There must be sufficient insect netting to cover all travel containers completely. Insect netting must be in good condition to minimise entry of insect vectors into the travel containers.

#### **Transit and transhipment**

- 1) Animals must transit or tranship only at an approved airport. Any transhipment requires the prior approval of the Department of Agriculture and Water Resources. Animals are not to leave the airport and must not be removed from their travel containers during transit or transhipment.
- 2) Animals must remain on board the aircraft at approved transit airports. Cargo doors can be opened at approved transit airports to allow for unloading or loading of freight. Immediately after the cargo hold doors are closed, a knockdown aerosol insecticide must be sprayed throughout the cargo hold, in the manner recommended by the manufacturer.
- 3) In cases where animals in travel containers are to be unloaded, before opening the cargo door, the travel containers must be completely covered in netting to prevent insect access to the animals. The netting must remain in place until the animals are reloaded onto an aircraft. Immediately after the animals are reloaded onto an aircraft and the cargo hold doors are closed, a knockdown aerosol insecticide spray must be sprayed throughout the

cargo hold in the manner recommended by the manufacturer. The insect netting must not be removed until 30 minutes after spraying.

#### **Delayed take off and unscheduled landings**

- 1) Exporters or their agents must have contingency plans for the management of delayed take off and unscheduled landings.
- 2) If the aircraft lands at any airport other than in an approved country, the department must be informed immediately and the animal must not proceed to Australia without approval from the department. The decision as to whether the animal can continue to travel to Australia, and additional biosecurity measures that may be required, will be made by the Department of Agriculture and Water Resources on a case-by-case basis after assessing the risks.

#### **Arrival in Australia**

- 1) Importers or their agents must have a plan developed in consultation with the Department of Agriculture and Water Resources to cover post-arrival procedures. The plan must include roles and responsibilities for their staff, vehicles for transporting animals to the Approved Arrangement site (AA site) and road transport arrangements including contingency plans for vehicle and equipment failures.
- 2) Vehicles for transporting the animals from the port of entry to the AA site must be cleaned and disinfected to the satisfaction of the Australian government biosecurity officer before loading the animals. The Department of Agriculture and Water Resources must be advised of the transport route to the AA site.
- 3) After the animals arrive at an Australian airport they must be transferred in their transport containers onto vehicles, along with personnel and equipment, and proceed directly to the AA site.
- 4) All biosecurity risk material (e.g. bedding, feed, water and waste material) remaining at the airport must be sealed in bags, ordered into quarantine and disposed of under supervision of the Department of Agriculture and Water Resources.
- 5) All other equipment used during transport that has been in contact with the animal (including the inside of the crate, bedding, waste and water) must be cleaned and disinfected under supervision of the Department of Agriculture and Water Resources before leaving the airport.

### **5.1.5 Post-arrival quarantine requirements**

#### **Post-arrival quarantine**

The minimum post-arrival quarantine period of 30 days applies.

Any variation from the post-arrival quarantine requirements must be specifically authorised by the Department of Agriculture and Water Resources.

#### **Location**

The AA site must be located within a secure part of a zoo, wildlife park or research institute approved under relevant Australian State or Territory legislation to hold the species being

imported, separated from public access areas and where it is under regular supervision by a registered veterinarian.

**Facilities**

The post-arrival quarantine facility must meet the Department of Agriculture and Water Resources requirements of a class 7.9 AA site.

**Operation**

- 1) The AA site must be approved by the Department of Agriculture and Water Resources before entry of an animal into the AA.
- 2) All post-arrival quarantine operations and procedures must follow those outlined for an AA class 7.9 facility and also include:
  - a) A registered veterinarian must inspect the AA site within 72 hours before entry of any animal to ensure it has been cleaned and disinfectant has been applied to their satisfaction.
  - b) The post-arrival quarantine period will commence from the time of entry into the facility of the last animal.
  - c) Vehicles for transporting animals must not leave the AA site until thoroughly cleaned and disinfected.
  - d) If any animal dies during post-arrival quarantine, the Department of Agriculture and Water Resources must be notified within 48 hours and the animal must undergo a post mortem investigation by a registered veterinarian to determine the cause of death.
  - e) The Department of Agriculture and Water Resources is to be advised within 48 hours of any disease incident and its outcome.
  - f) Animals and goods subject to biosecurity control must not leave the AA site during post-arrival quarantine without permission of the Department of Agriculture and Water Resources.
  - g) At the satisfactory completion of post-arrival quarantine, the animals will be released from biosecurity control into premises approved by the appropriate State or Territory governments for the holding of zoo bovids.

## 5.2 Proposed biosecurity measures for the importation of zoo bovid semen from approved countries

### 5.2.1 Documentation

Each consignment of zoo bovid semen must travel with an original international veterinary health certificate that conforms to Article 5.10.2 of the OIE Code, signed by the Official Veterinarian of the country of export.

These biosecurity requirements apply to zoo bovid semen.

An **Official Veterinarian** means a veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the OIE Code.

The veterinary health certificate must:

- be written in English and a language understood by the Official Veterinarian of the country of export
- meet the requirements of the 'Certification – Zoo bovid semen' section and state that all the pre-export quarantine requirements have been met
- include the name and address of the zoological or wildlife park of origin
- include the name and address of the exporter and importer
- identify the import permit against which it was issued
- the name and species for each semen donor
- the herd or stud book number for each semen donor
- provide unique identification for each zoo bovid (International Standards Organisation (ISO) microchip number) including description, species, sex and age
- the date(s) of the semen collection period(s) for each donor
- date of entry that donor entered the collection centre/resident herd
- the number of straws in this consignment for each donor and contain the means to verify the identification of the semen straws with the identification details of the donor
- the dates of sampling for any tests required, the type of test used and the test results. This information must be contained in a table against donor information
- the dates of isolation of the semen donor from other animals that did not meet the same biosecurity conditions.

The Official Veterinarian must:

- sign, date and stamp (with the stamp of the Veterinary Authority) each page of the veterinary health certificate and all attached documents (e.g. laboratory reports) that form part of the veterinary health certification
- endorse each page of copies of supporting documents with date, signature and Official Veterinarian stamp

- record his/her name, signature and contact details on the veterinary health certificate.

## **5.2.2 Certification – Zoo bovid semen**

### **1) GENERAL**

- a) The semen was not removed from containers for further processing or aggregation unless previously arranged with the department.
- b) All semen collected for export to Australia must meet all the conditions specified in the following clauses.

### **2) SEMEN COLLECTION AND PROCESSING**

- a) The semen was hygienically collected, handled and processed:
  - i) at a registered zoo or wildlife park in the exporting country that meets the general risk measures for zoo semen as detailed at the start of Section 5.
  - ii) using properly disinfected or sterilised implements, and
  - iii) using products of animal origin, including additives or a diluent, that were obtained from sources which present no animal health risk or were treated prior to use such that the risk is managed.

### **3) DIAGNOSTIC TESTING**

- a) The samples for diagnostic testing were collected by veterinarians approved by the Veterinary Authority for export certification.
- b) Tests for disease were carried out at a laboratory approved by the competent authority.
- c) The tests were conducted in accordance with the current OIE Manual for Diagnostic Tests and Vaccines for Terrestrial Animals or were approved by the department.
- d) All disease testing results are tabulated, including donor identification, dates of sampling for test, type of tests used, test results and are verified by the Official Veterinarian.

### **4) THE OFFICIAL VETERINARIAN**

- a) The Official Veterinarian:
  - i) Ensured all samples for testing were taken by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian
  - ii) ensured that the donors were tested in accordance with all requirements
  - iii) recorded the required details for each donor on the table attached to this veterinary health certificate
  - iv) ensured the hygienic collection, handling and processing of the semen
  - v) verified the permanent identification of the semen straws with the identification details of the donor and the date of collection or a code from which this information could be determined.

### **5) DISEASE FREEDOM AND RISK MANAGEMENT**

- a) Donors showed no clinical signs of infectious or contagious disease on the day(s) of semen collection and for thirty (30) days after.

b) Specific risk management measures for the following diseases apply:

[Where multiple options are available the veterinary health certificate must indicate the option that applies]

### **BLUETONGUE**

#### **Option ONE**

Blood samples were drawn from the donor:

- Between 28 and 60 days immediately after the semen collection period finished. The blood samples gave negative results to the competitive ELISA for BTV antibodies.

OR

- On the first day, the last day, and at least every 7 days during the semen collection period. The blood samples gave negative results to a virus isolation test for BTV.

OR

- On the first day and the last day of the semen collection period. The blood samples gave negative results to an approved RT-PCR test for BTV.

[The veterinary health certificate must indicate the option that applies].

#### **Option TWO**

- All donors were kept in a country free or seasonally free from BTV as recognised by Australia\* at least 60 days prior to, and at the time of, semen collection.

\*Countries recognised as free from BTV or having seasonally free periods from BT:

- Canada (not including the Okanagan Valley of British Columbia)—country seasonally free between 1 January and 15 May.
- New Zealand – country free from BTV.

AND

- The animal:
    - Was not vaccinated.
- OR
- Was vaccinated, and the vaccine was: inactivated, approved by the competent authority in the exporting country, and administered more than 60 days prior to semen collection.

[The veterinary health certificate must indicate the option that applies].

### **BOVINE TUBERCULOSIS**

#### **Option ONE**

- 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 bovine tuberculosis has occurred during the previous five years and the disease is compulsorily notifiable.

AND

- The animals from which the semen for export was collected were each subject to a test for bovine tuberculosis performed between the 210 and 72 days immediately before semen collection. The test must be:
  - A TST or CTST. The test was read 72 hours post-inoculation, with negative results.
- OR
- 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.

AND

- The animals from which the semen for export was collected were each subject to a tuberculin skin test (TST) *or* comparative tuberculin skin test (CTST) performed in the 30 days immediately before semen collection. The test was read 72 hours post-inoculation, with negative results.

#### **Option TWO**

- 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 bovine tuberculosis has occurred during the previous three years and the disease is compulsorily notifiable.

AND

- For 12 months immediately before export the animals for export were part of a collection subject to a documented tuberculosis screening program. The screening program must include:
  - Diagnostic testing of the zoo bovids in the collection, performed at least annually, with negative results. The diagnostic tests must be of a type approved by the department (e.g. TST, CTST, approved gamma interferon, approved serological test).
  - The collection must have been a ‘closed-herd’<sup>11</sup> during that time.
  - The collection must contain at least four zoo bovids.
  - Full post mortem investigations were conducted on any dead ungulate species to determine the cause of death.

AND

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<sup>11</sup> A ‘closed-herd’ in this context means that new animals susceptible to BTB were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

- The animals from which the semen for export was collected were subject to a test for bovine tuberculosis, performed in the 30 days immediately before semen collection. This test must be separate to the herd screen program test. The test must be either:
    - a TST or CTST. The test was read 72 hours post-inoculation, with negative results. If the herd test was also a TST or CTST, then this second test must not be performed within 42 days of the herd test.
- OR
- Performed on a blood sample taken in the 30 days immediately before semen collection and tested using *either* a gamma interferon assay approved by the department *or* a serological test approved by the department, with negative results.

#### BOVINE VIRAL DIARRHOEA (Type 2)

##### **Option ONE**

- The semen was tested by an approved virus isolation or approved RT-PCR test for BVD, with negative results.

##### **Option TWO**

- On the *last* day of the semen collection period, a blood sample was drawn from the donor animal and tested by:
  - A RT-PCR test or antigen ELISA test approved by the department. The test was negative to BVD.

AND

- An antibody ELISA test or VNT approved by the department. The test was negative to BVD.

#### BRUCELLOSIS (*B. Abortus* and *B. Melitensis*)

##### **Option ONE**

- For 12 months immediately before semen collection or since birth the animal was continuously resident in a country *and* premises where no clinical, epidemiological or other evidence of brucellosis (*B. abortus* or *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable.

##### **Option TWO**

- For 12 months immediately before semen collection or since birth the animal has not resided on any premises in the country of export where clinical, epidemiological or other evidence of brucellosis (*B. abortus* or *B. melitensis*) has occurred in any species during the previous two years and the disease is compulsorily notifiable.

AND

- The donor animal was subjected to an approved ELISA or BBAT test for brucellosis (*B. abortus* or *B. melitensis*) within the 30 days immediately after collection. The test result was negative.

**CONTAGIOUS CAPRINE PLEUROPNEUMONIA**

- Since birth, the animal was continuously resident in a country (or countries) where no clinical, epidemiological or other evidence of CCPP has occurred in any species and the disease is compulsorily notifiable.

**FOOT AND MOUTH DISEASE**

- For 90 days immediately before collection, the animals from which the semen for export was collected resided in a country on the department's FMD-free approved country list.

**INFECTIOUS BOVINE RHINOTRACHEITIS****Option ONE**

- The semen was tested for BoHV-1 by RT-PCR in accordance with the OIE prescribed preparation and testing regime for semen (OIE 2017b).

**Option TWO**

- For 180 days immediately before the semen collection period the donor animal was continuously resident on any premises where no clinical, epidemiological or other evidence of BoHV-1 has occurred during the previous 12 months.

AND

- For 180 days immediately before the semen collection period the donor animal was part of a zoo collection subject to a documented BoHV-1 screening program. The screening program must include:
  - Diagnostic testing of all zoo bovids in the collection, performed at least annually. The diagnostic tests must be of a type approved by the department.
  - The collection must have been a 'closed-herd'<sup>12</sup> during that time.
  - The collection must contain at least four zoo bovids.

AND

- The donor animal was tested by the screening program during this time.

**LUMPY SKIN DISEASE**

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

**MALIGNANT CATARRHAL FEVER (WILDEBEEST ASSOCIATED)**

- The animals from which the semen for export was collected are not members of the genus *Connochaetes*.

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<sup>12</sup> A 'closed-herd' in this context means that new animals susceptible to BoHV-1 were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).

### PESTE DES PETITS RUMINANTS

- Since birth the donor was continuously resident in a country where no clinical, epidemiological or other evidence of PPR has occurred during the previous 2 years and the disease is compulsorily notifiable.

### RIFT VALLEY FEVER

- For 90 days immediately prior to collection the animal was continuously resident in a country where no clinical, epidemiological or other evidence of RVF has occurred in any species during the previous ten years and the disease is compulsorily notifiable.

### SCHMALLENBERG

#### **Option ONE**

- For 30 days immediately prior to collection the donor was continuously resident and in a country where no clinical, epidemiological or other evidence of SBV has previously occurred and the disease is compulsorily notifiable.

#### **Option TWO**

- The animal tested seronegative to SBV between 28 and 90 days after semen collection, by an approved virus neutralisation test or approved ELISA.

#### **Option THREE**

- The semen for export was tested by an approved qRT-PCR method, with negative results.

#### **Option FOUR**

- The semen was collected before 1 June 2011.

### VESICULAR STOMATITIS

- During the 30 days immediately prior to collection of the semen there were no clinical signs or other evidence of vesicular stomatitis in the premises of origin and the disease is compulsorily notifiable.

## **9) STORAGE AND TRANSPORT**

- a) From the time of freezing until export, the reproductive material in this consignment was:
  - i) kept in sealed sterile containers (e.g. straws, ampoules or vials) and code marked in line with the international standards of the International Committee for Animal Recording (ICAR)
  - ii) stored and transported EITHER only with other zoo bovid semen collected for export to Australia, or of equivalent health status OR with other export certified germplasm eligible for export to Australia provided ALL germplasm were intact and there were no damaged or broken straws, ampoules or vials in the shipping container
  - iii) kept in a secure place within an approved centre or laboratory and under the supervision of the Official Veterinarian
  - iv) stored and transported in storage or shipping containers containing only new, unused liquid nitrogen

v) stored for at least 30 days.

i)

6) SHIPPING CONTAINERS (LIQUID NITROGEN SHIPPERS/TANKS)

EITHER

a) The shipping container was new.

OR

b) Immediately prior to loading, the shipping container was emptied and inspected and any loose straws removed. The shipping container, including all surfaces in contact with the straws, ampoules or vials was then disinfected with one of the following disinfectants: 2 per cent available chlorine (e.g. chlorine bleach), 2 per cent Virkon or irradiated at 50 kGy.

c) The veterinary health certificate must indicate the option that applies. For used shipping containers, the date of disinfection, the disinfectant used and its active chemical must be recorded on the health certificate.

7) OFFICIAL GOVERNMENT SEALS

a) Under the supervision of an Official Veterinarian prior to export to Australia:

i) the identity of the semen was checked prior to being placed into new, unused liquid nitrogen in a shipping container for export that was new or disinfected as specified in this veterinary health certificate

ii) the containers (e.g. straws, ampoules or vials) for reproductive material in this consignment were checked and confirmed as being sealed

b) Only zoo bovid semen that met Australian import conditions was included in the shipping container.

c) An official government seal was applied by an Official Veterinarian to the shipping container and the number or mark on the seal recorded on the certificate.

# Glossary

Term or abbreviation	Definition
Anergy	Absence of the normal immune response to a particular antigen or allergen.
Appropriate level of protection (ALOP) for Australia	The <i>Biosecurity Act 2015</i> defines the appropriate level of protection (or ALOP) for Australia as a high level of sanitary and phytosanitary protection aimed at reducing biosecurity risks to very low, but not to zero.
Australian territory	Australian territory as referenced in the <i>Biosecurity Act 2015</i> refers to Australia, Christmas Island and Cocos (Keeling) Islands.
Biosecurity	The prevention of the entry, establishment or spread of unwanted pests and infectious disease agents to protect human, animal or plant health or life, and the environment.
Biosecurity measures	The <i>Biosecurity Act 2015</i> defines biosecurity measures as measures to manage any of the following: biosecurity risk, the risk of contagion of a listed human disease, the risk of listed human diseases entering, emerging, establishing themselves or spreading in Australian territory, and biosecurity emergencies and human biosecurity emergencies.
Biosecurity risk	The <i>Biosecurity Act 2015</i> refers to biosecurity risk as the likelihood of a disease or pest entering, establishing or spreading in Australian territory, and the potential for the disease or pest causing harm to human, animal or plant health, the environment, economic or community activities.
Biosecurity import risk analysis (BIRA)	The <i>Biosecurity Act 2015</i> defines a BIRA as an evaluation of the level of biosecurity risk associated with particular goods, or a particular class of goods, that may be imported, or proposed to be imported, into Australian territory, including, if necessary, the identification of conditions that must be met to manage the level of biosecurity risk associated with the goods, or the class of goods, to a level that achieves the ALOP for Australia. The risk analysis process is regulated under legislation.
'Closed Herd'	A 'closed-herd' as used in this document means that new animals susceptible to the relevant disease were not introduced to the collection (including animals that were part of the collection for a time and were removed for a period prior to reintroduction).
The department	The Australian Government Department of Agriculture and Water Resources.
Endemic	Belonging to, native to, or prevalent in a particular geography, area or environment.
Goods	The <i>Biosecurity Act 2015</i> defines goods as an animal, a plant (whether moveable or not), a sample or specimen of a disease agent, a pest, mail or any other article, substance or thing (including, but not limited to, any kind of moveable property).
Host	An organism that harbours a parasite, mutual partner, or commensal partner, typically providing nourishment and shelter.
Import permit	Official document authorising a person to bring or import particular goods into Australian territory in accordance with specified import requirements.
Non-regulated risk analysis	Refers to the process for conducting a risk analysis that is not regulated under legislation ( <i>Biosecurity import risk analysis guidelines 2016</i> ).
Official Veterinarian	A veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the OIE Code.
Pathogen	A biological agent that can cause disease to its host.
Quarantine	Official confinement of regulated articles for observation and research or for further inspection, testing or treatment.

Term or abbreviation	Definition
Risk analysis	Refers to the technical or scientific process for assessing the level of biosecurity risk associated with the goods, or the class of goods, and if necessary, the identification of conditions that must be met to manage the level of biosecurity risk associated with the goods, or class of goods to a level that achieves the ALOP for Australia.
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures.
Stakeholders	Government agencies, individuals, community or industry groups or organizations, whether in Australia or overseas, including the proponent/applicant for a specific proposal, who have an interest in the policy issues.
Surveillance	An official process which collects and analyses information related to animal health.
Unrestricted risk	Unrestricted risk estimates apply in the absence of risk mitigation measures.
Vector	An organism that does not cause disease itself, but which causes infection by conveying pathogens from one host to another.
Zoo Bovid	Species within Family Bovidae, order Artiodactyla, that are exhibited in zoos, i.e. most species other than domestic cattle, sheep, and goat breeds. Zoo bovids do not include species from the <i>Bovini</i> tribe (e.g. domestic cattle, buffalo) or <i>Caprinae</i> subfamily (e.g. sheep, goats).

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