



Australian Government  
Department of Agriculture

# Importation of dogs and cats and their semen from approved countries Final policy review

---

NOVEMBER 2013



© Commonwealth of Australia

### **Ownership of intellectual property rights**

Unless otherwise noted, copyright (and any other intellectual property rights, if any) in this publication is owned by the Commonwealth of Australia (referred to as the Commonwealth).

### **Creative Commons licence**

All material in this publication is licensed under a Creative Commons Attribution 3.0 Australia Licence, save for content supplied by third parties, photographic images, logos, and the Commonwealth Coat of Arms.



Creative Commons Attribution 3.0 Australia Licence is a standard form licence agreement that allows you to copy, distribute, transmit and adapt this publication provided that you attribute the work. A summary of the licence terms is available from [creativecommons.org/licenses/by/3.0/au/deed.en](http://creativecommons.org/licenses/by/3.0/au/deed.en). The full licence terms are available from [creativecommons.org/licenses/by/3.0/au/legalcode](http://creativecommons.org/licenses/by/3.0/au/legalcode).

This publication (and any material sourced from it) should be attributed as:

Department of Agriculture (2013), *Importation of dogs and cats and their semen from approved countries: final policy review* CC BY 3.0.

### **Cataloguing data**

Department of Agriculture (2013), *Importation of dogs and cats and their semen from approved countries: final policy review*, Department of Agriculture, Canberra

### **Internet**

*Importation of dogs and cats and their semen from approved countries: final policy review* is available at [daff.gov.au](http://daff.gov.au).

Inquiries regarding the licence and any use of this document should be sent to:

[copyright@daff.gov.au](mailto:copyright@daff.gov.au).

### **Disclaimer**

The Australian Government acting through the Department of Agriculture has exercised due care and skill in the preparation and compilation of the information in this publication.

Notwithstanding, the Department of Agriculture, its employees and advisers disclaim all liability, including liability for negligence, for any loss, damage, injury, expense or cost incurred by any person as a result of accessing, using or relying upon any of the information in this publication to the maximum extent permitted by law.

Front cover images by: Stuart Doyle and Andrea Roberts.

# Contents

---

Acronyms and abbreviations .....	vii
Summary.....	ix
<b>1 Introduction .....</b>	<b>1</b>
1.1 Background.....	1
1.2 Australia’s biosecurity policy.....	2
1.3 Scope .....	2
1.4 Conditions for importation.....	3
1.5 Biosecurity policy since 1993.....	4
1.6 Quarantine facilities and demand .....	6
1.7 Potentially affected Australian sectors .....	6
1.8 Native fauna .....	6
<b>2 Method.....</b>	<b>9</b>
2.1 Background.....	9
2.2 Risk review .....	9
2.3 Review of hazard identification.....	10
2.4 Review of risk assessment .....	12
2.5 Review of risk management.....	12
2.6 Risk communication.....	13
<b>3 Hazard identification .....</b>	<b>15</b>
<b>4 Risk reviews.....</b>	<b>37</b>
4.1 Canine bartonellosis .....	37
4.2 Canine brucellosis.....	45
4.3 Canine influenza .....	51
4.4 Canine monocytic ehrlichiosis .....	54
4.5 Canine pulmonary angiostrongylosis.....	65

4.6	Chagas' disease .....	70
4.7	Hepatozoonosis.....	74
4.8	Leishmaniasis .....	80
4.9	Leptospirosis.....	86
4.10	Lyme disease .....	93
4.11	Nagana .....	101
4.12	Nipah virus encephalitis.....	103
4.13	Parasites—external .....	108
4.14	Parasites—internal.....	114
4.15	Piroplasmosis.....	117
4.16	Rabies.....	128
4.17	Rift Valley fever.....	154
4.18	Screw-worm fly myiasis.....	158
4.19	Surra.....	161
4.20	Tularaemia .....	165
4.21	Yersiniosis .....	169
<b>5</b>	<b>Risk management.....</b>	<b>173</b>
5.1	Risk management options .....	174
5.2	Biosecurity policy perspective .....	179
<b>6</b>	<b>Biosecurity measures for dogs, cats and their semen.....</b>	<b>183</b>
6.1	Hazard-specific biosecurity measures for dogs.....	185
6.2	Hazard-specific biosecurity measures for cats .....	205
6.3	Hazard-specific biosecurity measures for dog and cat semen .....	216
6.4	Revised category listings of approved countries.....	219
	<b>Appendix 1—Current category listings .....</b>	<b>221</b>
	<b>Appendix 2—Risk management for rabies .....</b>	<b>223</b>

## Tables

Table 1	Summary comparison of biosecurity measures under the previous and revised policies for the importation of dogs and cats and their semen .....	xiii
Table 2	Dog and cat imports into Australia (2007–2011).....	6
Table 3	Hazard identification and refinement for diseases affecting dogs and/or cats .....	17
Table 4	Examples of significant tick-transmitted diseases of dogs .....	109
Table 5	Summary of piroplasmid spp. reported in dogs and cats .....	120
Table 6	Species in the Lyssavirus genus and their host species .....	132
Table 7	Known reservoir hosts of rabies virus biotypes.....	133
Table 8	Guidelines for the vaccination of dogs and cats .....	140
Table 9	Categories of countries approved to export dogs and cats to Australia .....	179
Table 10	Revised categories of countries approved to export dogs and cats to Australia.....	181

## Figures

Figure 1	Decision tree for hazard identification and refinement .....	11
----------	--	----



# Acronyms and abbreviations

---

AGID	agar gel immunodiffusion
ALOP	appropriate level of protection
AQIS	Australian Quarantine and Inspection Service
AQPM	Animal Quarantine Policy Memorandum
AUSVETPLAN	Australian Veterinary Emergency Plan
BRS	Bureau of Resource Sciences
BRS review	<i>Review of quarantine policy for dogs and cats, with particular reference to rabies</i> (conducted by the Bureau of Resource Sciences)
CME	canine monocytic ehrlichiosis
Code	OIE <i>Terrestrial Animal Health Code</i>
CPA	canine pulmonary angiostrongylosis
CPAg	cytoplasmic protein antigen
CPAg-AGID	cytoplasmic antigen agar gel immunodiffusion test
DAFF	Australian Government Department of Agriculture, Fisheries and Forestry (now the Department of Agriculture)
ELISA	enzyme-linked immunosorbent assay
EUPMP	European Union Pet Movement Policy
FAVN	fluorescent antibody virus neutralisation
FeLV	feline leukaemia virus
FIV	feline immunodeficiency virus
IFAT	indirect fluorescent antibody test
IRA	import risk analysis
ISO	International Organization for Standardization
IU	international units
MAT	microscopic agglutination test
NiV	Nipah virus
NWSWF	New World screw-worm fly
OIE	World Organisation for Animal Health (formerly known as the Office International des Epizooties)
OIE Manual	<i>OIE Manual of diagnostic tests and vaccines for terrestrial animals</i>
OWSWF	Old World screw-worm fly
PAQ	post-arrival quarantine
PCR	polymerase chain reaction
PETS	Pet Travel Scheme
RFFIT	rapid fluorescent focus inhibition test
RNATT	rabies neutralising antibody titre test
RSAT	rapid slide agglutination test
SAT	serum agglutination test
SWF	screw-worm fly
WHO	World Health Organization
WTO	World Trade Organization
2-ME	2-mercaptoethanol



# Summary

---

This policy review considers the biosecurity risks for Australia associated with the importation of dogs and cats and their semen from approved countries. The previous major review of biosecurity requirements for the importation of domestic dogs and cats into Australia was conducted in 1993. That review had a primary focus on rabies, but also included a number of other diseases of biosecurity concern. Rabies has been eliminated as a significant public health risk in most parts of the developed world. However, annually 55 000 human deaths due to rabies are reported, most of them children in the developing world after being bitten by an infected dog (WHO 2013).

This policy review for the importation of dogs and cats and their semen was undertaken by the Australian Government Department of Agriculture. It takes into account stakeholder comments received on the draft policy review after it was released for public consultation on 26 July 2012. A total of 155 stakeholder submissions were received.

A general conclusion of this policy review is that an increased emphasis on offshore management (i.e. before export to Australia) of dogs and cats is an effective and practical approach to achieve Australia's appropriate level of protection.

For rabies, this policy review recommends that for the importation of dogs and cats from approved, rabies-affected countries, appropriate risk management can be achieved offshore through vaccination and verification of protective immunity by an appropriate serological test, at least 180 days immediately before export to Australia. The draft policy review proposed that rabies vaccination and serological testing for rabies should also apply to dogs and cats imported from rabies-free countries. Following consideration of stakeholder submissions, this policy review recommends that country certification of rabies freedom is an appropriate biosecurity measure for importation from approved rabies-free countries. Subject to animals satisfying rabies requirements for importation, this policy review recommends that post-arrival quarantine (PAQ) is no longer required as a biosecurity measure for rabies.

Dogs and cats in countries that are not approved by the Department of Agriculture continue to only be eligible for importation via an approved country and must meet all of the Department of Agriculture's biosecurity requirements for that approved country. However, for importation from approved countries not recognised by the Department of Agriculture as rabies-free, this policy review recommends the removal of the pre-export residency requirement of six months in an approved country. All pre-export preparations must still be performed in an approved country, including a valid rabies neutralising antibody titre test at least 180 days before export to Australia.

In addition to biosecurity measures for rabies, this policy review recommends that biosecurity measures apply for the following diseases: canine brucellosis, canine

influenza virus, canine monocytic ehrlichiosis, hepatozoonosis, leishmaniasis, Lyme disease, leptospirosis, piroplasmosis, screw-worm fly myiasis, tularaemia and yersiniosis.

For external parasites and associated vector-borne diseases of biosecurity concern (canine monocytic ehrlichiosis, hepatozoonosis, Lyme disease, piroplasmosis, tularaemia and yersiniosis) the conclusion of this policy review is that offshore measures can be enhanced through an increased duration of pre-export treatment to reduce the likelihood of exposure to parasite vectors. This will reduce the likelihood that dogs and cats are harbouring external parasites, or incubating infection with a vector-borne hazard of biosecurity concern when imported.

However this policy review recommends that PAQ remains a necessary biosecurity measure, with a minimum PAQ period of 10 days to apply for both dogs and cats. Ten days in quarantine is a significant reduction from the minimum PAQ period of 30 days that applied under the previous policy.

For dog semen, the draft policy proposed that importation be restricted to semen processed in straws, primarily to facilitate identification of individual semen doses with the corresponding donor. Detailed technical submissions were received in support of the continued importation of semen in pellet form. This policy review recommends that the Department of Agriculture continues to allow dog semen to be imported in pellet form, subject to compliance with minimum packaging and identification standards that enable semen pellets to be identifiable by donor and date of collection.

Key revisions from the previous biosecurity policy for dogs and cats and their semen are outlined below and summarised in Table 1.

Countries, administrative regions and territories from which Australia permits the importation of dogs and cats and their semen are referred to as approved countries. This policy review recommends that the five categories for approved countries in the previous policy be consolidated into three categories to simplify requirements for importation. The recommended categories are:

- Category 1—rabies-free, with dog and cat health status at least equivalent to Australia
- Category 2—other rabies-free countries
- Category 3—all other approved countries.

For importation from New Zealand, the requirement for a continuous period of residency of at least 90 days before importation has been removed. Removal of this requirement recognises the close harmonisation of biosecurity measures between Australia and New Zealand as well as New Zealand's favourable health status for pests and diseases of dogs and cats.

In addition to the changes recommended to biosecurity measures for rabies, this policy review also recommends that biosecurity measures for other diseases of biosecurity concern be amended as follows:

- canine brucellosis (dogs only)—serological testing should only apply to breeding dogs, not desexed dogs
- external parasites:
  - for cats, two parasiticide treatments within 45 days before export (the previous policy required one pre-export treatment)
  - for dogs, parasiticide treatment to commence at least 21 days before blood sample collection for canine monocytic ehrlichiosis serology (the previous policy required treatment to commence at the time of blood sample collection)
  - a minimum PAQ period of 10 days<sup>1</sup> for both dogs and cats (the previous policy required a minimum PAQ period of 30 days)
- internal parasites—two parasiticide treatments within 45 days before export (the previous policy required one pre-export treatment)
- leptospirosis (dogs only)—either serological testing or vaccination or antibiotic treatment (the previous policy only allowed for serological testing)
- Nipah virus encephalitis—for dogs and cats imported from Malaysia, the serological test requirement has been removed.

For dog semen, this policy review recommends that biosecurity measures should apply for canine brucellosis, leishmaniasis and leptospirosis. The previous policy required pre-collection serological testing of donors for canine brucellosis and leptospirosis but testing for leishmaniasis was not required. Testing of donors for leishmaniasis is recommended on the basis of evidence in the scientific literature that leishmaniasis can be transmitted via semen and that leishmaniasis has a wide geographic distribution.

To better manage the risk that donors may be incubating infection with a disease agent of biosecurity concern at the time of semen collection, it is recommended that serological testing of donors be conducted between 30 and 45 days after semen collection. This policy review also recommends pre-collection vaccination of semen donors against leptospirosis as an appropriate biosecurity measure.

Table 1 provides a summary of the biosecurity measures for each disease of biosecurity concern under the previous and revised policies. Full details of the review and conclusions for each disease are provided in Chapter 4. Recommended biosecurity measures are provided in Chapter 6.

---

<sup>1</sup> This PAQ requirement does not apply to dogs and cats imported from Cocos (Keeling) Islands, New Zealand or Norfolk Island.

Implementation of the revised policy will be subject to the development of appropriate operational conditions. Until notified otherwise, the import conditions under the previous policy continue to apply for the importation of dogs and cats and their semen from approved countries. Further information about implementation is available at [daff.gov.au/catsanddogs](http://daff.gov.au/catsanddogs).

Table 1 Summary comparison of biosecurity measures under the previous and revised policies for the importation of dogs and cats and their semen

Dogs and cats	Biosecurity measures—previous policy					Biosecurity measures—revised policy	
Rabies	Category 1 Country free, no PAQ	Category 2 Country free, 30 days PAQ	Category 3 Country free, 60 days PAQ	Category 4 Vaccination, RNATT <sup>a</sup> , min 30–120 days PAQ	Category 5 Vaccination, RNATT, 90 days PEQ, min 30–120 days PAQ	Category 1 & 2 Country free, no PAQ	Category 3 Vaccination, RNATT <sup>b</sup> 180 days pre-export, no PAQ
Canine brucellosis*	(Dogs) Pre-export serology with negative results					(Breeding dogs) As for previous policy (Desexed dogs) Documented evidence of desexing	
Canine influenza	(Dogs from the United States) Pre-export vaccination					As for previous policy	
Canine monocytic ehrlichiosis*	(Dogs) Pre-export serology with negative results. Treatment against tick vectors administered at the time of blood sample collection					Serology as for previous policy. Treatment against tick vectors to begin 21 days before blood sample collection	
Leishmaniasis*	(Dogs) Pre-export serology with negative results					As for previous policy	
Leptospirosis*	(Dogs) Pre-export serology with negative results Repeat testing for weak seropositives, serial testing for vaccinates					Pre-export serology with negative results or vaccination or treatment	
Piroplasmosis	(Dogs that have been on the African continent) Pre-export treatment					As for previous policy	
Nipah virus encephalitis	(Dogs, cats from Malaysia) Pre-export serology with negative results					Serology requirement removed	
External parasites* <sup>c</sup>	(Dogs) Pre-export parasiticide treatment at blood sample collection for <i>Ehrlichia canis</i> , repeated to provide continuous protection and again within 4 days of export. (Cats) Pre-export parasiticide treatment within 4 days of export					Min 10 days PAQ (Dogs) Parasiticide treatment to commence at least 21 days before blood sample collection for <i>E. canis</i> , repeated to provide continuous protection until at least the day of export. (Cats) Parasiticide treatment at least 21 days before export, repeated to provide continuous protection until at least the day of export	
Internal parasites*	(Dogs, cats) Single pre-export parasiticide treatment within 4 days of export					Two parasiticide treatments at an interval of not less than 14 days; the second within 5 days of export	
Dog semen	Biosecurity measures—previous policy					Biosecurity measures—revised policy	
Canine brucellosis*	(Donor dogs) Pre-collection serology with negative results					Post-collection serology with negative results	
Leishmaniasis*	(Donor dogs) No biosecurity measures					Post-collection serology with negative results	
Leptospirosis*	(Donor dogs) Pre-collection serology with negative results					Vaccination pre-collection or post-collection serology with negative results	
Rabies	(Donor dogs) Country freedom or vaccination					Rabies requirements do not apply	

PAQ = post-arrival quarantine; PEQ = pre-export quarantine; RNATT = rabies neutralising antibody titre test

\* Modified requirements apply to animals imported from New Zealand, Cocos (Keeling) Islands and Norfolk Island

<sup>a</sup> Required between 3–12 months before export in previous policy

<sup>b</sup> Required between 6–24 months before export in revised policy

<sup>c</sup> Risk management for external parasites also provides risk management for canine ehrlichiosis, hepatozoonosis, Lyme disease, piroplasmosis, screw-worm fly myiasis, tularaemia and yersiniosis

## References

WHO (World Health Organization) (2013). Rabies. WHO, Geneva.  
<http://www.who.int/zoonoses/diseases/rabies/en/> (accessed 20 May 2013)

# 1 Introduction

---

## 1.1 Background

Thousands of dogs and cats are imported into Australia each year, accompanied by an import permit that specifies the detailed biosecurity preparations and documentation necessary for importation.

Rabies is the most significant disease of biosecurity concern associated with the importation of dogs and cats, primarily due to the fatal consequences of rabies virus infection in all mammals, including humans. Under the previous biosecurity policy risk management of dogs was required for canine brucellosis, canine influenza (United States only), canine monocytic ehrlichiosis, canine piroplasmosis, leishmaniasis, leptospirosis and Nipah virus encephalitis (Malaysia only). For cats, in addition to rabies, risk management was previously required for Nipah virus encephalitis (Malaysia only). The previous major review of Australian biosecurity requirements for the importation of dogs and cats was completed in 1993 (BRS 1993). Biosecurity requirements for the importation of dog and cat semen were amended in 1997 to align with the requirements for the importation of live dogs and cats. A number of minor (disease-specific) reviews have since been conducted, including for canine influenza, leishmaniasis and leptospirosis, with associated amendments to biosecurity policy.

The importation of live animals necessarily entails some risk, as all animals have the potential to harbour infections that may not be detectable by either clinical examination or a non-invasive laboratory test procedure, such as serology.

Since 1993, an estimated 80 000 dogs and cats have been imported into Australia, largely without any incident of biosecurity concern. In 2001, *Babesia gibsoni*, a canine piroplasm previously unreported in Australia was detected in three dogs in Victoria that had been in direct contact with an imported bull terrier. A cluster of 14 dogs, seropositive for *B. gibsoni*, was subsequently identified (Jefferies et al. 2007). Although the consequences of the outbreak of *B. gibsoni* were relatively minor, its occurrence provides an example of the potential for incursions by exotic disease agents associated with live animal imports.

In this policy review, the biosecurity requirements for the importation of dogs and cats and their semen under the previous policy were reviewed by the Australian Government Department of Agriculture, with due regard to their appropriateness to maintain Australia's biosecurity. On 26 July 2012, a draft policy review was released for a public consultation period of 60 days, with the comment period closing on 24 September 2012.

In reviewing Australia's previous biosecurity policy, consideration was given to current scientific information, international standards developed by the World Organisation for Animal Health (OIE), biosecurity measures adopted by other

countries, Australian experience in the importation of dogs and cats and submissions received from stakeholders. Consequently, this policy review recommends updated biosecurity measures that reflect current scientific knowledge and, when implemented, the Department of Agriculture considers would achieve Australia's appropriate level of protection (ALOP) for the safe importation of dogs and cats, while also streamlining the importation process as much as possible.

## **1.2 Australia's biosecurity policy**

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

The Department of Agriculture is responsible for developing and reviewing biosecurity policy for the importation of animals and their products. This is done through a science-based risk evaluation process. At the completion of the risk evaluation process and following consideration of stakeholder comments, the Department of Agriculture is responsible for developing and implementing an import protocol, including any biosecurity measures.

The Department of Agriculture's science-based risk evaluation process is consistent with Australian Government policy, and Australia's rights and obligations under the World Trade Organization's (WTO) Agreement on the Application of Sanitary and Phytosanitary Measures.

Australia implements a risk-based approach to biosecurity management. This approach is expressed in terms of Australia's ALOP, which reflects community expectations through government policy and aims to reduce risks to a very low level but not to zero.

If the level of risk associated with an importation is deemed to exceed Australia's ALOP, biosecurity measures are recommended to reduce the risk to an acceptable level. However, if it is not possible to reduce the level of risk to an acceptable level, then importation will not be allowed.

## **1.3 Scope**

This policy review assesses the biosecurity risks posed by disease agents associated with the importation into Australia of:

- dogs and cats from New Zealand, Cocos (Keeling) Islands and Norfolk Island
- dogs and cats from all other approved countries
- disability assistance dogs
- dogs for emergency relief work following disaster in Australia
- Australian dogs returning from relief work following disasters in other countries

- dog and cat semen

Dogs and cats are defined as *Canis lupus familiaris* (domestic dog) and *Felis catus* (domestic cat).

In addition to the biosecurity requirements for dogs and cats administered by the Department of Agriculture, the import of live animals into Australia is also regulated under the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act), which is administered by the Australian Government Department of Sustainability, Environment, Water, Population and Communities (DSEWPaC). Under the EPBC Act, live specimens may only be imported if they appear on the List of Specimens taken to be Suitable for Live Import (live import list).

As a rule, the potential risks that some hybrids present to the Australian environment means that hybrid dogs and cats are not eligible for import unless they are specifically listed on the live import list. A hybrid is defined as the result of interbreeding between a domestic dog or cat and a wild species or wild sub-species of dog or cat, regardless of the generational distance from the wild specimen/s—for example a wolf-dog hybrid or Savannah cat.

The Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES)—an international agreement between governments to ensure that international trade in specimens of wild animals and plants does not threaten their survival—is also implemented in Australia under the EPBC Act by DSEWPaC. Any dog or cat that is a hybrid of a species included in CITES must be at least four generations removed from that species, or it is subject to the provisions of CITES as if it were the original species. This definition is relevant for the regulation of trade in endangered species, but is not relevant to regulatory arrangements put in place to manage environmental risks associated with the import of exotic wildlife (including cats and dogs) to Australia.

## 1.4 Conditions for importation

Conditions for the importation of dogs and cats into Australia under the previous policy were established following a review of Australia's biosecurity policy for dogs and cats in 1993 and were primarily based around biosecurity measures for rabies.

Key aspects of the import conditions under the previous policy were that dogs and cats must meet the requirements specified in an import permit issued by the Department of Agriculture, and may only be exported to Australia from an approved country. A period of post-arrival quarantine (PAQ) was required, which varied from 30 to 120 days, depending primarily on the country of export.

Details of import conditions under the previous policy for dogs and cats can be accessed on the the Department of Agriculture website at [daff.gov.au/aqis/cat-dogs](http://daff.gov.au/aqis/cat-dogs).

The *Quarantine Act 1908* and its subordinate legislation provide the legal basis under which biosecurity requirements for the importation into Australia of live animals,

and products derived from animals, are regulated. The Department of Agriculture implements and administers these requirements.

In addition to rabies-specific requirements for dogs and cats imported from rabies-affected countries, serological testing and parasite treatments apply to minimise the risk of entry of other pests and diseases of biosecurity concern.

Because of its favourable animal health status, neither an import permit nor PAQ is required for the importation of dogs and cats from New Zealand. Dogs and cats must have been continuously resident in New Zealand for a minimum period of 90 days to be eligible for export to Australia and must be accompanied by a New Zealand Government veterinary certificate confirming that the animal has undergone pre-export treatment and inspection, in accordance with Australian requirements.

A brief overview of the development of Australia's biosecurity requirements for dogs and cats under the previous policy is summarised in the following section.

## 1.5 Biosecurity policy since 1993

In 1993, the Australian Quarantine and Inspection Service (AQIS) commissioned the Bureau of Resource Sciences (BRS) to undertake a *Review of quarantine policy for dogs and cats, with particular reference to rabies*. The BRS working group recommended the development and implementation of a five-category system for classification of countries with respect to rabies animal health status.

In addition, the BRS working group recommended that in rabies-endemic countries, licensed private facilities could be used as pre-export quarantine premises under direct government supervision.

In November 1995, *Animal Quarantine Policy Memorandum (AQPM) 1995/68* advised stakeholders of redefined country categories and revised conditions for the importation of dogs and cats, based on the 1993 working group's recommendations.

In 1996, AQPM 96/33 clarified the criteria for classifying approved countries in redefined categories as follows:

- Category 1—rabies status considered equivalent to Australia, i.e. Cocos (Keeling) Islands, Norfolk Island and New Zealand
- Category 2—approved rabies-free countries
- Category 3—approved rabies-free island countries
- Category 4—approved countries where dog-mediated rabies is absent or well controlled
- Category 5—approved countries where dog-mediated rabies is endemic.

Details of the categorisation of approved countries under the previous policy are provided in Appendix 1.

AQPM 1996/33 also advised that the Republic of South Africa had satisfied the following criteria for approval as a Category 5 country:

- AQIS evaluation and approval of the country's Veterinary Services as competent to provide reliable certification for dogs and cats
- the country's rabies status has been established, the country monitors and reports rabies, and they may have active programs to control the disease on a regional or national basis
- the country has an approved rabies-free facility to hold dogs and cats before export
- the country has provided AQIS with details of their rabies status, including any control programs and their Veterinary Services.

From 1996, biosecurity risks associated with canine piroplasmiasis were reviewed and risk management for *Babesia canis rossi* was implemented for dogs imported from the Republic of South Africa (AQPM 1997/85).

AQPM 1999/62 provided details of the criteria Australia takes into account when considering the approval of a country to export animals and/or animal products to Australia, including:

- the animal health status of the country
- the effectiveness of Veterinary Services and other relevant certifying authorities
- legislative controls over animal health, including biosecurity policies and practices
- the standard of reporting to the OIE of major contagious disease outbreaks
- the effectiveness of veterinary laboratory services, including compliance with relevant international standards
- the effectiveness of systems for control over certification/documentation of products intended for export to Australia.

In June 1997, notification of revised conditions (AQPM 1997/40) for the importation of dog and cat semen was provided to align them with conditions for the importation of live dogs and cats.

In March 1999, commencement of an import risk analysis (IRA) for dogs and cats to review biosecurity policy in light of scientific advances was announced (AQPM 1999/18) and a Technical Issues Paper was released for stakeholder comment in October 2001 (ABPM 2001/29). Resource constraints prevented the completion of further substantial work on the IRA for dogs and cats.

In 2000, separate conditions were introduced (AQPM 2000/22) for the importation of disability assistance dogs. These import conditions specifically apply to circumstances in which an importer has a demonstrated disability for which daily assistance of an appropriately trained dog is essential for the importer's welfare.

Other amendments to the biosecurity policy for dogs included the addition of quarantine requirements for leishmaniasis in 2006 (*Biosecurity Australia Policy Memorandum 2006/04*) and canine influenza in 2010.

## 1.6 Quarantine facilities and demand

Australia currently has three quarantine stations for the importation of dogs and cats—Eastern Creek Quarantine Station (Sydney, New South Wales), Spotswood Quarantine Station (Melbourne, Victoria) and Byford Quarantine Station (Perth, Western Australia). The majority of dogs and cats are imported into Eastern Creek and Spotswood Quarantine Stations.

Annual data (aggregated) for the importation of dogs and cats from 2010–2012 requiring PAQ are shown in Table 2. The majority of import permits issued (approximately 60%) were for dogs and cats from the United Kingdom and the United States. Approximately a further 15% of import permits were issued for dogs and cats from Canada, the Republic of South Africa and Singapore.

Table 2 Dog and cat imports into Australian quarantine facilities (2010–2012)

Year	Dogs	Cats
2010	3604	1871
2011	3935	2254
2012	3600	1722

## 1.7 Potentially affected Australian sectors

Australian sectors potentially affected by changes to the dog and cat biosecurity policy can be divided into the companion dog and cat sector, the working and racing dog sector, the breeding dog and cat sector, and associated production and retail industries.

## 1.8 Native fauna

Australian native fauna include representatives of three mammalian subclasses: monotremes (Prototherian), marsupials (Metatherian) and placentals (Eutherian). Species in each subclass are classified as threatened under the EPBC Act. Australia's diverse native fauna is unique and has an extremely high conservation value. If the importation of dogs and cats and their semen were to result in the establishment of an exotic disease agent to which native fauna are susceptible, it is plausible that establishment or spread of the introduced disease agent may have a significant adverse impact on biodiversity.

## References

Australian Companion Animal Council Inc. (2010). Pet ownership statistics. Australian Companions Animal Council Inc, Sydney.  
[http://www.acac.org.au/pet\\_care.html](http://www.acac.org.au/pet_care.html) (accessed 16 December 2011)

ANKC (Australian National Kennel Council) (2010). National animal registration analysis 1986–2010., National registration statistics.  
<http://www.ankc.org.au/National-Registration-Statistics.aspx> (accessed 30 March 2010)

BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper. BRS, Canberra.

Hill M (2006). *Contribution of the pet care industry to the Australian economy, 2006*, M4612/MH. Australian Companion Animal Council Inc., Sydney.

Jefferies R, Ryan U, Jardine J, Broughton DK, Robertson ID, Irwin PJ (2007). Blood, bull terriers and babesiosis: further evidence for direct transmission of *Babesia gibsoni* in dogs. *Australian Veterinary Journal* 85: 459–463.

NSW CFA (New South Wales Cat Fanciers' Association) (2009). Welcome to NSW Cat Fanciers' Association. NSW Cat Fanciers' Association.  
<http://www.nswcfa.asn.au> (accessed 16 September 2009)



## 2 Method

---

### 2.1 Background

The World Organisation for Animal Health (OIE), in its *Terrestrial animal health code* (the Code; OIE 2011), describes 'General obligations related to certification' in Chapter 5.1.

The Code states in Article 5.1.2. that:

The import requirements included in the international veterinary certificate should assure that commodities introduced into the importing country comply with the OIE standards. Importing countries should restrict their requirements to those necessary to achieve the national appropriate level of protection (ALOP). If these are stricter than the OIE standards, they should be based on an import risk analysis.

Article 5.1.2. further states that:

The international veterinary 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 risk analysis as described in Chapter 2.1. of the 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 policy review. Risk communication is conducted as an ongoing process, and includes both formal and informal consultation with stakeholders.

### 2.2 Risk review

Although not defined or described in the Code, risk review is recognised by risk analysts as an essential component of the risk analysis process (Barry 2007; Brett et al. 1989; FSA 2006).

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 currently apply.

Risk review differs from the monitoring and review component of risk management, as described in the Code, in that each component of the IRA 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 technical information, risk management measures can be revised accordingly.

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

- the Code (OIE 2011)
- *Review of quarantine policy for dogs and cats, with particular reference to rabies* (BRS 1993)
- Technical Issues Paper for the dog and cat import risk analysis (ABPM 2001/29)
- The Department of Agriculture import conditions for the importation of dogs and cats into Australia
- The Department of Agriculture import conditions for the importation of dog and cat semen into Australia
- a review of relevant scientific literature.

Risk, defined by the 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' (OIE 2011), is dynamic in nature; it changes with time. Consequently, risk should be kept under regular review.

## 2.3 Review of hazard identification

Hazard identification is described in the 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 (OIE 2011).

In accordance with the Code, a disease agent was considered to be a potential hazard relevant to the importation of dogs and cats 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.

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

- it was not present in Australia, or present in Australia and subject to official control or eradication
- there was clear evidence of transmission via dogs and cats, and/or their semen.

This policy review considered the potential hazards identified in the 2001 Technical Issues Paper. Evaluation of the current scientific literature was conducted to

determine if hazards identified in 2001 should be retained for further consideration and whether additional hazards should be added.

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 dogs and cats or their semen in disease transmission.

In addition, all disease agents for which biosecurity measures applied under The Department of Agriculture's conditions for the importation of dogs and cats and/or their semen were included and retained for further review.

The steps involved in hazard identification and refinement are shown in Figure 1.

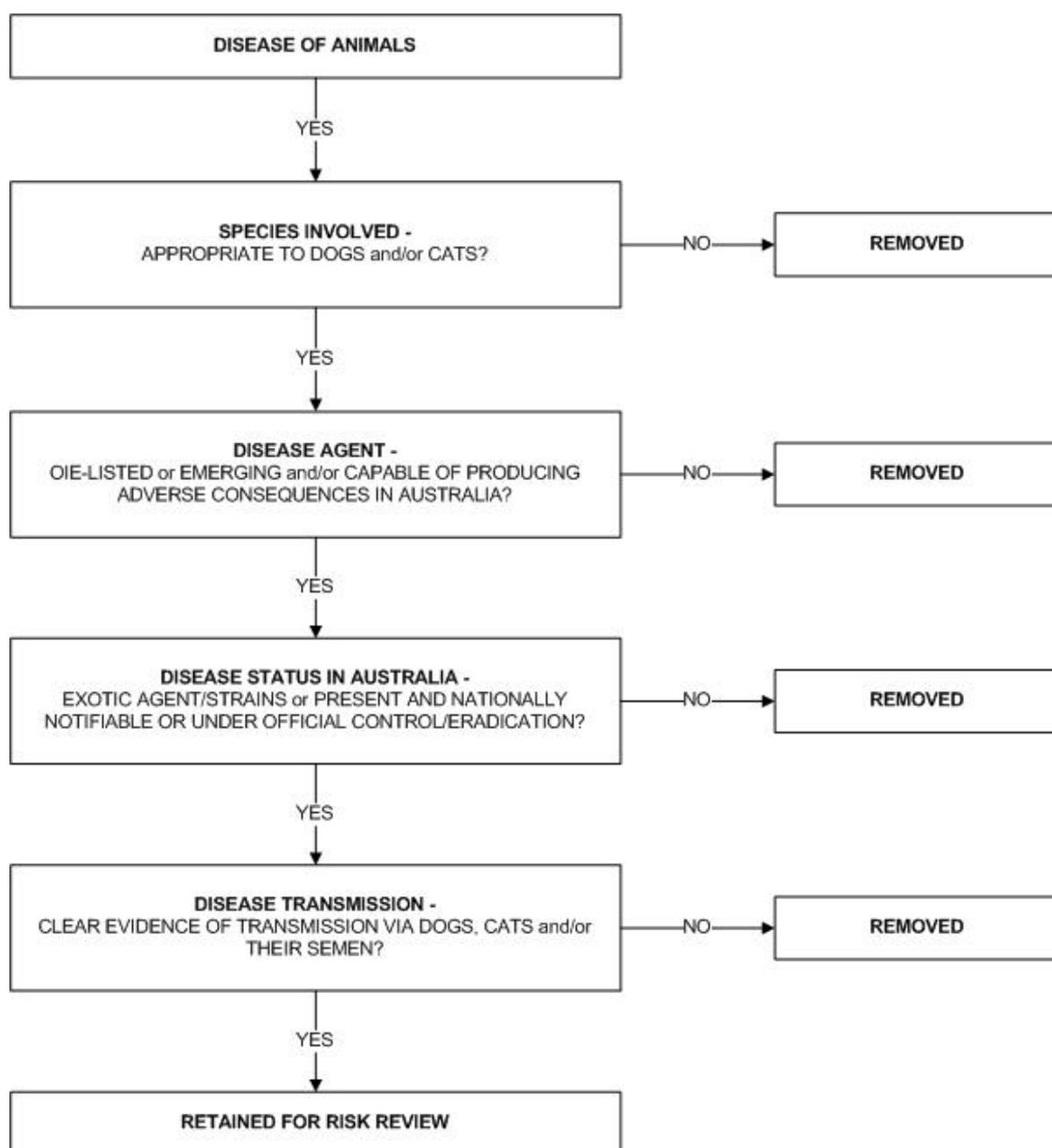


Figure 1 Decision tree for hazard identification and refinement

## 2.4 Review of risk assessment

For each hazard retained for further assessment, a review of the scientific literature was performed to identify any evidence of a significant change in the the risk factors relevant to the release, exposure and consequence assessment of the hazard that would be relevant to biosecurity considerations for Australia. The advice of experts with specialist knowledge of disease agents was also obtained in some instances.

If definitive information on risk factors was not found through literature review or contact with relevant experts, any uncertainties were identified and documented.

Based on the information reviewed, a conclusion was made for each hazard regarding whether a significant change in biosecurity risk had occurred that was relevant to the importation into Australia of dogs and cats and/or their semen. Assumptions and/or judgements made in drawing conclusions for each hazard retained for further review were documented in the relevant risk review section (see Chapter 4).

## 2.5 Review of risk management

The Code (Chapter 2.1.) divides risk management into four processes (OIE 2011):

1. *Risk evaluation—the process of comparing the risk estimated in the risk assessment with the member’s ALOP.*

In this policy review, it was assumed that Australia’s ALOP had not changed significantly since the last major review of biosecurity policy for dogs and cats. The conclusions drawn from the risk reviews conducted for each hazard were used as the basis for risk evaluation. A judgement was then made to determine whether risk management was warranted to achieve Australia’s ALOP. This method was considered to be appropriate to evaluate the biosecurity risks associated with the previous policy for the importation of dogs and cats into Australia.

2. *Option evaluation—the process of identifying, evaluating the efficacy and feasibility of, and selecting measures to reduce, the risk associated with an importation to bring it in line with the member’s ALOP. The efficacy is the degree to which an option reduces the likelihood and/or magnitude of adverse health and economic consequences. Evaluating the efficacy of the options selected is an iterative process that involves their incorporation into the risk assessment and then comparing the resulting level of risk with that considered acceptable. The evaluation for feasibility normally focuses on technical, operational and economic factors affecting the implementation of the risk management options.*

In this policy review, a detailed review of risk management options for rabies, the most significant biosecurity hazard associated with the importation of dogs and cats, was undertaken and documented (see Appendix 2). Reviews of risk management options for each other hazard retained for further assessment were

also undertaken and documented in the relevant risk review section (see Chapter 4).

3. *Implementation—the process of following through with the risk management decision and ensuring that the risk management measures are in place.*

For each hazard retained for further assessment, this policy review evaluated whether risk management was warranted for either the importation of dogs and cats, or their semen, or both. If it was concluded that risk management was warranted, then biosecurity measures under the previous policy were reviewed to determine if they were appropriate. If it was concluded that those biosecurity measures were not appropriate to achieve Australia's ALOP, alternative and/or complementary biosecurity measures were proposed.

4. *Monitoring and review—the ongoing process by which the risk management measures are continuously audited to ensure that they are achieving the results intended.*

The Department of Agriculture is responsible for implementing, monitoring and reviewing biosecurity measures to enable the safe importation of commodities into Australia, including dogs and cats, and their semen.

The biosecurity measures under the previous policy were reviewed in the context of updated scientific information, including expert advice where available, as well as operational practicality. Stakeholder submissions received in the consultation phase of this policy review provided guidance to identify issues of concern relevant to the importation of dogs and cats and their semen from approved countries.

## **2.6 Risk communication**

Risk communication is defined by the 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' (OIE 2011).

In conducting IRAs and policy reviews, the Department of Agriculture consults directly with the Australian Government Department of Health and Ageing 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 IRAs and policy reviews to enable stakeholder assessment and feedback on draft conclusions and recommendations about Australia's animal biosecurity policies.

## References

Barry M (2007). *Effective approaches to risk assessment in social work: an international literature review*. Education Information and Analytical Services, Scottish Executive, Edinburgh.

Brett SM, Rodricks JV, Chinchilli VM (1989). Review and update of leukemia risk potentially associated with occupational exposure to benzene. *Environmental Health Perspectives* 82:267–281.

FSA (Financial Services Authority) (2006). *The FSA's risk-assessment framework*. FSA, London.

OIE (World Organisation for Animal Health) (2011). *Terrestrial animal health code*. OIE, Paris.

<http://www.oie.int/en/international-standard-setting/terrestrial-code/access-online/> (accessed 3 November 2011)

### 3 Hazard identification

---

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

- diseases listed by the OIE as affecting dogs and/or cats (OIE 2012)
- diseases identified in the 2001 Technical Issues Paper
- other diseases identified as occurring in dogs and/or cats.

The method of hazard identification and refinement is described in Section 2.3. The preliminary list of diseases/disease agents is shown in Table 3.

Table 3 summarises the results of the hazard refinement process, including the reason for removal or retention of each identified hazard.

Due to their largely ubiquitous occurrence and the numerous species, external parasites (e.g. ticks, fleas, mites) and internal parasites (e.g. helminths, nematodes) were not specifically included in the hazard identification list (Table 3), with the exception of parasitic diseases that are either OIE-listed or were considered in the context of emerging threats to biosecurity. However, general consideration of both external and internal parasites, and their relevance to biosecurity risk, is provided in Sections 4.13 and 4.14, respectively.

Many disease agents of potential biosecurity concern associated with the importation of dogs and cats are opportunistic or ubiquitous, and/or the relevance of dogs and cats in disease epidemiology is uncertain due to limited or insufficient information. It was appropriate to list these disease agents here, not only to indicate that they were considered, but also in the event that significant evidence of the role of dogs and/or cats in disease spread is identified following completion of this review. These agents include:

- Viruses: Barmah Forest virus, Bunyaviridae (including California encephalitis group virus and Crimean-Congo haemorrhagic fever virus), canine acidophil hepatitis virus, enterovirus, feline foamy (syncytium-forming) virus, Getah virus, influenza viruses (type A, B and C), Murray Valley encephalitis virus, mumps virus, papillomavirus, Ross River virus, severe acute respiratory syndrome coronavirus, Sindbis virus, Western equine encephalomyelitis virus and yellow fever virus.
- Bacteria: *Actinomyces* spp., *Campylobacter* spp., *Clostridium* spp., *Corynebacterium* spp., *Dermatophilus congolensis*, *Ehrlichia* from *Ixodes ovatus*, *Ehrlichia muris*, *Escherichia coli*, *Helicobacter* spp., *Listeria monocytogenes*, *Mycobacterium* spp., *Mycoplasma* spp., *Neorickettsia sennetsu*, *Nocardia* spp., *Rhodococcus equi*, *Salmonella* spp., *Shigella* spp., *Streptobacillus moniliformis*, *Streptococcus* spp., *Vibrio* spp., *Wolbachia* spp. and *Yersinia* spp.
- Protozoa: *Cryptosporidia* spp., *Giardia* spp., *Hammondia* spp., *Isospora* spp., *Neospora caninum*, *Pentatrachomonas hominis*, *Prototheca* spp., *Sarcocystis* spp., *Tetratrachomonas felistomae* and *Tritrachomonas foetus*.

- Algae and fungi: *Aspergillus* spp., *Candida* spp., *Cryptococcus* spp., *Histoplasma* spp., *Microsporium* spp., *Pythium insidiosum*, *Rhinosporidium seeberi*, *Sporothrix schenckii*, *Trichophyton* spp. and *Trichosporon* spp.
- Chlamydia: *Chlamydophila* spp.
- Other: *Acanthamoeba* spp. and canine transmissible venereal tumour.

Table 3 Hazard identification and refinement for diseases affecting dogs and/or cats

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
African horse sickness (African horse sickness virus)	Dogs and equids	Yes	Yes	No	No. The role of dogs in disease epidemiology is not significant <sup>1, 17, 72</sup>
Anthrax ( <i>Bacillus anthracis</i> )	Mammals	Yes	Yes	Yes	No. Present in Australia but with official control measures. There are no recommendations in the Code for dogs or cats. Dogs and cats are incidental hosts <sup>75, 81</sup>
Aujeszky's disease (suid herpesvirus 1)	Multiple species including pigs, ruminants, cats, dogs and rats	Yes	Yes	No	No. The Code contains recommendations for only pigs and swine. Species other than pigs considered to be dead-end hosts <sup>82, 86, 104</sup>
Besnoitiosis ( <i>Besnoitia</i> spp.)	Mammals including cats	No	Yes	<i>B. wallacei</i> present; other species have not been isolated	No. The definitive host of <i>B. besnoiti</i> has not been identified. Cats have been identified as a definitive host for a number of <i>Besnoitia</i> spp., but their role in disease epidemiology has not been established <sup>10, 37, 40, 42, 43, 48, 71, 78, 98</sup>
Blastomycosis ( <i>Blastomyces dermatitidis</i> )	Multiple species including canids, felids, horses and humans	No	Yes	No	No. <i>B. dermatitidis</i> is a soil saprophyte; spread occurs via contaminated soil – specific biosecurity requirements apply for soil. No mammalian reservoir hosts have been identified <sup>8, 67</sup>
Bluetongue (bluetongue virus)	African carnivores, cats, dogs and ruminants	Yes	Yes	Some serotypes present	No. The role of dogs and cats in disease epidemiology is not significant <sup>2, 85</sup>
Borna disease (Borna disease virus)	Cats, cattle, horses, humans, llamas, ostriches and sheep	No	Yes	Not isolated	No. Cats seropositive to Borna-like virus in SA and NSW; however, no virus has been isolated in Australia or from cases of staggering disease in cats overseas <sup>52, 61</sup>
Bovine tuberculosis ( <i>Mycobacterium bovis</i> )	Multiple species including livestock, humans, cats and dogs,	Yes	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>22, 35, 54, 73, 74, 103</sup>
Brucellosis ( <i>Brucella abortus</i> )	Cattle, dogs and humans	Yes	Yes	No	Yes—reviewed with canine brucellosis. Meets criteria for listing as a potential hazard

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Canine bartonellosis ( <i>Bartonella vinsonii</i> subsp. <i>berkhoffii</i> )	Canids, cats and humans	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Canine brucellosis ( <i>Brucella</i> spp.)	Canids and humans	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Canine distemper (canine distemper virus)	Canids and felids	No	Yes	Yes	No. Present in Australia
Canine granulocytic anaplasmosis ( <i>Anaplasma phagocytophilum</i> )	Mammals including canids, cats, horses, humans and ruminants	No	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>12, 23, 45</sup>
Canine granulocytic ehrlichiosis ( <i>Ehrlichia ewingii</i> )	Dogs, humans and white-tailed deer	No	Yes	No	No. Primary tick vector ( <i>Amblyomma americanum</i> ) is not present in Australia <sup>33, 106</sup>
Canine infectious tracheobronchitis, kennel cough (canine parainfluenza virus and <i>Bordetella bronchiseptica</i> –respiratory complex)	Cats, dogs and humans	No	Yes	Yes	No. Present in Australia
Canine influenza (canine influenza virus)	Dogs	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Canine monocytic ehrlichiosis ( <i>Ehrlichia canis</i> , <i>E. chaffeensis</i> )	Multiple species including cats, dogs and humans	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Canine pulmonary angiostrongylosis ( <i>Angiostrongylus vasorum</i> )	Canids	No	Yes	No	Yes. Meets the criteria for listing as a potential hazard
Canine respiratory, genital, neonatal herpesvirus infections (canine herpesvirus)	Canids	No	Yes	Yes	No. Present in Australia
Canine viral enteritis (canine coronavirus, canine rotavirus)	Canids	No	Yes	Yes	No. Present in Australia

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Canine viral enteritis, parvovirus infection (canine parvovirus 1, canine parvovirus 2)	Canids and felids	No	Yes	Yes	No. Present in Australia
Chagas' disease, American trypanosomiasis ( <i>Trypanosoma cruzi</i> )	Mammals including cats, dogs and humans	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Coccidioidomycosis ( <i>Coccidioides immitis</i> and <i>C. posadasii</i> )	Mammals including cats, dogs and humans	No	Yes	No	No. Not commonly a transmissible disease. Zoonotic infection is rare <sup>6, 31, 47, 50, 64, 66, 97</sup>
Cytauxzoonosis ( <i>Cytauxzoon felis</i> )	Felids	No	Yes	No	No. The role of cats in disease epidemiology has not been established. Proposed tick vectors ( <i>Amblyomma americanum</i> and <i>Dermacentor variabilis</i> ) are not present in Australia <sup>11, 13, 21, 90</sup>
Eastern equine encephalomyelitis (eastern equine encephalomyelitis virus)	Birds, equids and other animals including dogs and humans	Yes	Yes	No	No. The role of dogs in disease epidemiology is not significant <sup>4, 46, 107</sup>
Echinococcosis/hydatidosis ( <i>Echinococcus multilocularis</i> , <i>E. oligarthrus</i> , <i>E. granulosus</i> , <i>E. shiquicus</i> , <i>E. vogeli</i> )	Canids, felids and humans	Yes	Yes	Species other than <i>E. granulosus</i> absent	Yes—treatment reviewed with internal parasites. Meets criteria for listing as a potential hazard
Encephalitozoonosis ( <i>Encephalitozoon cuniculi</i> )	Mammals including canids, cats, humans, mice and rabbits	No	Yes	Yes	No. Present in Australia

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Equine protozoal myeloencephalitis ( <i>Sarcocystis neurona</i> )	Multiple species including cats, dogs, horses, marine mammals, mink, American opossums, raccoons and skunks	No	Yes	Yes	No. The definitive hosts (opossums) are not present in Australia. The role of dogs and cats in disease epidemiology has not been established <sup>39, 41, 58, 99</sup>
Feline AIDS (feline immunodeficiency virus)	Felids	No	Yes	Yes	No. Present in Australia
Feline bartonellosis (cat scratch disease) ( <i>Bartonella</i> spp.)	Cats, dogs and humans	No	Yes	Yes	No. <i>B. henslae</i> present in Australia. <i>B. clarridgeiae</i> DNA identified in cat blood and vector, <i>Ctenocephalides felis</i> in Australia. The role of cats in the epidemiology of <i>B. bovis</i> , <i>B. koehlariae</i> and <i>B. quintana</i> infection has not been established <sup>7, 18</sup>
Feline infectious peritonitis and coronavirus enteritis (feline coronavirus, feline infectious peritonitis virus)	Felids and pigs	No	Yes	Yes	No. Present in Australia
Feline leukaemia (feline leukaemia virus and feline sarcoma virus)	Cats	No	Yes	Yes	No. Present in Australia
Feline panleukopaenia, feline enteritis (feline parvovirus)	Felids, and some procyonids, mustelids and veverrids	No	Yes	Yes	No. Present in Australia
Feline respiratory disease (feline herpesvirus-1, feline calicivirus, <i>Bordetella bronchiseptica</i> )	Cats and dogs	No	Yes	Yes	No. Present in Australia

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Feline spongiform encephalopathy (prion spongiform encephalopathy agent)	Carnivorous mammals including felids, primates (including humans) and ruminants	No	Yes	No	No. No evidence of horizontal transmission. Ingestion of bovine spongiform encephalopathy contaminated feedstuffs necessary for transmission <sup>28, 105</sup>
Glanders ( <i>Burkholderia mallei</i> )	Equids, cats, dogs and humans	Yes	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>44, 80</sup>
Haemorrhagic fever with renal syndrome, hantavirus pulmonary syndrome (hantaviruses)	Rodents, cats, dogs and humans	No	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>30, 52</sup>
Heartwater ( <i>Ehrlichia ruminantium</i> )	Ruminants and other species including dogs	Yes	Yes	No	No. Pathogenic importance, infectivity and host range of the genotype reported in dogs has not been established <sup>3, 29</sup>
Hendra disease (Hendra virus)	Horses, cats, dogs <sup>a</sup> , humans and pteropid bats	No	Yes	Yes (sporadic occurrence)	No. Present in Australia
Hepatozoonosis ( <i>Hepatozoon canis</i> and <i>H. americanum</i> )	Dogs and possibly other canids, bobcats and ocelots	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Infectious canine cyclic thrombocytopaenia ( <i>Anaplasma platys</i> )	Dogs	No	Yes	Yes	No. Present in Australia <sup>20, 76</sup>
Infectious canine hepatitis (canine adenovirus –1) CAV respiratory disease (canine adenovirus –2)	Domestic and non-domestic carnivores	No	Yes	Yes	No. Present in Australia

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Japanese encephalitis (Japanese encephalitis virus)	Birds, reptiles and mammals including pigs, dogs and equids	Yes	Yes	Occasional cases occur in the Torres Strait (not connected to mainland Australia)	No. The role of dogs in disease epidemiology has not been established <sup>19</sup>
La Crosse encephalitis (La Crosse encephalitis virus)	Dogs, humans and small mammals (e.g. chipmunks)	No	Yes	No	No. Dogs rarely infected. The role of dogs in disease epidemiology has not been established <sup>16, 49, 102</sup>
Leishmaniasis ( <i>Leishmania</i> spp.—Old World and New World)	Multiple species including cats, dogs and humans	Yes	Yes	A novel species has been isolated in Australia	Yes. Meets criteria for listing as a potential hazard
Leptospirosis ( <i>Leptospira</i> spp.)	Multiple species including cats, dogs and humans	Yes	Yes	Multiple serovars present	Yes. Strains not present in Australia (serovar Canicola) meet criteria for listing as a potential hazard
Louping ill (louping ill virus)	Multiple species including cattle, deer, dogs, horses, humans, pigs and sheep	No	Yes	No	No. Dogs are considered incidental hosts and do not develop sufficient viraemia to infect tick vectors. No known vector species are present in Australia <sup>12, 26, 91, 100</sup>
Lyme disease ( <i>Borrelia burgdorferi</i> sensu lato)	Mammals including cats, dogs and humans, and birds	No	Yes	Not isolated	Yes. Meets criteria for listing as a potential hazard
Lyssavirus infection (non-rabies) (Australian bat lyssavirus, Duvenhage virus, European bat lyssavirus, Lagos bat virus, Mokola virus)	Bats and some mammals	No	Yes	Viruses other than Australian bat lyssavirus absent	No. Terrestrial mammals are spill over hosts for European bat lyssavirus strains. The role of dogs and cats in the disease epidemiology of other lyssaviruses has not been established <sup>55, 77, 95</sup>
Melioidosis ( <i>Burkholderia pseudomallei</i> )	Mammals including cats, dogs, horses and humans	No	Yes	Yes	No. Present in Australia

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Nagana ( <i>Trypanosoma brucei brucei</i> , <i>T. congolense</i> , <i>T. vivax</i> )	Mammals including cats, dogs and humans	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
New World screw-worm ( <i>Cochliomyia hominivorax</i> )	Mammals	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
Newcastle disease (Newcastle disease virus)	Birds and mammals including cats and dogs	Yes	Yes	No	No. The role of cats and dogs in disease epidemiology has not been established <sup>14, 65</sup>
Nipah virus encephalitis (Nipah virus)	Cats, dogs, horses, humans, pigs and pteropid bats	No	Yes	No	Yes. Meets criteria for listing as a potential hazard
Old World screw-worm ( <i>Chrysomya bezziana</i> )	Mammals	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
Piroplasmiasis (infecting dogs and cats) ( <i>Babesia canis canis</i> , <i>B. canis rossii</i> , <i>B. canis vogeli</i> , <i>B. gibsoni</i> , <i>B. conradae</i> , <i>T. annae</i> , <i>B. felis</i> , <i>B. cati</i> , <i>B. herpailuri</i> , <i>B. pantherae</i> )	Multiple species including cats, dogs and humans	No	Yes	<i>B. canis vogeli</i> and <i>B. gibsoni</i> present; other species absent	Yes. Meets criteria for listing as a potential hazard
Potomac horse fever, equine monocytic ehrlichiosis ( <i>Neorickettsia risticii</i> )	Horses and other species, including cats, dogs, cattle and pigs	No	Yes	No	No. Dogs and cats have been proposed as reservoir hosts, but their role in disease epidemiology has not been established <sup>34, 76, 87, 89, 93</sup>
Powassan encephalitis, tick-borne encephalitis (Powassan virus)	Medium-sized wild mammals (rodents, skunks, woodchucks), cats, dogs and humans	No	Yes	No	No. Role of dogs and cats in disease epidemiology has not been established <sup>38, 51, 59, 63</sup>

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Poxvirus infection (cow poxvirus)	Small mammals (rodents), cats, dogs, cattle and humans	No	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>9, 24</sup>
Q fever ( <i>Coxiella burnetii</i> )	Multiple species	Yes	Yes	Yes	No. Present in Australia
Rabies (rabies virus genotype 1)	Mammals, including cats, dogs and humans	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
Rift Valley fever (Rift Valley fever virus)	Ruminants, cats, dogs and humans	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
Rocky Mountain spotted fever (RMSF), Mediterranean spotted fever (MSF), Boutonneuse fever ( <i>Rickettsia conorii</i> , <i>R. rickettsii</i> , other spotted fever group Rickettsias)	Cats, dogs and humans	No	Yes	Not reported	No—see external parasites. Dogs and cats do not have a significant role in disease epidemiology. The main vector of RMSF ( <i>Dermacentor</i> spp.) is not present in Australia and, although a vector ( <i>Rhipicephalus sanguineus</i> ) for RMSF and MSF is present in Australia, it is not regarded as the primary vector for RMSF in the US. <sup>53, 94, 101</sup> Dogs were able to transmit <i>R. conorii</i> infection to ticks experimentally <sup>68</sup>
Salmon poisoning disease ( <i>Neorickettsia helminthoeca</i> )	Canids	No	Yes	No	No. Three-host life cycle is unlikely to be viable in Australia, as intermediate snail host is restricted to Pacific northwest of the US. Not considered to be highly contagious in dogs <sup>15, 57</sup>
St Louis encephalitis (St Louis encephalitis virus)	Birds, cats, dogs and humans	No	Yes	No	No. The role of dogs and cats in disease epidemiology has not been established <sup>92</sup>
Surra ( <i>Trypanosoma evansi</i> )	Mammals including cats, dogs, horses and livestock	Yes	Yes	No	Yes. Meets criteria for listing as a potential hazard
Tenshaw or Tensaw disease (Tenshaw or Tensaw disease virus)	Canids, cats, humans, rabbits, raccoons and small rodents	No	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>51</sup>

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Tick-borne encephalitis (tick-borne encephalitis virus)	Dogs, humans, rodents and ruminants	No	Yes	No	No. The role of dogs in disease epidemiology is not significant <sup>70, 88</sup>
Toxoplasmosis ( <i>Toxoplasma gondii</i> )	Cats and warm-blooded mammals can act as the intermediate host	No	Yes	Yes	No. Present in Australia
Transmissible gastroenteritis (Transmissible gastroenteritis virus)	Pigs, cats, dogs and foxes	Yes	Yes	No	No. The role of dogs and cats in disease epidemiology is not significant <sup>83</sup>
Trichinellosis ( <i>Trichinella spiralis</i> )	Mammals, especially pigs and carnivores, including cats, dogs and humans	Yes	Yes	No	No. No evidence of horizontal transmission. Transmission would only occur if infected dog or cat muscle is ingested by susceptible species <sup>36, 62</sup>
Tuberculosis ( <i>Mycobacterium avium</i> complex, <i>M. lepraemurium</i> , <i>M. microti</i> , <i>M. tuberculosis</i> )	Multiple species including cats, dogs, humans and rodents	No	Yes	<i>M. tuberculosis</i> and <i>M. avium</i> complex present <i>M. microti</i> absent and novel feline leprosy has been identified in Australia	No. Cats are incidental hosts of <i>M. microti</i> . Other species present in Australia <sup>54, 56, 69</sup>
Tularaemia ( <i>Francisella tularensis</i> types A and B)	Multiple species including cats, dogs and humans	Yes	Yes	Type A absent. Two cases of Type B detected in humans in 2011	Yes. Meets criteria for listing as a potential hazard
Typhus (including murine) ( <i>Rickettsia felis</i> , <i>R. prowazekii</i> and <i>R. typhi</i> )	Cats, humans and rodents or small mammals	No	Yes	<i>R. typhus</i> present, <i>R. felis</i> presumed present, <i>R. prowazekii</i> absent	No—see external parasites. <i>R. typhus</i> present; <i>R. felis</i> DNA detected in fleas in Australia. The role of dogs in the epidemiology of <i>R. prowazekii</i> infection is not significant, but mammalian fleas may be able to transmit epidemic typhus <sup>7, 25, 60, 96</sup>

Disease (disease agent)	Susceptible species	OIE-listed disease <sup>84</sup> ?	Adverse consequences in Australia?	Present in Australia?	Retained for risk review?
Venezuelan equine encephalomyelitis (Venezuelan equine encephalitis virus)	Birds, equids and other species including dogs and humans	Yes	Yes	No	No. The role of dogs in disease epidemiology is not significant <sup>32</sup>
Wesselsbron disease (Wesselsbron disease virus)	Cattle, dogs, horses, humans, sheep and wildlife	No	Yes	No	No. Infection in dogs is rare and their role in disease epidemiology is not significant <sup>27</sup>
West Nile fever (West Nile virus)	Birds, equids and other animals including cats, dogs and humans	Yes	Yes	Yes	No. WNV strains present in Australia.
Yersiniosis (plague) ( <i>Yersinia pestis</i> )	Dogs, cats, humans and rodents	No	Yes	No	Yes. Meets criteria for listing as a potential hazard

NSW = New South Wales; SA = South Australia; Code = OIE *Terrestrial animal health code*; US = United States  
a One seropositive dog was identified in 2011

## Conclusion

The following diseases were retained for risk review (Chapter 4) on the basis of information provided in Table 3.

### OIE-listed diseases

- brucellosis (*Brucella abortus*)
- echinococcosis/hydatidosis—risk management reviewed with internal parasites
- leishmaniosis
- leptospirosis
- New World screw-worm (*Cochliomyia hominivorax*)
- Nipah virus encephalitis
- Old World screw-worm (*Chrysomya bezziana*)
- rabies
- Rift Valley fever
- surra

### Other diseases

#### Viruses

- canine influenza

#### Bacteria

- canine bartonellosis
- canine brucellosis (*Brucella canis*)
- canine monocytic ehrlichiosis
- Lyme disease
- tularaemia
- yersiniosis

#### Protozoa

- canine piroplasmosis
- Chagas' disease
- hepatozoonosis
- nagana

#### Nematodes

- canine pulmonary angiostrongylosis

## References

1. Alexander KA, Kat PW, House J, House C, O'Brien SJ, Laurenson MK, McNutt JW, Osburn BI (1995). African horse sickness and African carnivores. *Veterinary Microbiology* 47:133–140.
2. Alexander KA, MacLachlan NJ, Kat PW, House C, O'Brien SJ, Lerche NW, Sawyer M, Frank LG, Holekamp K, Smale L, McNutt JW, Laurenson MK, Mills MGL, Osburn BI (1994). Evidence of natural bluetongue virus infection among African carnivores. *The American Journal of Tropical Medicine and Hygiene* 51:568–576.
3. Allsopp MTEP, Allsopp BA (2001). Novel *Ehrlichia* genotype detected in dogs in South Africa. *Journal of Clinical Microbiology* 39:4204–4207.
4. Arrigo NC, Adams AP, Weaver SC (2010). Evolutionary patterns of eastern equine encephalitis virus in North versus South America suggest ecological differences and taxonomic revision. *Journal of Virology* 84:1014–1025.
5. Austgen LE, Bowen RA, Bunning ML, Davis BS, Mitchell CJ, Chang G-JJ (2004). Experimental infection of cats and dogs with West Nile virus. *Emerging Infectious Diseases* 10:82–86.
6. Baird RW, Teichtahl H, Ednie HM, Tasiopoulos A, Ryan N, Gee D (1999). A fluffy white traveller: imported *Coccidioides immitis* infection in an Australian tourist. *Pathology* 31:47–50.
7. Barrs VR, Beatty JA, Wilson BJ, Evans N, Gowan R, Baral RM, Lingard AE, Perkovic G, Hawley JR, Lappin MR (2010). Prevalence of *Bartonella* species, *Rickettsia felis*, haemoplasmas and the *Ehrlichia* group in the blood of cats and fleas in eastern Australia. *Australian Veterinary Journal* 88:160–165.
8. Baumgardner DJ, Paretsky DP, Baeseman ZJ, Schreiber A (2011). Effects of season and weather on blastomycosis in dogs: Northern Wisconsin, USA. *Medical Mycology* 49:49–55.
9. Bennett M, Gaskell RM, Baxby D (2006). Poxvirus infection. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.). Saunders, Elsevier, St. Louis, pp. 158–160.
10. Bigalke RD, Prozesky L (2004). Besnoitiosis. In *Infectious diseases of livestock*, 2nd edn, Coetzer JAW, Tustin RC (eds), Oxford University Press, Oxford, pp. 351–359.
11. Biosecurity Australia (2002). *Importation of non-domestic felidae into Australia: import risk analysis*, final report. Australian Government, Department of Agriculture, Fisheries and Forestry, Canberra.

12. Biosecurity Australia (2010). *Import risk analysis report for horses from approved countries: final report*. Biosecurity Australia, Canberra.
13. Blouin EF, Kocan AA, Kocan KM (1992). Development and transmission of *Cytauxzoon felis* by *Dermacentor variabilis*. In *First international conference on tick-borne pathogens at the host-vector interface: an agenda for research: proceedings and abstracts*, 15–18 September 1992, University of Minnesota, St Paul, Minnesota, pp. 75–81.
14. Bolin FM (1948). Isolation of Newcastle disease virus from feces of the domestic cat and the common chicken louse. In *Abstracts of papers presented at the 48th general meeting of the Society of American Bacteriologists*, Minneapolis, Society of American Bacteriologists, Washington, D.C., p. 43.
15. Booth AJ, Stogdale L, Grigor JA (1984). Salmon poisoning disease in dogs on southern Vancouver Island. *Canadian Veterinary Journal-Revue Veterinaire Canadienne* 25:2–6.
16. Borucki MK, Kempf BJ, Blitvich BJ, Blair CD, Beaty BJ (2002). La Crosse virus: replication in vertebrate and invertebrate hosts. *Microbes and Infection* 4:341–350.
17. Braverman Y, Chizov-Ginzburg A (1996). Role of dogs (*Canis domesticus*) as hosts for African horse sickness virus. *Veterinary Microbiology* 51:19–25.
18. Breitschwerdt EB (2008). Feline bartonellosis and cat scratch disease. *Veterinary Immunology and Immunopathology* 123:167–171.
19. Brown C (2008). Japanese encephalitis. In *Foreign animal diseases*, Committee on Foreign and Emerging Diseases of the United States Animal Health Association (ed.), United States Animal Health Association, St. Joseph, pp. 311–315.
20. Brown GK, Martin AR, Roberts TK, Aitken RJ (2001). Detection of *Ehrlichia platys* in dogs in Australia. *Australian Veterinary Journal* 79:554–558.
21. Brown HM, Lockhart JM, Latimer KS, Peterson DS (2010). Identification and genetic characterization of *Cytauxzoon felis* in asymptomatic domestic cats and bobcats. *Veterinary Parasitology* 172:311–316.
22. Buick W (2006). TB in domestic species other than cattle and badgers. *Government Veterinary Journal* 16:87–91.
23. Carrade DD, Foley JE, Borjesson DL, Sykes JE (2009). Canine granulocytic anaplasmosis: a review. *Journal of Veterinary Internal Medicine* 23:1129–1141.
24. CDC (Centers for Disease Control and Prevention) (2008). Questions and answers about monkeypox. CDC, Washington DC.  
<http://www.cdc.gov/ncidod/monkeypox/qa.htm> (accessed 15 June 2010)

25. CFSPH (Center for Food Security and Public Health) (2004) Typhus fever—*Rickettsia prowazekii*. CFSPH, Iowa State University.  
[www.cfsph.iastate.edu/Factsheets/pdfs/typhus\\_fever.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/typhus_fever.pdf) (accessed 21 July 2011)
26. CFSPH (Center for Food Security and Public Health) (2005) Louping ill. CFSPH, Iowa State University.  
<http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm> (accessed 17 February 2009)
27. CFSPH (Center for Food Security and Public Health) (2006). Wesselsbron disease. CFSPH, Iowa State University.  
<http://www.cfsph.iastate.edu/Factsheets/pdfs/wesselsbron.pdf> (accessed 6 October 2010)
28. CFSPH (Center for Food Security and Public Health) (2007). Feline spongiform encephalopathy. CFSPH, Iowa State University.  
[http://www.cfsph.iastate.edu/Factsheets/pdfs/feline\\_spongiform\\_encephalopathy.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/feline_spongiform_encephalopathy.pdf) (accessed 20 June 2011)
29. CFSPH (Center for Food Security and Public Health) (2007). Heartwater. CFSPH, Iowa State University.  
<http://www.cfsph.iastate.edu/Factsheets/pdfs/heartwater.pdf> (accessed 21 September 2010)
30. CFSPH (Center for Food Security and Public Health) (2008). Hantavirus. CFSPH, Iowa State University.  
[www.cfsph.iastate.edu/Factsheets/pdfs/hantavirus.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/hantavirus.pdf) (accessed 20 June 2011)
31. CFSPH (Center for Food Security and Public Health) (2010). Coccidioidomycosis. CFSPH, Iowa State University.  
[www.cfsph.iastate.edu/Factsheets/pdfs/coccidioidomycosis.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/coccidioidomycosis.pdf) (accessed 28 June 2011)
32. Coffey LL, Crawford C, Dee J, Miller R, Freier J, Weaver SC (2006). Serologic evidence of widespread everglades virus activity in dogs, Florida. *Emerging Infectious Diseases* 12:1873–1879.
33. Cohen SB, Yabsley MJ, Freye JD, Dunlap BG, Rowland ME, Huang J, Dunn JR, Jones TF, Moncayo AC (2010). Prevalence of *Ehrlichia chaffeensis* and *Ehrlichia ewingii* in ticks from Tennessee. *Vector-Borne and Zoonotic Diseases* 10:435–440.
34. Dawson JE, Abeygunawardena I, Holland CJ, Buese MM, Ristic M (1988). Susceptibility of cats to infection with *Ehrlichia risticii*, causative agent of equine monocytic ehrlichiosis. *American Journal of Veterinary Research* 49:2096–2100.

35. Dean R, Gunn-Moore D, Shaw S, Harvey A (2006). Bovine tuberculosis in cats. *Veterinary Record* 158:419–420.
36. Despommier DD, Gwadz RW, Hotez PJ, Knirsch CA (2005). *Trichinella spiralis* (Raillet 1896). In *Parasitic diseases*, 5th edn, Apple Trees Productions, LCC, New York, pp. 135–142.
37. Diesing L, Heydorn AO, Matuschka FR, Bauer C, Pipano E, Waal DT, Potgieter FT (1988). *Besnoitia besnoiti*: studies on the definitive host and experimental infections in cattle. *Parasitology Research* 75:114–117.
38. Dobler G (2010). Zoonotic tick-borne flaviviruses. *Veterinary Microbiology* 140:221–228.
39. Dubey JP, Chapman JL, Rosenthal BM, Mense M, Schueler RL (2006). Clinical *Sarcocystis neurona*, *Sarcocystis canis*, *Toxoplasma gondii*, and *Neospora caninum* infections in dogs. *Veterinary Parasitology* 137:36–49.
40. Dubey JP, Lindsay DS, Rosenthal BM, Sreekumar C, Hill DE, Shen SK, Kwok OCH, Rickard LG, Black SS, Rashmir-Raven A (2002). Establishment of *Besnoitia darlingi* from opossums (*Didelphis virginiana*) in experimental intermediate and definitive hosts, propagation in cell culture, and description of ultrastructural and genetic characteristics. *International Journal for Parasitology* 32:1053–1064.
41. Dubey JP, Saville WJA, Lindsay DS, Stich RW, Stanek JF, Speer CA, Rosenthal BM, Njoku CJ, Kwok OCH, Shen SK, Reed SM (2000). Completion of the life cycle of *Sarcocystis neurona*. *Journal of Parasitology* 86:1276–1280.
42. Dubey JP, Sreekumar C, Lindsay DS, HILL D, Rosenthal BM, Venturini L, Venturini MC, Greiner EC (2003). *Besnoitia oryctofelisi* n. sp. (Protozoa: Apicomplexa) from domestic rabbits. *Parasitology* 126:521–539.
43. Dubey JP, Yabsley MJ (2010). *Besnoitia neotomofelis* n. sp. (Protozoa: Apicomplexa) from the southern plains woodrat (*Neotoma micropus*). *Parasitology* 137:1731–1747.
44. Dvorak GD, Spickler AR (2008). Glanders. *Journal of the American Veterinary Medical Association* 233:570–577.
45. Ebani V, Cerri D, Fratini F, Ampola M, Andreani E (2008). Seroprevalence of *Anaplasma phagocytophilum* in domestic and wild animals from central Italy. *New Microbiologica* 31:371–375.
46. Farrar MD, Miller DL, Baldwin CA, Stiver SL, Hall CL (2005). Eastern equine encephalitis in dogs. *Journal of Veterinary Diagnostic Investigation* 17:614–617.
47. Gaidici A, Saubolle MA (2009). Transmission of coccidioidomycosis to a human via a cat bite. *Journal of Clinical Microbiology* 47:505–506.

48. Glover GJ, Swendrowski M, Cawthorn RJ (1990). An epizootic of besnoitiosis in captive caribou (*Rangifer tarandus caribou*), reindeer (*Rangifer tarandus tarandus*) and mule deer (*Odocoileus hemionus hemionus*). *Journal of Wildlife Diseases* 26:186–195.
49. Godsey MS, Amoo F, Yuill TM, Defoliart GR (1988). California serogroup virus infections in Wisconsin domestic animals. *The American Journal of Tropical Medicine and Hygiene* 39:409–416.
50. Graupmann-Kuzma A, Valentine BA, Shubitz LS, Dial SM, Watrous B, Tornquist SJ (2008). Coccidioidomycosis in dogs and cats: a review. *Journal of the American Animal Hospital Association* 44:226–235.
51. Greene CE, Baldwin CA (2006). Arthropod-borne viral infections: mosquito and gnat-borne infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 188–192.
52. Greene CE, Berg A-L (2006). Miscellaneous viral infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 162–167.
53. Greene CE, Breitschwerdt EB (2006). Rocky Mountain spotted fever, murine typhus-like disease, rickettsialpox, typhus and Q fever. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders, Elsevier, St. Louis, pp. 232–245.
54. Greene CE, Gunn-Moore DA (2006). Mycobacterial infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 462–488.
55. Greene CE, Rupprecht CE (2006). Rabies and other lyssavirus infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 167–183.
56. Gunn-Moore D, Dean R, Shaw S (2011). Mycobacterial infections in cats and dogs. *In Practice* 32:444–452.
57. Headley SA, Scorpio DG, Vidotto O, Dumler JS (2011). Neorickettsia helminthoeca and salmon poisoning disease: a review. *The Veterinary Journal* 187:165–173.
58. Hsu V, Grant DC, Dubey JP, Zajac AM, Lindsay DS (2010). Prevalence of antibodies to *Sarcocystis neurona* in cats from Virginia and Pennsylvania. *Journal of Parasitology* 96:800–801.
59. Johnson DKH, Staples JE, Sotir MJ, Warshauer DM, Davis JP (2010). Tick-borne Powassan virus infections among Wisconsin residents. *Wisconsin Medical Journal* 109:91–97.

60. Jones SL, Athan E, O'Brien D, Graves SR, Nguyen C, Stenos J (2004). Murine typhus: the first reported case from Victoria. *Medical Journal of Australia* 180:482.
61. Kamhieh S, Hodgson JI, Bode L, Ludwig H, Flower RLP (2008). Borna disease virus: evidence of naturally-occurring infection in cats in Australia. *Acta Pathologica, Microbiologica et Immunologica Scandinavica* 116:50–52.
62. Kapel CMO (2000). Host diversity and biological characteristics of the *Trichinella* genotypes and their effect on transmission. *Veterinary Parasitology* 93:263–278.
63. Keane DP, Parent J, Little PB (1987). California serogroup and Powassan virus infection of cats. *Canadian Journal of Microbiology* 33:693–697.
64. Kohn GJ, Linne SR, Smith CM, Hoepflich PD (1992). Acquisition of coccidioidomycosis at necropsy by inhalation of coccidioidal endospores. *Diagnostic Microbiology and Infectious Disease* 15:527–530.
65. Lancaster JE (1963). Newcastle disease: modes of spread part II. *The Veterinary Bulletin* 33:279–285.
66. Landiado-Laborin RAFA (2007). Expanding understanding of epidemiology of coccidioidomycosis in the Western Hemisphere. *Annals of the New York Academy of Sciences* 1111:19–34.
67. Legendre AM (2006). Blastomycosis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 569–584.
68. Levin ML, Killmaster LF, Zemtsova GE (2012). Domestic dogs (*Canis familiaris*) as reservoir hosts for *Rickettsia conorii*. *Vector-Borne and Zoonotic Diseases* 12:28–33.
69. Lumb R, Bastian I, Crighton T, Gilpin C, Haverkort F, Sievers A (2004). Tuberculosis in Australia: bacteriologically confirmed cases and drug resistance, 2004. A report of the Australian Mycobacterium Reference Laboratory Network. *Communicable Diseases Intelligence* 30:102–108.
70. Mansfield KL, Johnson N, Phipps LP, Stephenson JR, Fooks AR, Solomon T (2009). Tick-borne encephalitis virus—a review of an emerging zoonosis. *Journal of General Virology* 90:1781–1794.
71. Mason RW (1980). The discovery of *Besnoitia wallacei* in Australia and the identification of a free-living intermediate host. *Parasitology Research* 61:173–178.
72. Mellor PS, Hamblin C (2004). African horse sickness. *Veterinary Research* 35:445–466.

73. Monies B, de la Rua R, Jahans K (2006). Bovine tuberculosis in cats. *Veterinary Record* 158:490–491.
74. Monies B, Jahans K, de la Rua R (2006). Bovine tuberculosis in cats. *Veterinary Record* 158:245–246.
75. Moore GE, Greene CE (2006). Anthrax. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 312–315.
76. Neer TM, Harrus S (2006). Ehrlichiosis, neorickettsiosis, anaplasmosis and Wolbachia infection. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 203–216.
77. Nel LH, Markotter W (2007). Lyssaviruses. *Critical Reviews in Microbiology* 33:301–324.
78. Ng'ang'a CJ, Kasigazi S (1994). *Caprine besnoitiosis: studies on the experimental intermediate hosts and the role of the domestic cat in transmission*. *Veterinary Parasitology* 52:207–210.
79. Njaa BL (2008). Emerging viral encephalitides in dogs and cats. *The Veterinary Clinics of North America Small Animal Practice* 38:863–878.
80. O'Brien CR, Greene CE, Greene RT (2006). Miscellaneous bacterial infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 436–438.
81. OIE (World Organisation for Animal Health) (2011). Anthrax. *Terrestrial animal health code 2011*. OIE, Paris.  
[http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre\\_1.8.1.htm](http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.8.1.htm)  
(accessed 28 November 2011)
82. OIE (World Organisation for Animal Health) (2011). Aujeszky's disease. *Terrestrial animal health code 2011*. OIE, Paris.  
[http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre\\_1.8.2.htm](http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.8.2.htm)  
(accessed 29 November 2011)
83. OIE (World Organisation for Animal Health) (2011). *Manual of diagnostic tests and vaccines for terrestrial animals 2011*. OIE, Paris.  
[http://www.oie.int/eng/normes/mmanual/A\\_summry.htm](http://www.oie.int/eng/normes/mmanual/A_summry.htm) (Accessed 7 July 2011)
84. OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/>  
(accessed 16 January 2012)
85. Oura CAL, el Harrak M (2011). Midge-transmitted bluetongue in domestic dogs. *Epidemiology and Infection* 139:1396–1400.

86. Pensaert MB, Kluge JP (1989). Pseudorabies virus (Aujeszky's disease). In *Virus infections of porcines* Pensaert MB (ed.), Elsevier Science, Amsterdam, pp. 39–64.
87. Perry BD, Schmidtman ET, Rice RM, Hansen JW, Fletcher M, Turner EC, Robl MG, Hahn NE (1989). Epidemiology of Potomac horse fever: an investigation into the possible role of non-equine mammals. *The Veterinary Record* 125:83–86.
88. Pfeffer M, Dobler G (2011). Tick-borne encephalitis virus in dogs—is this an issue? *Parasites & Vectors* 4:59–67.
89. Pusterla N, Berger Pusterla J, DeRock E, Madigan JE (2001). Susceptibility of cattle to *Ehrlichia risticii*, the causative agent of Potomac horse fever. *The Veterinary Record* 148:86–87.
90. Reichard MV, Meinkoth JH, Edwards AC, Snider TA, Kocan KM, Blouin EF, Little SE (2009). Transmission of *Cytauxzoon felis* to a domestic cat by *Amblyomma americanum*. *Veterinary Parasitology* 161:110–115.
91. Reid HW (2006). Louping ill. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 196–197.
92. Reisen WK (2003). Epidemiology of St. Louis encephalitis virus. *Advances in Virus Research* 61:139–183.
93. Ristic M, Dawson J, Holland CJ, Jenny A (1988). Susceptibility of dogs to infection with *Ehrlichia risticii*, causative agent of equine monocytic ehrlichiosis (Potomac horse fever). *American Journal of Veterinary Research* 49:1497–1500.
94. Rovey C, Brouqui P, Raoult D (2008). Questions on Mediterranean spotted fever a century after its discovery. *Emerging Infectious Diseases* 14:1360–1367.
95. Sabeta CT, Markotter W, Mohale DK, Shumba W, Wandeler AI, Nel LH (2007). Mokola virus in domestic mammals, South Africa. *Emerging Infectious Diseases* 13:1371–1373.
96. Schloderer D, Owen H, Clark P, Stenos J, Fenwick SG (2006). *Rickettsia felis* in fleas, Western Australia. *Emerging Infectious Diseases* 12:841–843.
97. Shubitz LS (2007). Comparative aspects of coccidioidomycosis in animals and humans. *Annals of the New York Academy of Sciences* 1111:395–403.
98. Smith DD, Frenkel JK (1977). *Besnoitia darlingi* (protozoa: Toxoplasmatinae): cyclic transmission by cats. *Journal of Parasitology* 63:1066–1071.

99. Stanek JF, Stich RW, Dubey JP, Reed SM, Njoku CJ, Lindsay DS, Schmall LM, Johnson GK, LaFave BM, Saville WJA (2003). Epidemiology of *Sarcocystis neurona* infections in domestic cats (*Felis domesticus*) and its association with equine protozoal myeloencephalitis (EPM) case farms and feral cats from a mobile spay and neuter clinic. *Veterinary Parasitology* 117:239–249.
100. Studdert MJ (1996). Louping ill. In *Virus infections of equines*, 6 edn, Studdert MJ (ed.), Elsevier Science Publishers, Amsterdam, pp. 167–168.
101. TAGS Inc. (2009). Ticks of Australia. Tick Alert: Group Support. <http://web.archive.org/web/20090209112446/http://tickalert.org.au/rhripsang.htm> (accessed 7 May 2012)
102. Tatum LM, Pacy JM, Frazier KS (1999). Canine LaCrosse viral meningoencephalomyelitis with possible public health implications. *Journal of Veterinary Diagnostic Investigation* 11:184–188.
103. Thoen CO, LoBue PA, Enarson DA, Kaneene JB, de Kantor IN (2009). Tuberculosis: a re-emerging disease in animals and humans. *Veterinaria Italiana* 45:135–181.
104. van Oirschot JT (2004). Aujeszky's disease. In *Infectious diseases of livestock*, 2nd edn, Coetzer JAW, Tustin RC (eds), Oxford University Press, Oxford, pp. 909–915.
105. Vandeveld M, Greene CE (2006). Neurological diseases of suspected infectious origin and prion disease: prion diseases and feline spongy encephalopathy. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 803–806.
106. Yabsley MJ, Varela AS, Tate CM, Dugan VG, Stallknecht DE, Little SE, Davidson WR (2002). *Ehrlichia ewingii* infection in white-tailed deer (*Odocoileus virginianus*). *Emerging Infectious Diseases* 8:668–671.
107. Zacks MA, Paessler S (2010). Encephalitic alphaviruses. *Veterinary Microbiology* 140(3–4):281–286.

## 4 Risk reviews

---

### 4.1 Canine bartonellosis

#### 4.1.1 Background

Canine bartonellosis, caused by the bacteria *Bartonella vinsonii* subsp. *berkhoffii* (*B. berkhoffii*), has been identified as an important emerging zoonotic disease (Chomel et al. 2009c). Infection with this disease agent has been reported in domestic dogs, coyotes (*Canis latrans*), grey foxes (*Urocyon cinereoargenteus*) and humans (Cockwill et al. 2007). The disease agent has also been isolated in a domestic cat (Varanat et al. 2009).

*B. berkhoffii* was first isolated in 1993 from the blood of a dog with intermittent epistaxis and endocarditis (Breitschwerdt et al. 1995), and is now considered the most frequently identified *Bartonella* spp. causing disease in dogs (Breitschwerdt and Chomel 2006).

A number of other *Bartonella* spp. have been associated with infection or disease in dogs, including *B. clarridgeiae*, *B. elizabethae*, *B. henselae*, *B. koehlerae*, *B. quintana*, *B. rochalimae* and *B. washoensis* (Breitschwerdt et al. 2010a; Chomel et al. 2006; Fenimore et al. 2011; Guptill 2010). However, dogs are either not considered a reservoir for these bacteria (Chomel et al. 2003; Chomel et al. 2006; Jacomo et al. 2002) or their status as natural reservoirs has not been confirmed (Chomel et al. 2009b; Henn et al. 2009).

*B. berkhoffii* infection in domestic dogs has been largely associated with endocarditis; however, the clinical spectrum of signs attributed to infection with this disease agent is expanding (Chomel et al. 2006). The extent to which dogs serve as reservoirs for *B. berkhoffii*, and whether arthropod vectors are involved in transmission, is not well documented (Billeter et al. 2008; Breitschwerdt et al. 2010c; Breitschwerdt et al. 2010a; Breitschwerdt and Kordick 2000; Pappalardo et al. 1997).

Evidence of exposure to *B. berkhoffii* can reportedly be found in most tropical and subtropical regions of the world (Breitschwerdt et al. 2010a). Four genotypes of the subspecies have been identified: genotypes I, II and III identified in the United States; genotype III in Europe and genotype IV in Canada (Maggi et al. 2006).

There are no known reports of *B. berkhoffii* being isolated in Australia.

Canine bartonellosis is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (the Department of Agriculture 2011).

#### 4.1.2 Technical information

##### Epidemiology

Infection of mammalian reservoir hosts with *Bartonella* spp. causes a prolonged intra-erythrocytic bacteraemia (Chomel et al. 2009a; Guptill 2010).

Coyotes in the United States may be important hosts for *B. berkhoffii* and as a wildlife reservoir could serve as a source of infection for domestic dogs and people (Billeter et al. 2008). A survey of coyotes in a county in California demonstrated 28% were bacteraemic and 76% had antibodies to *B. berkhoffii* (Chang et al. 2000). Another study in California found an overall seroprevalence of 28% in coyotes with some seasonal fluctuation (Beldomenico et al. 2005). This contrasts with seroprevalence estimates in domestic dogs in areas of the United States that range between 0.7% and 3.6% (Henn et al. 2005; Hinrichsen et al. 2001; Pappalardo et al. 1997).

Seroprevalence reported in dogs in other geographic regions include 1.5% in Brazil (Diniz et al. 2007), 6.6% in Turkey (Celebi et al. 2010), 8.2% in Grenada (Yabsley et al. 2008), 10% in Israel (Baneth et al. 1998) and 38% in Thailand (Suksawat et al. 2001) and Morocco (Henn et al. 2006). Dogs living in a rural environment, outdoor dogs and dogs with heavy tick burdens are more likely to be exposed to *B. berkhoffii* (Chomel et al. 2004; Honadel et al. 2001; Pappalardo et al. 1997; Solano-Gallego et al. 2004).

Co-infection with vector-borne disease agents has been reported (Guptill 2010). Correlations have been reported between positive serology to *B. berkhoffii* and tick-borne disease agents such as *Ehrlichia canis*, *Babesia canis*, *Rickettsia rickettsii* and *Anaplasma phagocytophilum* (Breitschwerdt et al. 1998; Breitschwerdt et al. 2010a; MacDonald et al. 2004; Pappalardo et al. 1997; Suksawat et al. 2001).

Proposed tick vectors have included *Rhipicephalus sanguineus*, *Amblyomma americanum*, *Ixodes scapularis*, *I. pacificus* and *Dermacentor* spp. (e.g. *Dermacentor variabilis*) (Chomel et al. 2004; Pappalardo et al. 1997). Partial sequences of this disease agent have been detected in ticks (Chang et al. 2001), but there are no known reports documenting transmission of *B. berkhoffii* via ticks to vertebrate hosts. Further evidence is required to confirm vector transmission (Angelakis et al. 2010; Telford and Wormser 2010).

Proposed modes of transmission to humans include transmission via needle-stick injury, dog bites and perinatal transmission (Breitschwerdt et al. 2010b; Chang et al. 1999; Oliveira et al. 2010; Rolain et al. 2009). Further investigation is required to evaluate the risk of transmission through these routes.

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

## Clinical signs

Infection of dogs with *B. berkhoffii* has been associated with cardiac arrhythmias, endocarditis, myocarditis, granulomatous lymphadenitis and granulomatous rhinitis. The disease agent has also been implicated in cases of polyarthritis, neutrophilic or granulomatous meningoencephalitis, anterior uveitis and chorioretinitis in dogs (Breitschwerdt and Maggi 2009; Chomel et al. 2004). The factors that produce the clinical signs caused by *B. berkhoffii* have not been established (Breitschwerdt and Chomel 2006).

*B. berkhoffii* has also been isolated from clinically healthy dogs (Chomel et al. 2004). In one study, the disease agent was isolated from a clinically healthy dog on eight occasions from ten culture attempts over 16 months (Kordick and Breitschwerdt 1998). Experimentally infected dogs have been reported to have transient pyrexia, but otherwise remain clinically healthy (Pappalardo et al. 2000; Pappalardo et al. 2001).

Concurrent infection with agents such as *E. canis*, *B. canis* and *R. rickettsii*, may complicate the interpretation of clinical signs observed in natural cases of canine bartonellosis (Breitschwerdt et al. 2004).

Although infection with *B. berkhoffii* may result in cardiac dysfunction, infection should also be suspected in dogs with prolonged or intermittent pyrexia, lethargy, unexplained lameness or granulomatous disease; or dogs that may have been exposed to ticks (Chomel et al. 2004).

## Diagnosis

Serological testing, using an indirect fluorescent antibody test (IFAT) is considered the most effective means of screening for exposure to *B. berkhoffii* because bacterial culture is an insensitive diagnostic method (Breitschwerdt et al. 2004; Guptill 2010).

Information about the sensitivity and specificity of tests in dogs is limited (Chomel et al. 2006; Diniz et al. 2007; Duncan et al. 2008; Guptill 2010).

Polymerase chain reaction (PCR) may provide a sensitive means of detecting *Bartonella* DNA (Guptill 2010). Because serological results do not always correlate with PCR results a combined approach, using pre-enrichment culture and PCR, has been recommended to confirm a diagnosis of canine bartonellosis (Duncan et al. 2007).

## Treatment

Antibiotic treatment has not proven to be effective in eliminating infection in dogs. Treatment with doxycycline, azithromycin, enrofloxacin and other antibiotics has been reported, but an optimal treatment regime for dogs has not been established (Guptill 2010).

### 4.1.3 Current biosecurity measures

There are no specific biosecurity measures for canine bartonellosis.

#### 4.1.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by canine bartonellosis:

- Canine bartonellosis is not an OIE-listed disease and is not a nationally notifiable disease in Australia.
- *B. berkhoffii* has a wide geographic distribution, and has been reported in dog populations in Europe, North and South America, Asia and Africa.
- There is a history of importation of dogs from infected countries, with no known reported cases in Australia.
- The status of dogs as reservoirs for *B. berkhoffii* is yet to be definitively established.
- Arthropod vectors such as ticks may be involved in the transmission of *B. berkhoffii*, but their role requires further investigation.
- No evidence for venereal routes of transmission was found in the scientific literature.
- IFAT is currently considered the most effective diagnostic test but may be associated with false negative results.
- A recommended treatment regimen for canine bartonellosis has not been established.

#### 4.1.5 Conclusion

Based on the preceding factors, it was concluded that risk management measures for canine bartonellosis are not warranted for dogs, cats or their semen.

#### References

- Angelakis E, Billeter SA, Breitschwerdt EB, Chomel BB, Raoult D (2010). Potential for tick-borne bartonellosis. *Emerging Infectious Diseases* 16:385–391.
- Baneth G, Breitschwerdt EB, Hegarty BC, Pappalardo B, Ryan J (1998). A survey of tick-borne bacteria and protozoa in naturally exposed dogs from Israel. *Veterinary Parasitology* 74:133–142.
- Beldomenico PM, Chomel BB, Foley JE, Sacks BN, Baldi CJ, Kasten RW, Gardner IA (2005). Environmental factors associated with *Bartonella vinsonii* subsp *berkhoffii* seropositivity in free-ranging coyotes from northern California. *Vector-Borne and Zoonotic Diseases* 5:110–119.
- Billeter SA, Levy MG, Chomel BB, Breitschwerdt EB (2008). Vector transmission of *Bartonella* species with emphasis on the potential for tick transmission. *Medical and Veterinary Entomology* 22:1–15.

- Breitschwerdt EB, Blann KR, Stebbins ME, Munana KR, Davidson MG, Jackson HA, Willard MD (2004). Clinicopathological abnormalities and treatment response in 24 dogs seroreactive to *Bartonella vinsonii* (*berkhoffii*) antigens. *Journal of the American Animal Hospital Association* 40:92–101.
- Breitschwerdt EB, Chomel BB (2006). Canine bartonellosis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 518–524.
- Breitschwerdt EB, Hegarty BC, Hancock SI (1998). Sequential evaluation of dogs naturally infected with *Ehrlichia canis*, *Ehrlichia chaffeensis*, *Ehrlichia equi*, *Ehrlichia ewingii*, or *Bartonella vinsonii*. *Journal of Clinical Microbiology* 36:2645–2651.
- Breitschwerdt EB, Kordick DL (2000). *Bartonella* infection in animals: carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. *Clinical Microbiology Reviews* 13:428–438.
- Breitschwerdt EB, Kordick DL, Malarkey DE, Keene B, Hadfield TL, Wilson K (1995). Endocarditis in a dog due to infection with a novel *Bartonella* subspecies. *Journal of Clinical Microbiology* 33:154–160.
- Breitschwerdt EB, Maggi RG (2009). Comparative medical features of canine and human bartonellosis. *Clinical Microbiology and Infection* 15:106–107.
- Breitschwerdt EB, Maggi RG, Chomel BB, Lappin MR (2010a). Bartonellosis: an emerging infectious disease of zoonotic importance to animals and human beings. *Journal of Veterinary Emergency and Critical Care* 20:8–30.
- Breitschwerdt EB, Maggi RG, Farmer P, Mascarelli PE (2010b). Molecular evidence of perinatal transmission of *Bartonella vinsonii* subsp. *berkhoffii* and *Bartonella henselae* to a child. *Journal of Clinical Microbiology* 48:2289–2293.
- Breitschwerdt EB, Maggi RG, Lantos PM, Woods CW, Hegarty BC, Bradley JM (2010c). *Bartonella vinsonii* subsp. *berkhoffii* and *Bartonella henselae* bacteremia in a father and daughter with neurological disease. *Parasites & Vectors* 3:1–9.
- Celebi B, Ozkan AT, Kilic S, Akca A, Koenhems L, Pasa S, Yildiz K, Mamak N, Guzel M (2010). Seroprevalence of *Bartonella vinsonii* subsp. *berkhoffii* in urban and rural dogs in Turkey. *Journal of Veterinary Medical Science* 72:1491–1494.
- Chang C-C, Kasten RW, Chomel BB, Simpson DC, Hew CM, Kordick DL, Heller R, Piemont Y, Breitschwerdt EB (2000). Coyotes (*Canis latrans*) as the reservoir for a human pathogenic *Bartonella* sp.: molecular epidemiology of *Bartonella vinsonii* subsp. *berkhoffii* infection in coyotes from central coastal California. *Journal of Clinical Microbiology* 38:4193–4200.
- Chang CC, Chomel BB, Kasten RW, Romano V, Tietze N (2001). Molecular evidence of *Bartonella* spp. in questing adult *Ixodes pacificus* ticks in California. *Journal of Clinical Microbiology* 39:1221–1226.

- Chang CC, Yamamoto K, Chomel BB, Kasten RW, Simpson DC, Smith CR, Kramer VL (1999). Seroepidemiology of *Bartonella vinsonii* subsp. *berkhoffii* infection in California coyotes, 1994–1998. *Emerging Infectious Diseases* 5:711–715.
- Chomel BB, Boulouis H-J, Breitschwerdt EB, Kasten RW, Vayssier-Taussat M, Birtles RJ, Koehler JE, Dehio C (2009a). Ecological fitness and strategies of adaptation of *Bartonella* species to their hosts and vectors. *Veterinary Research* 40:1–22.
- Chomel BB, Boulouis HJ, Breitschwerdt EB (2004). Cat scratch disease and other zoonotic *Bartonella* infections. *Javma-Journal of the American Veterinary Medical Association* 224:1270–1279.
- Chomel BB, Boulouis HJ, Maruyama S, Breitschwerdt EB (2006). *Bartonella* spp. in pets and effect on human health. *Emerging Infectious Diseases* 12:389–394.
- Chomel BB, Henn JB, Kasten RW, Nieto NC, Foley J, Papageorgiou S, Allen C, Koehler JE (2009b). Dogs are more permissive than cats or guinea pigs to experimental infection with a human isolate of *Bartonella rochalimae*. *Veterinary Research* 40:1–8.
- Chomel BB, Kasten RW, Williams C, Wey AC, Henn JB, Maggi R, Carrasco S, Mazet J, Boulouis HJ, Maillard R, Breitschwerdt EB (2009c). *Bartonella endocarditis*. *Annals of the New York Academy of Sciences* 1166:120–126.
- Chomel BB, Wey AC, Kasten RW (2003). Isolation of *Bartonella washoensis* from a dog with mitral valve endocarditis. *Journal of Clinical Microbiology* 41:5327–5332.
- Cockwill KR, Taylor SM, Philibert HM, Breitschwerdt EB, Maggi RG (2007). *Bartonella vinsonii* subsp. *berkhoffii* endocarditis in a dog from Saskatchewan. *Canadian Veterinary Journal-Revue Veterinaire Canadienne* 48:839–844.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Diniz PPVP, Maggi RG, Schwartz DS, Cadenas MB, Bradley JM, Hegarty B, Breitschwerdt EB (2007). Canine bartonellosis: serological and molecular prevalence in Brazil and evidence of co-infection with *Bartonella henselae* and *Bartonella vinsonii* subsp. *berkhoffii*. *Veterinary Research* 38:697–710.
- Duncan AW, Maggi RG, Breitschwerdt EB (2007). A combined approach for the enhanced detection and isolation of *Bartonella* species in dog blood samples: pre-enrichment liquid culture followed by PCR and subculture onto agar plates. *Journal of Microbiological Methods* 69:273–281.
- Duncan AW, Marr HS, Birkenheuer AJ, Maggi RG, Williams LE, Correa MT, Breitschwerdt EB (2008). *Bartonella* DNA in the blood and lymph nodes of golden

retrievers with lymphoma and in healthy controls. *Journal of Veterinary Internal Medicine* 22:89–95.

Fenimore A, Varanat M, Maggi R, Schultheiss P, Breitschwerdt E, Lappin M (2011). *Bartonella* spp. DNA in cardiac tissues from dogs in Colorado and Wyoming. *Journal of Veterinary Internal Medicine* 25:613–616.

Guptill L (2010). Bartonellosis. *Veterinary Microbiology* 140:347–359.

Henn JB, Gabriel MW, Kasten RW, Brown RN, Koehler JE, MacDonald KA, Kittleson MD, Thomas W, Chomel BB (2009). Infective endocarditis in a dog and the phylogenetic relationship of the associated *Bartonella rochalimae* strain with isolates from dogs, gray foxes, and a human. *Journal of Clinical Microbiology* 47:787–790.

Henn JB, Liu C-H, Kasten RW, VanHorn BA, Beckett LA, Kass PH, Chomel BB (2005). Seroprevalence of antibodies against *Bartonella* species and evaluation of risk factors and clinical signs associated with seropositivity in dogs. *American Journal of Veterinary Research* 66:688–694.

Henn JB, VanHorn BA, Kasten RW, Kachani M, Chomel BB (2006) Antibodies to *Bartonella vinsonii* subsp. *berkhoffii* in Moroccan dogs. *The American Journal of Tropical Medicine and Hygiene* 74: 222-223.

Hinrichsen VL, Whitworth UG, Breitschwerdt EB, Hegarty BC, Mather TN (2001). Assessing the association between the geographic distribution of deer ticks and seropositivity rates to various tick-transmitted disease organisms in dogs. *Journal of the American Veterinary Medical Association* 218:1092–1097.

Honadel TE, Chomel BB, Yamamoto K, Chang CC, Farver TB (2001). Seroepidemiology of *Bartonella vinsonii* subsp. *berkhoffii* exposure among healthy dogs. *Journal of the American Veterinary Medical Association* 219:480–484.

Jacomo V, Kelly PJ, Raoult D (2002). Natural history of *Bartonella* infections (an exception to Koch's postulate). *Clinical and Diagnostic Laboratory Immunology* 9:8–18.

Kordick DL, Breitschwerdt EB (1998). Persistent infection of pets within a household with three *Bartonella* species. *Emerging Infectious Diseases* 4:325–328.

MacDonald KA, Chomel BB, Kittleson MD, Kasten RW, Thomas WP, Pesavento P (2004). A prospective study of canine infective endocarditis in northern California (1999–2001): emergence of *Bartonella* as a prevalent etiologic agent. *Journal of Veterinary Internal Medicine* 18:56–64.

Maggi RG, Chomel B, Hegarty BC, Henn J, Breitschwerdt EB (2006). A *Bartonella vinsonii berkhoffii* typing scheme based upon 16S-23S ITS and Pap31 sequences from dog, coyote, gray fox, and human isolates. *Molecular and Cellular Probes* 20:128–134.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012> (accessed 16 January 2012)

Oliveira A, Maggi R, Woods C, Breitschwerdt E (2010). Suspected needle stick transmission of *Bartonella vinsonii* subspecies *berkhoffii* to a veterinarian. *Journal of Veterinary Internal Medicine* 24:1229–1232.

Pappalardo BL, Brown T, Gebhardt D, Sontakke S, Breitschwerdt EB (2000). Cyclic CD8+ lymphopenia in dogs experimentally infected with *Bartonella vinsonii* subsp. *berkhoffii*. *Veterinary Immunology and Immunopathology* 75:43–57.

Pappalardo BL, Brown TT, Tompkins M, Breitschwerdt EB (2001). Immunopathology of *Bartonella vinsonii* (*berkhoffii*) in experimentally infected dogs. *Veterinary Immunology and Immunopathology* 83:125–147.

Pappalardo BL, Correa MT, York CC, Peat CY, Breitschwerdt EB (1997). Epidemiologic evaluation of the risk factors associated with exposure and seroreactivity to *Bartonella vinsonii* in dogs. *American Journal of Veterinary Research* 58:467–471.

Rolain JM, Boureau-Voultoury A, Raoult D (2009). Serological evidence of *Bartonella vinsonii* lymphadenopathies in a child bitten by a dog. *Clinical Microbiology and Infection* 15:122–123.

Solano-Gallego L, Bradley J, Hegarty B, Sigmon B, Breitschwerdt E (2004). *Bartonella henselae* IgG antibodies are prevalent in dogs from southeastern USA. *Veterinary Research* 35:585–595.

Suksawat J, Xuejie Y, Hancock SI, Hegarty BC, Nilkumhang P, Breitschwerdt EB (2001). Serologic and molecular evidence of coinfection with multiple vector-borne pathogens in dogs from Thailand. *Journal of Veterinary Internal Medicine* 15:453–462.

Telford SR, Wormser GP (2010). *Bartonella* spp. transmission by ticks not established. *Emerging Infectious Diseases* 16:379–384.

Varanat M, Travis A, Lee W, Maggi RG, Bissett SA, Linder KE, Breitschwerdt EB (2009). Recurrent osteomyelitis in a cat due to infection with *Bartonella vinsonii* subsp. *berkhoffii* genotype II. *Journal of Veterinary Internal Medicine* 23:1273–1277.

Yabsley MJ, McKibben J, Macpherson CN, Cattan PF, Cherry NA, Hegarty BC, Breitschwerdt EB, O'Connor T, Chandrashekar R, Paterson T, Perea ML, Ball G, Friesen S, Goedde J, Henderson B, Sylvester W (2008). Prevalence of *Ehrlichia canis*, *Anaplasma platys*, *Babesia canis vogeli*, *Hepatozoon canis*, *Bartonella vinsonii berkhoffii*, and *Rickettsia* spp. in dogs from Grenada. *Veterinary Parasitology* 151:279–285.

## 4.2 Canine brucellosis

### 4.2.1 Background

Brucellosis is a contagious disease that produces late abortions in females, and epididymitis and prostatitis in males. It is caused by Gram-negative, aerobic coccobacilli of the *Brucella* genus (Corbel and MacMillan 1998). There are six *Brucella* spp. that produce characteristic infections depending on the host and species: *Brucella abortus* (cattle), *B. canis* (dogs), *B. melitensis* (goats and sheep), *B. neotomae* (rodents), *B. ovis* (sheep) and *B. suis* and its biovars (pigs, cattle, hares, rodents and wild ungulates) (Greene and Carmichael 2006).

In addition to *B. canis*, dogs are also susceptible to infection with *B. abortus*, *B. melitensis* and *B. suis* (CFSPH 2011). Natural infection of dogs with *Brucella* spp. other than *B. canis* is thought to occur after ingestion of contaminated placentas and aborted fetuses from infected livestock. However, dogs are believed to only be important in the spread and maintenance of *B. canis*, as infection of dogs with other *Brucella* spp. appears to be self-limiting (Forbes 1990; Greene and Carmichael 2006). Only *B. canis* is considered further in this review.

*B. canis* has a limited host range predominantly affecting dogs and wild canids. Cats are relatively resistant to infection. Human cases have been reported as a result of laboratory accidents and contact with infected dogs (Greene and Carmichael 2006).

Canine brucellosis is not an OIE-listed disease (OIE 2012), but it is a nationally notifiable disease in Australia (DAFF 2011).

### 4.2.2 Technical information

#### Epidemiology

*B. canis* infection has been diagnosed in many countries including Argentina, Brazil, Canada, Chile, China, Europe, Mexico, the United Kingdom and the United States (Hollett 2006; Wanke 2004). Australia and New Zealand are free from canine brucellosis.

There is a period of approximately three weeks between initial exposure and bacteraemia. The organism typically localises in targeted genital tissues to establish a recurring source of infection (Hollett 2006). In dogs, the urine and seminal fluid of males and vaginal secretions of females are the main sources of infection via the venereal, oral, nasal or conjunctival routes. In male dogs, *B. canis* typically localises in the prostate gland and epididymides. Intermittent shedding of the organism has been reported to persist for at least two years. In female dogs, shedding of *B. canis* may occur for periods up to six weeks after abortion (Greene and Carmichael 2006).

Transmission has also been associated with vaginoscopy, blood transfusion and the use of contaminated syringes (Greene and Carmichael 2006). However, these modes of transmission are of minor epidemiological importance. Desexing mitigates the risk

of the venereal route of transmission—the predominant mode of transmission associated with establishment or spread.

### **Pathogenesis**

*Brucella* spp. are facultative intracellular disease agents that establish infection by invading macrophages and evading macrophage-induced host protection mechanisms (Glynn and Lynn 2008). These characteristics contribute to the clinical signs, and also make both diagnosis and treatment difficult.

After infection, the bacteria are likely phagocytosed at mucosal sites by tissue macrophages and other phagocytic cells, and then transported to lymphatic and genital tract tissues where they multiply. Infection leads to bacteraemia, which is usually transient; the organisms ultimately settle in the reproductive tissues. Localised infection outside reproductive tissues may occur in some dogs with lesions reported in the vertebral column, ocular tissue and skin (Wanke 2004).

### **Clinical signs**

Dogs infected with *B. canis* may have initial signs of general reproductive tract disorders, including abortions during the last third of a pregnancy, stillbirths or conception failures. However, *Brucella*-infected dogs might also have signs of other disorders including ocular, musculoskeletal or dermatologic lesions (Wanke 2004).

### **Diagnosis**

Bacteriological isolation of the disease agent provides the definitive confirmatory diagnostic test for canine brucellosis. However, while *Brucella* spp. are amenable to culture, bacteriological isolation is time consuming and impractical for diagnostic screening.

Serological testing is the most frequently used diagnostic screening technique for brucellosis. For breeding animals, testing should be conducted at least one incubation period after the last insemination or mating to mitigate the risk that the animal may be incubating infection with *B. canis* at the time of blood sample collection.

Different test techniques vary in sensitivity and specificity, which can lead to false positives and negatives, depending upon the stage of the disease, the test antigen and the test method used. The most widely used serological tests are the rapid slide agglutination test (RSAT), 2-mercaptoethanol RSAT (2-ME-RSAT), tube agglutination tests (TAT or 2-ME-TAT) and the agar gel immunodiffusion (AGID) test. Indirect fluorescent antibody tests (IFAT) are also available that appear to have comparable diagnostic performance characteristics to the agglutination tests (VMRD, 1995). Although RSAT, ME-RSAT, ME-TAT and AGID are considered tests of high diagnostic sensitivity (Greene and Carmichael 2006; Wanke 2004), the occurrence of false negative results in some studies indicate that serological tests may not be ideal screening tests for canine brucellosis (Keid et al. 2004, 2007, 2009). PCR testing shows superior sensitivity and specificity when compared to standard serological tests (Keid et al. 2004, 2007, 2009).

Despite the limitations of serological testing, PCR testing is not readily commercially available. Therefore, for diagnosis or screening, a series of tests may be necessary to establish the *B. canis* status of dogs. RSAT is highly sensitive, particularly in the first five weeks following infection and accurately identifies non-infected dogs (Badakhsh et al. 1982). However the RSAT is relatively non-specific and false positive test results are not uncommon. Positive test results may be further clarified by use of 2-ME RSAT or 2-ME-TAT as mercaptoethanol removes non-specific agglutinins and increases test specificity. The mercaptoethanol agglutination tests are best suited to testing from eight to twelve weeks post-infection to around three months after the dog is abacteraemic (Wanke 2004).

AGID tests using either cell wall antigen or cytoplasmic protein antigen (CPAg) may be used for confirmatory testing, but are less sensitive than agglutination tests for initial screening (Greene and Carmichael 2006). AGID tests using cell wall antigens detect brucellosis earlier (five to ten weeks post-infection) than AGID using cytoplasmic antigens (eight to twelve weeks post-infection). Chronic infection, up to three years after a dog is abacteraemic, can be detected using the cytoplasmic antigen agar gel immunodiffusion test (CPAg-AGID), even when other tests have given negative results (Wanke 2004).

#### **Treatment**

Due to the persistent intracellular location of the organism, treatment with antibiotics is often ineffective at eliminating infection and relapses are common. None of the treatment regimes are 100% successful and it is deemed inappropriate to treat dogs using a single antibiotic regime (Hollett 2006).

Antibiotic therapy may suppress bacteraemia and the associated serological response, leading to false negative serological results. Therefore, it is recommended that antibacterial medications not be administered until diagnostic tests have been completed (Hollett 2006).

For breeding dogs, it is recommended that infected dogs be isolated and eliminated from breeding programs. Ideally, infected pet dogs should be desexed and receive a course of antibiotic therapy. While antibiotic therapy may not eliminate infection it has been reported to reduce bacterial shedding (Greene and Carmichael 2006).

#### **4.2.3 Current biosecurity measures**

##### *Dogs*

- Within 30 days before export, dogs must be tested for *Brucella canis* infection using a serum agglutination test (SAT) at a laboratory approved by the Competent Authority of the exporting country. The result must be negative.
- Once blood is collected for *B. canis* testing, dogs must not be mated (including artificial insemination of females) before export to Australia.

#### *Semen donors*

- Within 45 days before collection of semen for export, the donor must be tested for *Brucella canis* infection using a serum agglutination test (SAT) at a laboratory approved by the Competent Authority of the exporting country. The result must be negative.
- Donor dogs must not be naturally mated between blood collection and the last collection of semen for export to Australia.

#### **4.2.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by canine brucellosis:

- Canine brucellosis is a nationally notifiable disease in Australia.
- Canine brucellosis is primarily a sexually transmissible disease of breeding dogs and is also transmissible by artificial insemination with infected semen. It has not been reported in cats.
- Although transmission of *B. canis* has been associated with vaginoscopy, blood transfusion and the use of contaminated syringes, these modes of transmission are of minor epidemiological importance.
- Chronic infections are common and treatment with antibiotics is often ineffective at eliminating infection because of the persistent intracellular location of the organism.
- Desexed animals can harbour infection but desexing eliminates the principal mode of transmission associated with establishment or spread.
- For breeding animals, negative serological testing conducted at least one incubation period after the last mating, insemination or semen collection, mitigates the risk that the dog may be incubating infection at the time of blood sample collection.

#### **4.2.5 Conclusion**

Based on the preceding key points, it was concluded that risk management measures for canine brucellosis continue to be warranted for breeding dogs and dog semen. In addition, it was concluded that risk management measures for canine brucellosis are not warranted for cats or their semen.

The following biosecurity measures would provide appropriate risk management.

### Pre-export measures (dogs)

#### Breeding dogs<sup>2</sup>—serology:

- Within the 45 days immediately before export, a blood sample must be collected from the dog and tested using a rapid slide agglutination test (RSAT), a tube agglutination test (TAT)<sup>3</sup>, or an indirect fluorescent antibody test (IFAT) for *Brucella canis* with a negative result. If test results by RSAT, TAT or IFAT are positive or inconclusive, the dog will be eligible for export if serum is tested within the 21 days immediately before export with a negative result by cytoplasmic antigen agar gel immunodiffusion test (CPAg-AGID).
- If the dog is mated or inseminated within the 30 days immediately before export, blood sample collection must be conducted at least 21 days after the date of last mating or insemination.<sup>4</sup>

NOTE: Any breeding dog diagnosed with *B. canis* infection based on serological test results is not eligible for import, regardless of antibiotic treatment.

OR

#### Desexed dogs—documentation:

- Appropriate documentary evidence that the dog is desexed.

OR

- Serological testing must be conducted as for breeding dogs, with a negative result within the 21 days immediately before export.

### Pre-export measures (dog semen)

#### Donor dogs—serology:

- Between 30 and 45 days following the last collection of semen in the consignment for export, a blood sample must be collected from the donor dog and tested using an RSAT, TAT or IFAT for *Brucella canis* with a negative result.
- If test results by RSAT, TAT or IFAT are positive or inconclusive, the semen will be eligible for export if the donor is tested 30–45 days following the last collection of semen in the consignment for export, by CPAg-AGID, with a negative result.

### References

Badakhsh FF, Carmichael LE, Douglass JA (1982). Improved rapid slide agglutination test for presumptive diagnosis of canine brucellosis. *Journal of Clinical Microbiology* 15:286–289.

CFSPH (Center for Food Security and Public Health) (2011). Canine brucellosis: *Brucella canis*. CFSPH, Ames, Iowa.

<sup>2</sup> Dogs over 16 weeks of age that are not desexed

<sup>3</sup> Both the rapid slide agglutination test and the tube agglutination test are serum agglutination tests. Modified test procedures using mercaptoethanol should be used to minimise false positive results

<sup>4</sup> To be eligible for export to Australia, dogs must not be more than 30 days pregnant

[http://www.cfsph.iastate.edu/Factsheets/pdfs/brucellosis\\_canis.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/brucellosis_canis.pdf) (accessed 3 February 2012)

Corbel MJ, MacMillan AP (1998). Brucellosis. In *Topley and Wilson's microbiology and microbial infections*, 9th edn, Collier L, Balows A, Sussman M (eds), Arnold, London, pp. 819–847.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Forbes LB (1990). *Brucella abortus* infection in 14 farm dogs. *Journal of the American Veterinary Medical Association* 196:911–916.

Glynn MK, Lynn TV (2008). Brucellosis. *Journal of the American Veterinary Medical Association* 233:900–908.

Greene CE, Carmichael LE (2006). Canine brucellosis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 369–381.

Hollett RB (2006). Canine brucellosis: outbreaks and compliance. *Theriogenology* 66:575–587.

Keid LB, Soares RM, Morais ZM, Richtzenhain LJ, Vasconcellos SA (2004). *Brucella* spp. isolation from dogs from commercial breeding kennels in Sao Paulo state, Brazil. *Brazilian Journal of Microbiology* 35:161–166.

Keid LB, Soares RM, Vasconcellos SA, Chiebao DP, Salgado VR, Megid J, Richtzenhain LJ (2007). A polymerase chain reaction for detection of *Brucella canis* in vaginal swabs of naturally infected bitches. *Theriogenology* 68:1260–1270.

Keid LB, Soares RM, Vasconcellos SA, Megid J, Salgado VR, Richtzenhain LJ (2009). Comparison of agar gel immunodiffusion test, rapid slide agglutination test, microbiological culture and PCR for the diagnosis of canine brucellosis. *Research in Veterinary Science* 86:22–26.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012> (accessed 16 January 2012)

Veterinary Medical Research and Development (1995). IFA test for canine brucellosis.  
<http://www.vmr.com/Pages/TechnicalLibraryDetail.aspx?productId=TECH-1195-1&PI=0&RPP=25&Cat=All%20Categories&mode=technicallibrary> (accessed 18 February 2013)

Wanke MM (2004). Canine brucellosis. *Animal Reproduction Science* 82–83:195–207.

## 4.3 Canine influenza

### 4.3.1 Background

Canine influenza is caused by the canine influenza virus (CIV), A/canine/Florida/43/H3N8. It is an emerging disease that was first recognised in the United States in 2004, and has been diagnosed in dogs in at least 30 states in that country (AVMA 2009; Buonavoglia and Martella 2007; Crawford et al. 2006). Infection with canine influenza virus is associated with a low mortality but high morbidity (Crawford et al. 2006), and is most apparent in kennels and shelters (Buonavoglia and Martella 2007). The virus plays a significant role in the pathogenesis of the canine respiratory disease complex.

Canine influenza has only been reported in dogs and all breeds are considered to be susceptible. As of May 2013, infection with canine influenza virus has not been reported in other species, including humans.

No evidence for venereal routes of transmission was found in the scientific literature.

Canine bartonellosis is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

### 4.3.2 Technical information

#### Epidemiology

The incubation period for CIV ranges from two to five days with infected dogs shedding virus for four to ten days from the initial day of clinical signs. Dogs of any age, breed and health status are susceptible. Peak virus shedding occurs during the preclinical incubation period and rapidly declines over the ensuing days until cessation by day 7 to 10. A proportion of infected dogs are asymptomatic but act as silent shedders of the virus. Transmission is by oronasal contact with infected dogs or contaminated fomites, and by inhalation of aerosols generated by coughing and sneezing (Crawford 2009).

Dogs housed in communal facilities such as kennels, shelters, pet stores, veterinary clinics or attending dog shows are at highest risk of exposure. Vaccination against CIV decreases both the likelihood of dogs becoming infected with CIV and viral shedding in subclinically infected animals (Cole and McNally 2009). Furthermore, vaccination reduces the severity of clinical signs (Larson et al. 2011). A CIV vaccine is available for use in the United States (Intervet 2010).

Currently, CIV is the only known influenza subtype circulating in dogs. It is established in dog populations in the United States. Experimental studies have shown that horses are susceptible to CIV infection but infected horses show only very mild clinical signs or infection is subclinical (Long et al. 2007).

## **Pathogenesis**

Influenza virus replicates in mucosal epithelium cells lining the airways from the nose to the terminal airways, in bronchiole gland epithelium and in pulmonary macrophages. Viral replication causes epithelial cell necrosis and destruction of the respiratory epithelial barrier, predisposing to secondary infection by a variety of commensal bacteria, including *E. coli*, *Klebsiella* spp., *Mycoplasma* spp, *Pasteurella multocida*, *Staphylococcus* spp.and *Streptococcus* spp. Most clinically affected dogs recover without complications but a proportion of dogs—less than 20%—infection leads to bronchopneumonia associated with virus-induced cell damage in the lower airway epithelium and complicated by secondary bacterial infection (Crawford 2009).

## **Clinical signs**

Clinical disease consists of acute onset of coughing, sneezing, nasal discharge and some ocular discharge. Coughing is the predominant sign and typically persists for two to three weeks. Dogs with pneumonia have high fever, inappetance, productive cough, increased respiratory rate and effort (Crawford 2009).

## **Diagnosis**

Canine influenza cannot be diagnosed on clinical signs alone as the clinical spectrum overlaps with that associated with other respiratory infections. Definitive diagnosis of canine influenza requires detection of virus in acutely ill dogs coupled with serology. Methods for virus detection include enzyme linked immunosorbent assay, (ELISA) for antigen, polymerase chain reaction (PCR) for nucleic acid and virus isolation. Successful diagnosis depends on sample collection during peak virus shedding early in the course of clinical disease. Serology is the most accurate and reliable diagnostic test to confirm CIV infection, especially in cases where PCR results are negative but the index of suspicion is high. Paired acute (sick for < 7 days ) and convalescent (10 to 14 days later) serum samples are necessary for diagnosis of recent active infection. Seroconversion is defined as at least a four-fold increase in CIV antibody titres between acute and convalescent sera (Crawford 2009).

## **Treatment**

Treatment consists of supportive care based on clinical signs and laboratory tests. Although there is no specific antiviral treatment for canine influenza, a variety of secondary bacterial infections may play a role in the course of infection. Antibiotics are indicated for dogs with fever, purulent nasal discharge, productive cough and pneumonia (Crawford 2009).

### **4.3.3 Current biosecurity measures**

- Dogs imported from the United States must be fully vaccinated against canine influenza.
- The second vaccination of an initial vaccination course or an annual booster vaccination must be administered between 12 months and 14 days before export.

#### 4.3.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by canine influenza:

- CIV infection appears to be restricted to dogs in the United States mainland.
- Peak shedding of virus occurs in the preclinical incubation phase of infection with a lesser amount of virus shedding that continues up to 10 days following the commencement of clinical signs.
- Subclinically infected dogs can also shed virus. A prolonged carrier and shedding status has not been reported following infection.
- Vaccination against CIV decreases both the likelihood of dogs becoming infected with CIV and viral shedding in subclinically infected animals.
- The consequences of CIV infection are typically moderate; most dogs recover without complications. Pneumonia associated with secondary bacterial infection occurs in a low proportion of CIV infected dogs but can be fatal.

#### 4.3.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for canine influenza continue to be warranted for dogs. It was also concluded that risk management measures for canine influenza are not warranted for dog semen.

The current biosecurity measure of pre-export vaccination would continue to provide appropriate risk management.

#### References

AVMA (American Veterinary Medical Association) (2009). Canine influenza virus background. AVMA, Schaumburg, Illinois.  
[http://www.avma.org/public\\_health/influenza/canine\\_bgnd.asp](http://www.avma.org/public_health/influenza/canine_bgnd.asp) (accessed 11 October 2011)

Buonavoglia C, Martella V (2007). Canine respiratory viruses. *Veterinary Research* 38:355–373.

Cole L, McNally A (2009). APHIS issues conditional license for canine influenza virus vaccine. United States Department of Agriculture, Animal and Plant Health Inspection Service, Washington DC.  
<http://www.aphis.usda.gov/newsroom/content/2009/06/caninevacc.shtml> (accessed 21 November 2011)

Crawford C, Dubovi EJ, Donis RO, Castleman WL, Gibbs EPJ, Hill RC, Katz JM, Ferro P, Anderson TC (2006). Canine influenza virus infection. In *Small animal and exotics: proceedings of the North American Veterinary Conference, 7–11 January 2006*, Orlando, Florida, North American Veterinary Conference (NAVC), Gainesville, pp. 625–626.

Crawford PC (2009) Canine influenza: epidemiology, clinical disease, and diagnosis. In *Michigan Veterinary Conference 2008 proceedings, 24–27 January, 2008, Lansing, Michigan*, pp. 1–4, Michigan Veterinary Medical Association, Michigan.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Intervet (2010). Nobivac® canine flu vaccine granted license by USDA. Schering-Plough Animal Health.  
<http://www.intervetusa.com/news/2010-06-09.aspx> (accessed 27 April 2011)

Larson LJ, Henningson J, Sharp P, Thiel B, Deshpande MS, Davis T, Jayappa H, Wasmoen T, Lakshmanan N, Schultz RD (2011). Efficacy of the canine influenza virus H3N8 vaccine to decrease severity of clinical disease after cochallenge with canine influenza virus and *Streptococcus equi* subsp. *zooepidemicus*. *Clinical and Vaccine Immunology* 18:559–564.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012> (accessed 16 January 2012)

## 4.4 Canine monocytic ehrlichiosis

### 4.4.1 Background

Canine monocytic ehrlichiosis (CME) is a tick-borne disease principally caused by infection with *Ehrlichia canis* (Neer and Harrus 2006). *E. canis* is a Gram-negative obligate intracellular bacterium belonging to the family Anaplasmataceae (CFSPH 2005; Dumler et al. 2001). CME is a multisystemic disorder that has high mortality rates in dogs during the chronic phase of infection (Mylonakis et al. 2004; Sidoti and Tringali 2009).

Canids, including domestic dogs, are vertebrate hosts for *E. canis* (Neer and Harrus 2006). Although there is evidence of infection in cats with an *E. canis*-like disease agent, species of *Ehrlichia* that naturally infect cats remain to be thoroughly investigated and identified (Breitschwerdt et al. 2002; Lappin et al. 2006; Lappin and Breitschwerdt 2006). A case of human infection with *E. canis* has also been reported (Perez et al. 2006), but the disease agent is not considered to have significant zoonotic potential (Day 2011).

CME may occasionally be caused by *E. chaffeensis* (CFSPH 2005). This zoonotic disease agent may infect a variety of vertebrate species, including dogs, and is the cause of human monocytic ehrlichiosis (Paddock and Childs 2003).

CME has been reported in Africa, America, Asia and Europe (Waner and Harrus 2000), particularly in tropical and temperate climates (CVBD World Forum 2011). *E. canis* and *E. chaffeensis* are exotic to Australia and New Zealand (Biosecurity New Zealand 2009). Although *E. canis* is exotic to Australia, its primary tick vector, *Rhipicephalus sanguineus*, is present (Little 2010; TAGS Inc. 2009).

CME is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

#### 4.4.2 Technical information

##### Epidemiology

Dogs and wild canids are the principal reservoir host for *E. canis* and act as the maintenance host for the primary tick vector, *R. sanguineus* (Little 2010; Nicholson et al. 2010). This vector is a three-host tick (Dantas-Torres 2008). Immature stage ticks become infected after feeding on infected dogs and are able to maintain the infection trans-stadially<sup>5</sup>. Adult ticks have also been shown to transmit the bacterium intrastadially to multiple hosts. This may be important in outbreak situations because adult male ticks have been demonstrated to move between hosts as they feed intermittently (Bremer et al. 2005; Little et al. 2007; Little 2010; Stich et al. 2008).

Other competent tick vectors for *E. canis* have been proposed, including *Dermacentor variabilis* (Johnson et al. 1998; Little 2010). The minimum duration of attachment by an infected tick that is required for transmission of *E. canis* to a naïve host is not known. The highest risk period for *E. canis* transmission is thought to be 24–72 hours after tick attachment, during the rapid ‘soaking’ phase of feeding (Davoust et al. 2003).

Transmission of *E. canis* occurs mainly during warmer months, when the tick vector is active (Harrus and Waner 2011). CME has an incubation period of 8–20 days following exposure of a dog to an infective tick. Infection may be acute (non-myelosuppressive), subclinical or chronic (myelosuppressive) and may progress through each phase (Harrus et al. 1999). The infection phases can range from 2 to 4 weeks (acute phase) and months to years (subclinical phase). An unknown percentage of subclinically infected dogs will eventually develop the chronic, severe form of the disease (Mylonakis et al. 2004; Skotarczak 2003).

Following acute infection, appropriate treatment leads to clinical recovery in most dogs (Harrus and Waner 2011). However, the ability of dogs to be effectively cured or eliminate the bacteria remains ambiguous (Neer et al. 2002).

---

<sup>5</sup> Passage of a microbial parasite from one life cycle stage of the vector to a subsequent stage

Seroprevalence data from endemic countries vary widely due to different testing methods and study populations. For dogs in Africa, infection prevalence estimates, based on serological and molecular detection techniques, range from 32% to 81%. In Europe, *E. canis* is primarily distributed in areas bordering the Mediterranean Sea. Seroprevalence estimates of 50% (Sardinia), 16.7% (northern Spain) and 9.7% (Italy) have been reported. In the United States, seroprevalences of 20.3% (Louisiana), 12% (Arizona) and 11.7% (Connecticut and New York) have been reported. Seroprevalence data from other countries include estimates of 30% (Israel), 22% (Thailand), 13.6% (Japan) and 0.2% (Malaysia) (Stich et al. 2008).

Infection with *E. chaffeensis* has been reported in dogs and humans in the United States (Paddock and Childs 2003). There has also been evidence of the disease agent in domestic dogs in Korea and Venezuela, and serological evidence of infection in humans in Argentina, Brazil, Burkina Faso, Chile, China, Israel, Italy, Korea, Mexico, Mozambique, Peru, Poland and Thailand (Yabsley 2010).

The epidemiology of *E. chaffeensis* is incompletely understood. White-tailed deer (*Odocoileus virginianus*) are currently recognised as the sole species capable of maintaining the transmission cycle of *E. chaffeensis* and are the primary reservoir host. However, this disease agent may infect a variety of vertebrate species, including dogs, coyotes, red foxes, deer, goats, lemurs and humans. *Amblyomma americanum* is thought to be the principal vector of *E. chaffeensis*, but other possible tick vectors have been identified including other *Amblyomma* spp., *D. variabilis*, *Ixodes pacificus* and *Ixodes ricinus* (CFSPH 2005; Nicholson et al. 2010; Paddock and Childs 2003; Yabsley 2010). *E. chaffeensis* DNA has also been detected in *R. sanguineus* ticks in Africa (Ndip et al. 2010).

Dogs experimentally infected with *E. chaffeensis* have shown evidence of bacteraemia up to 117 days post-infection (Yabsley 2010; Zhang et al. 2003). However, other experimental studies failed to demonstrate transmission of *E. chaffeensis* to naïve dogs from *A. americanum* that had been infected through exposure to experimentally inoculated dogs or deer (Ewing et al. 1995; Long et al. 2003; Yabsley 2010). The role of domestic animals in human monocytic ehrlichiosis is yet to be established (Neer et al. 2002).

No evidence for venereal transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

CME caused by *E. canis* is a multisystemic disorder with a variety of non-specific clinical signs that vary according to breed susceptibility and the pathogenicity of the particular infecting strain. The disease may also be complicated by co-infection with other arthropod-borne disease agents (Harrus and Waner 2011).

The acute phase of CME may be characterised by marked pyrexia, lethargy, depressed demeanour, anorexia, weight loss, lymphadenomegaly, splenomegaly,

bleeding abnormalities and ocular and neurological abnormalities (Harrus and Waner 2011).

The reasons for progression from the persistent, subclinical phase to the chronic phase of infection are unknown (Harrus and Waner 2011). The chronic phase of infection is associated with signs of emaciation, haemorrhage, peripheral oedema and hypotensive shock leading to death (Rikihiya et al. 1992). Bone marrow hypoplasia contributes to the terminal stage of the chronic phase. Bacterial septicæmia and/or bleeding may lead to death (Mylonakis et al. 2004).

Clinical disease may therefore be associated with severe morbidity and mortality (Little 2010). In a study conducted in Israel, 34.7% of dogs (17/49) diagnosed with CME died within one year of the onset of clinical signs despite receiving treatment (Harrus et al. 1997).

Disease caused by *E. chaffeensis* is typically subclinical or mild and may be clinically indistinguishable from disease caused by *E. canis* (CFSPH 2005; Nicholson et al. 2010). However, disease in dogs caused by *E. chaffeensis* is less well understood than that due to *E. canis* (CFSPH 2005).

### **Diagnosis**

Diagnosis of clinical CME is usually based on the history of the animal, clinical signs and laboratory test results (haematology and biochemistry). An indirect fluorescence antibody test (IFAT) is the gold standard serological test applied to detect immunoglobulin G (IgG) antibodies to *E. canis*. (Harrus and Waner 2011; Waner et al. 2001).

The time taken to seroconvert, as evidenced by first detection of IgG antibodies, may be influenced by the dose of infective organisms. Experimental studies based on IFAT results indicate seroconversion may occur between 2 and 28 days after infection (Iqbal et al. 1994; Neer et al. 2002; Neer and Harrus 2006; Waner et al. 2001). In a study of five dogs, all dogs seroconverted by day 6 post-inoculation with *E. canis*. In this study the anti-*E. canis* titre reached a plateau by days 10–12 (Iqbal et al. 1994). A study of six dogs reported detection of IgG titres by day 15 post-inoculation (Waner et al. 1996), while another study reported seroconversion in two dogs by day 10 with peak antibody levels on days 21 and 24 (Bremer et al. 2005). A study using a multivalent enzyme-linked immunosorbent assay (ELISA) found dogs seroconverted an average of 24 days post-inoculation (range: 17–35 days) (Gaunt et al. 2010). Dogs in which serological results are equivocal should have repeat testing performed at an interval of 2–3 weeks (Neer et al. 2002).

Anti-ehrlichial IgG antibodies may persist for several months to years after treatment (Bartsch and Greene 1996; Neer et al. 2002). The persistence of high IgG titres has been documented in clinically healthy dogs that are subclinically infected with *E. canis* (Waner et al. 2001).

Laboratories vary in the diagnostic serology end point used as evidence of exposure to *E. canis* (Neer et al. 2002). IFAT titre results of  $\geq 1:40$  are generally considered to be indicative of exposure to *E. canis* (Harrus and Waner 2011).

Due to serological cross-reactions between ehrlichial species, IFAT results cannot differentiate between antibodies to *E. chaffeensi*, *E. ewingi*, *E. ruminantium* and *E. canis* (Harrus and Waner 2011). Cross-reactivity may also occur with antibodies to *Neorickettsia helminthoeca* and *Anaplasma phagocytophilum* (Neer and Harrus 2006; Waner et al. 2001).

Western immunoblotting can be used to differentiate between infection by *E. canis* and *E. ewingii* (Harrus and Waner 2011).

Molecular detection methods, both conventional PCR and real-time PCR, have been developed for the detection of *E. canis* DNA, the latter being the more sensitive technique. Testing of spleen samples is considered to be more sensitive than blood or bone marrow samples in determining the effectiveness of treatment for CME (Harrus and Waner 2011).

PCR techniques provide advantages over serological screening techniques (e.g. IFAT), because they are able to detect the presence of *E. canis* in the acute phase of infection before seroconversion has occurred (Harrus and Waner 2011).

However, the diagnostic value of PCR based on blood samples is limited by the relatively short and variable period in which *E. canis* is present in the plasma of an infected dog (Waner et al. 1996). False negative results may occur, particularly if multiple assay (multiplex) real-time PCR testing is applied, which may reduce the sensitivity of *E. canis* detection. Therefore, negative results by PCR need to be interpreted with caution (Harrus and Waner 2011).

For the diagnosis of CME, it has been recommended that PCR should be done in conjunction with serology (Neer et al. 2002).

### **Treatment**

Amicarbalide, chloramphenicol, imidocarb dipropionate and tetracyclines have been reported to be effective in the treatment of CME. The Infectious Disease Study Group of the American College of Veterinary Internal Medicine recommends the oral administration of doxycycline (10 mg/kg daily for 28 days). Clinical improvement has been described within 1–2 days of administration in cases of acute phase disease and mild cases of chronic phase disease (Neer et al. 2002).

The efficacy of treatment with doxycycline to eliminate infection with *E. canis* remains unclear. Effective recovery from the acute phase of CME was demonstrated in each of five experimentally infected dogs, after treatment with doxycycline for 16 days (Harrus et al. 2004). Another study found that treatment of dogs in the post-acute, subclinical phase of infection with doxycycline for 14 days was not effective in eliminating infection. Juvenile ticks that were exposed to dogs (after doxycycline treatment) became infected with *E. canis* (Schaefer et al. 2007).

## Vector management

Prevention of CME in endemic areas requires an effective tick control strategy (Little et al. 2007). Effective tick control depends on regular treatment of dogs with an effective acaricide (in accordance with the manufacturer's recommendations) to prevent tick attachment. Kennels should be frequently treated since *R. sanguineus* can survive off the host for prolonged periods. For dogs visiting tick endemic regions, tick preventative measures should be practised. Owners should search animals daily for ticks, and physically remove and dispose of any ticks detected.

### 4.4.3 Current biosecurity measures

Within 30 days before export, blood samples must be obtained from dogs by a government-approved veterinarian for testing for evidence of *E. canis* infection by IFAT.<sup>6</sup> The test must produce a negative result at a dilution of 1:40.

## Vector management

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a registered acaricide effective against ticks on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs must be treated with a registered acaricide effective against ticks on contact; the acaricide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks.

### 4.4.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by CME:

- *E. canis* and *E. chaffeensis* are exotic to Australia.
- CME caused by *E. canis* may be associated with high morbidity and mortality rates in dogs.
- Dogs are considered the primary reservoir host for *E. canis*, and white-tailed deer are considered the primary reservoir host for *E. chaffeensis*.
- *Rhipicephalus sanguineus*, the primary tick vector for *E. canis*, is present in Australia.
- *Ehrlichia* spp. that naturally infect cats (and the significance of such infection) have not been determined.

---

<sup>6</sup> Dogs continuously resident in New Zealand since either birth or direct importation from Australia (whichever is applicable) do not require testing. Dogs imported from the Australian territory of Norfolk Island do not require testing.

- Persistent subclinical infection is a feature of *E. canis* infection in dogs and may last for years. Ticks are able to acquire *E. canis* infection from subclinically infected dogs post-treatment.
- Treatment of subclinically infected dogs with antibiotics (e.g. doxycycline) has not been shown to be reliable in eliminating *E. canis* infection. Antibody (IgG) titres following exposure to *E. canis* may persist for months to years. Persistently high IgG titres have been detected in subclinically infected dogs.
- PCR is a sensitive technique that is useful in detecting active infection. However, the short and variable time that *E. canis* is present in the plasma of an infected dog limits its value in detecting subclinical infection.
- PCR is most sensitive when performed on spleen samples. Collection of spleen samples is invasive and not practical for routine pre-export testing.
- Diagnostic serology using an IFAT is reliable for detecting the presence of IgG antibodies to *E. canis*.
- False positive serology (IFAT) results may occur due to cross-reactivity with other ehrlichial species (e.g. *E. chaffeensis* and *E. ewingii*).
- False negative serology results may occur in the acute phase if blood is collected before seroconversion has occurred.
- The incubation period for CME is 8–20 days with seroconversion reported to occur 2–28 days after experimental exposure to *E. canis*; seroconversion may be influenced by the dose of infectious organisms.
- Prophylactic treatment against tick vectors before serological testing for ehrlichiosis may reduce the risk that a dog is exposed to an infective tick immediately before blood sampling and be incubating infection at the time of export.

#### 4.4.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for CME continue to be warranted for dogs. In addition, it was concluded that risk management measures for CME are not warranted for cats, or for dog or cat semen.

The following biosecurity measures would provide appropriate risk management.

##### Pre-export measures (dogs)

###### Serology<sup>7</sup>

- Within the 21 days immediately before export, a blood sample must be collected from the dog and tested using an indirect fluorescent antibody test (IFAT) for *Ehrlichia canis* with a negative result at a serum dilution of 1:40 (unless an alternative cut-off value for a positive result is specified by the testing laboratory and is approved by the Department of Agriculture).

---

<sup>7</sup> Dogs continuously resident in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island since either birth or direct importation from Australia (whichever is applicable) do not require testing.

#### *Vector management*

- The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before IFAT blood sampling. The treatment must be repeated in accordance with the manufacturer's recommendation to maintain continued effectiveness until the day of export.
- The dog must be subjected to a thorough physical examination by a government-approved veterinarian within the five days<sup>10</sup> immediately before export and found to be visibly free from ticks.
- If a live and/or attached tick is detected at any examination, the tick must be removed and the following measures are to apply:
  - i. the dog must be re-treated with an acaricide effective against ticks on contact; and
  - ii. a blood sample must be collected from the dog at least 21 days following parasite detection and tested using an IFAT for *Ehrlichia canis* with a negative result at a serum dilution of 1:40.

#### **Post-arrival measures (dogs)**

##### *Vector management*

- As for post-arrival measures for external parasite control.
- To manage the biosecurity risks of CME, dogs may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### **References**

Bartsch RC, Greene RT (1996). Post-therapy antibody titers in dogs with ehrlichiosis: Follow-up study on 68 patients treated primarily with tetracycline and/or doxycycline. *Journal of Veterinary Internal Medicine* 10:271–274.

Biosecurity New Zealand (2009). *Import risk analysis: cats, dogs and canine semen*. New Zealand Government Ministry of Agriculture and Forestry, Wellington.

Breitschwerdt EB, Abrams-Ogg ACG, Lappin MR, Bienzle D, Hancock SI, Cowan SM, Clooten JK, Hegarty BC, Hawkins EC (2002). Molecular evidence supporting *Ehrlichia canis*-like infection in cats. *Journal of Veterinary Internal Medicine* 16:642–649.

Bremer WG, Schaefer JJ, Wagner ER, Ewing SA, Rikihisa Y, Needham GR, Jittapalapong S, Moore DL, Stich RW (2005). Transstadial and intrastadial experimental transmission of *Ehrlichia canis* by male *Rhipicephalus sanguineus*. *Veterinary Parasitology* 131:95–105.

CFSPH (The Center for Food Security and Public Health) (2005). Ehrlichiosis. CFSPH, Ames, Iowa State University.

<http://www.cfsph.iastate.edu/DiseaseInfo/disease.php?name=ehrlichiosis&lang=en> (accessed 27 June 2011)

CVBD (Canine Vector-Borne Diseases) World Forum (2011), Ehrlichiosis: pathogens. CVBD.

<http://www.cvbd.org/4029.0.html> (accessed 14 June 2011)

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. Department of Agriculture, Canberra.

<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Dantas-Torres F (2008). The brown dog tick, *Rhipicephalus sanguineus* (Latreille, 1806) (Acari: Ixodidae): from taxonomy to control. *Veterinary Parasitology* 152:173–185.

Davoust B, Mariq JL, Mercier S, Boni M, Vandeweghe A, Parzy D, Beugnet F (2003). Assay of fipronil efficacy to prevent canine monocytic ehrlichiosis in endemic areas. *Veterinary Parasitology* 112:91–100.

Day MJ (2011). One health: the importance of companion animal vector-borne diseases. *Parasites & Vectors* 4:49 doi:10.1186/1756-3305-4-49.

Dumler JS, Barbet AF, Bekker CPJ, Dasch GA, Palmer GH, Ray SC, Rikihisa Y, Rurangirwa FR (2001). Reorganization of genera in the families Rickettsiaceae and Anaplasmataceae in the order Rickettsiales: unification of some species of *Ehrlichia* with *Anaplasma*, *Cowdria* with *Ehrlichia* and *Ehrlichia* with *Neorickettsia*, descriptions of six new species combinations and designation of *Ehrlichia equi* and 'HGE agent' as subjective synonyms of *Ehrlichia phagocytophila*. *International Journal of Systematic and Evolutionary Microbiology* 51:2145–2165.

Ewing SA, Dawson JE, Kocan AA, Barker RW, Warner CK, Panciera RJ, Fox JC, Kocan KM, Blouin EF (1995). Experimental transmission of *Ehrlichia chaffeensis* (Rickettsiales: Ehrlichieae) among white-tailed deer by *Amblyomma americanum* (Acari: Ixodidae). *Journal of Medical Entomology* 32:368–374.

Gaunt SD, Beall MJ, Stillman BA, Lorentzen L, Diniz PPVP, Chandrashekar R, Breitschwerdt EB (2010). Experimental infection and co-infection of dogs with *Anaplasma platys* and *Ehrlichia canis*: haematologic, serologic and molecular findings. *Parasites and Vectors* 3:33–42.

Harrus S, Kass PH, Klement E, Waner T (1997). Canine monocytic ehrlichiosis: a retrospective study of 100 cases, and an epidemiological investigation of prognostic indicators for the disease. *Veterinary Record* 141:360–363.

- Harrus S, Kenny M, Miara L, Aizenberg I, Waner T, Shaw S (2004). Comparison of simultaneous splenic sample PCR with blood sample PCR for diagnosis and treatment of experimental *Ehrlichia canis* infection. *Antimicrobial Agents and Chemotherapy* 48:4488–4490.
- Harrus S, Waner T (2011). Diagnosis of canine monocytotropic ehrlichiosis (*Ehrlichia canis*): an overview. *Veterinary Journal* 187:292–296.
- Harrus S, Waner T, Bark H, Jongejan F, Cornelissen AWCA (1999). Recent advances in determining the pathogenesis of canine monocytic ehrlichiosis. *Journal of Clinical Microbiology* 37:2745–2749.
- Iqbal Z, Chaichanasiriwithaya W, Rikihisa Y (1994). Comparison of PCR with other tests for early diagnosis of canine ehrlichiosis. *Journal of Clinical Microbiology* 32:1658–1662.
- Johnson EM, Ewing SA, Barker RW, Fox JC, Crow DW, Kocan KM (1998). Experimental transmission of *Ehrlichia canis* (Rickettsiales : Ehrlichieae) by *Dermacentor variabilis* (Acari : Ixodidae). *Veterinary Parasitology* 74: 277-288.
- Lappin MR, Breitschwerdt EB (2006). Feline mononuclear ehrlichiosis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 224–227.
- Lappin MR, Griffin B, Brunt J, Riley A, Burney D, Hawley J, Brewer MM, Jensen WA (2006). Prevalence of *Bartonella* species, *Haemoplasma* species, *Ehrlichia* species, *Anaplasma phagocytophilum*, and *Neorickettsia risticii* DNA in the blood of cats and their fleas in the United States. *Journal of Feline Medicine and Surgery* 8:85–90.
- Little SE (2010). Ehrlichiosis and anaplasmosis in dogs and cats. *Veterinary Clinics of North America: Small Animal Practice* 40:1121–1140.
- Little SE, Hostetler J, Kocan KM (2007). Movement of *Rhipicephalus sanguineus* adults between co-housed dogs during active feeding. *Veterinary Parasitology* 150:139–145.
- Long SW, Zhang X, Zhang J, Ruble RP, Teel P, Yu XJ (2003). Evaluation of transovarial transmission and transmissibility of *Ehrlichia chaffeensis* (Rickettsiales: Anaplasmataceae) in *Amblyomma americanum* (Acari: Ixodidae). *Journal of Medical Entomology* 40:1000–1004.
- Mylonakis ME, Koutinas AF, Breitschwerdt EB, Hegarty BC, Billinis CD, Leontides LS, Kontos VS (2004). Chronic canine ehrlichiosis (*Ehrlichia canis*): a retrospective study of 19 natural cases. *Journal of the American Animal Hospital Association* 40:174–184.
- Ndip L, Ndip R, Esemu S, Walker D, McBride J (2010). Predominance of *Ehrlichia chaffeensis* in *Rhipicephalus sanguineus* ticks from kennel-confined dogs in Limbe, Cameroon. *Experimental and Applied Acarology* 50:163–168.

- Neer TM, Breitschwerdt EB, Greene RT, Lappin MR (2002). Consensus statement of ehrlichial disease of small animals from the infectious disease study group of the ACVIM. *Journal of Veterinary Internal Medicine* 16:309–315.
- Neer TM, Harrus S (2006). Ehrlichiosis, neorickettsiosis, anaplasmosis and Wolbachia infection. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 203–216.
- Nicholson WL, Allen KE, McQuiston JH, Breitschwerdt EB, Little SE (2010). The increasing recognition of rickettsial pathogens in dogs and people. *Trends in Parasitology* 26:205–212.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012> (accessed 16 January 2012)
- Paddock CD, Childs JE (2003). *Ehrlichia chaffeensis*: a prototypical emerging pathogen. *Clinical Microbiology Reviews* 16:37–64.
- Perez M, Bodor M, Zhang CB, Xiong QM, Rikihisa Y (2006). Human infection with *Ehrlichia canis* accompanied by clinical signs in Venezuela. *Century of Rickettsiology: Emerging, Reemerging Rickettsioses, Molecular Diagnostics, and Emerging Veterinary Rickettsioses* 1078:110–117.
- Rikihisa Y, Ewing SA, Fox JC, Siregar AG, Pasaribu FH, Malole MB (1992). Analyses of *Ehrlichia canis* and a canine granulocytic *Ehrlichia* infection. *Journal of Clinical Microbiology* 30:143–148.
- Schaefer JJ, Needham GR, Bremer WG, Rikihisa Y, Ewing SA, Stich RW (2007). Tick acquisition of *Ehrlichia canis* from dogs treated with doxycycline hyclate. *Antimicrobial Agents and Chemotherapy* 51:3394–3396.
- Sidoti E, Tringali G (2009). Ehrlichioses and anaplasmoses: (re)emerging tickborne zoonoses in humans and in animals. *Journal of Preventative Medicine and Hygiene* 50:9–18.
- Skotarczak B (2003). Canine ehrlichiosis. *Annals of Agricultural and Environmental Medicine* 10:137–141.
- Stich RW, Schaefer JJ, Bremer WG, Needham GR, Jittapalapong S (2008). Host surveys, ixodid tick biology and transmission scenarios as related to the tick-borne pathogen, *Ehrlichia canis*. *Veterinary Parasitology* 158:256–273.
- TAGS Inc. (2009). Ticks of Australia. Tick Alert: Group Support. <http://web.archive.org/web/20081007074227/http://www.tickalert.org.au/ticksaust.htm> (accessed 7 May 2012)
- Waner T, Harrus S (2000). Canine monocytic ehrlichiosis (CME). In *Recent advances in canine infectious diseases*, Carmichael LE (ed.), International Veterinary Information

Service, Ithaca, NY.

[www.ivis.org/advances/Infect\\_Dis\\_Carmichael/waner/IVIS.pdf](http://www.ivis.org/advances/Infect_Dis_Carmichael/waner/IVIS.pdf) (accessed 13 July 2011)

Waner T, Harrus S, Jongejan F, Bark H, Keysary A, Cornelissen AWCA (2001). Significance of serological testing for ehrlichial diseases in dogs with special emphasis on the diagnosis of canine monocytic ehrlichiosis caused by *Ehrlichia canis*. *Veterinary Parasitology* 95:1–15.

Waner T, Rosner M, Harrus S, Naveh A, Zass R, Keysary A (1996). Detection of ehrlichial antigen in plasma of beagle dogs with experimental acute *Ehrlichia canis* infection. *Veterinary Parasitology* 63:331–335.

Yabsley MJ (2010). Natural history of *Ehrlichia chaffeensis*: vertebrate hosts and tick vectors from the United States and evidence for endemic transmission in other countries. *Veterinary Parasitology* 167:136–148.

Zhang XF, Zhang JZ, Long SW, Ruble RP, Yu XJ (2003). Experimental *Ehrlichia chaffeensis* infection in beagles. *Journal of Medical Microbiology* 52:1021–1026.

## 4.5 Canine pulmonary angiostrongylosis

### 4.5.1 Background

Canine pulmonary angiostrongylosis (CPA) is a parasitic disease caused by infection with the metastrongyloid nematode *Angiostrongylus vasorum* (French heartworm) (Koch and Willeesen 2009; Verzberger-Epshtein et al. 2008).

Domestic dogs and wild canids are definitive hosts of the parasite and more than 25 species of gastropods (snails and slugs) have been identified as intermediate hosts (Koch and Willeesen 2009). Amphibians may also be able to act as paratenic and/or intermediate hosts<sup>8</sup> (Conboy 2011; Morgan et al. 2005).

Disease in dogs ranges from subclinical to fatal. Clinical signs associated with cardiorespiratory disease, central nervous system disease and/or coagulopathies may be observed (Conboy 2011).

*A. vasorum* infection has been identified as an emerging threat (Conboy 2011; Helm et al. 2010). Since first described in France in 1866, the parasite has since been identified in tropical, subtropical and temperate regions of Africa, Europe, and North and South America (Conboy 2011; Koch and Willeesen 2009). Distribution has traditionally been characterised by discrete endemic foci with only sporadic occurrences outside these foci. However, there has been an increase in the number of diagnosed cases and an apparent expansion of the geographic distribution of the parasite (Koch and Willeesen 2009).

---

<sup>8</sup> A paratenic host is one in which the agent is mechanically transmitted without further development; an intermediate host is one in which the agent undergoes some further development (Thrusfield 2005).

Suitable intermediate hosts are present in Australia, including *Bradybaema similaris*, *Deroceras reticulatum*, *Helix aspersa*, *Lehmannia flava* and *Physaspp.* (Tebb et al. 2007). However, the only known reports of *A. vasorum* in Australia are larvae identified in the faeces of a dog in Queensland (Roberts 1940) and the diagnosis of CPA in a dog imported from a known endemic area in the United Kingdom (Tebb et al. 2007).

CPA is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

#### 4.5.2 Technical information

##### Epidemiology

*A. vasorum* has an indirect life cycle—definitive hosts are infected by ingesting intermediate hosts that contain infective third-stage larvae (Helm et al. 2010). Infection may also occur through ingestion of liberated third-stage larvae present in the environment (Ferdushy and Hasan 2010).

There is a seasonal pattern for the occurrence of CPA, with the highest incidence of clinical disease described in winter and spring. This corresponds with a seasonal increase in exposure to populations of intermediate hosts and/or increased environmental survival of free larvae (Taubert et al. 2009).

Adult parasites reside in the pulmonary arteries and the right ventricle of the heart of definitive hosts (Schnyder et al. 2009). Ova hatch in pulmonary capillaries and mature into first stage (L1) larvae. These larvae migrate into the airways and are subsequently coughed up, swallowed and passed in the faeces of infected definitive hosts (Helm et al. 2010; Tebb et al. 2007). Reported prepatent period varies from 28 to 108 days (Helm et al. 2010).

If untreated, dogs may remain infected for life and excrete larvae for prolonged periods (Helm et al. 2010; Koch and Willesen 2009).

Wild canids have been proposed as a reservoir for the parasitic infection of domestic dogs (Chapman et al. 2004; Morgan et al. 2005).

No evidence for venereal routes of transmission in dogs was found in the scientific literature.

Mapping the global prevalence of *A. vasorum* infection is difficult due to the majority of cases remaining undiagnosed and varying levels of awareness of the disease (Helm et al. 2010). In endemic countries, the prevalence may be higher in fox populations than in dog populations (Helm et al. 2010). A study of the red fox population in Newfoundland, Canada, reported an estimated prevalence of infection with *A. vasorum* of 56% (Jeffery et al. 2004).

There is little published information on the prevalence of subclinical infection in domestic dogs in endemic areas (Helm et al. 2010). Studies have investigated the prevalence of infection in specific dog populations in endemic countries; for

example, in dogs presenting to veterinary practices with consistent clinical signs or in specific groups of dogs (e.g. hunting dogs or greyhounds). Estimates of prevalence ranged from 0.3% to 9.8%, with higher prevalence reported in hunting dogs (Koch and Willesen 2009; Taubert et al. 2009).

### **Clinical signs**

There is a wide range of clinical signs associated with *A. vasorum* infection in dogs. Cardiorespiratory signs, particularly those relating to verminous pneumonia, such as coughing, dyspnoea, tachypnoea and gagging, are the most common presenting signs. A variety of signs associated with coagulopathies and neurological disease may also be observed (Helm et al. 2010).

Non-specific clinical signs such as weight loss, anorexia, lethargy and depression may be observed. Although sudden death following infection can occur, there is evidence that disease is typically subclinical after infection and dogs may be infected for months to years before showing clinical signs (Helm et al. 2010; Verzberger-Epshtein et al. 2008).

The range of non-specific clinical presentations associated with CPA means that clinical signs cannot be relied upon to confirm or exclude the presence of infection (Helm et al. 2010).

### **Diagnosis**

CPA can be diagnosed through the identification of L1 larvae in faeces or bronchial mucus (Conboy 2011). The Baermann technique, or modifications of this technique, remains the test of choice for faecal examination (Conboy 2011; Koch and Willesen 2009). However, faecal examination techniques are dependent on knowledge of larval shedding patterns to detect infection and are ineffective in detecting infection during the prepatent period, which can be prolonged (Helm et al. 2010; Verzberger-Epshtein et al. 2008). A single Baermann test is likely to detect up to 50% of infected dogs (Morgan and Shaw 2010). To increase sensitivity of the Baermann test, it has been recommended that samples are collected and tested over three consecutive days (Ferdushy and Hasan 2010).

Molecular and serological tests have been developed to improve the accuracy and sensitivity of diagnosis. This includes polymerase chain reaction and serological tests such as enzyme-linked immunosorbent assay and Western immunoblotting. These tests are not currently commercially available (Conboy 2011; Helm et al. 2010).

### **Treatment**

A number of treatment options for CPA have been described. Oral administration of fenbendazole for 5 to 21 days (Helm et al. 2010) has replaced the administration of levamisole and ivermectin as the preferred treatment method (Koch and Willesen 2009). However, an optimal dose and duration of therapy has not been defined (Tebb et al. 2007).

Treatment with newer formulations of macrocyclic lactones, milbemycin oxime and moxidectin have been reported (Conboy 2004; Helm et al. 2010; Schnyder et al. 2009; Willesen et al. 2007). This includes a topical imidacloprid/moxidectin spot-on solution (Schnyder et al. 2009; Willesen et al. 2007). In one study using Baermann tests, the efficacies of a single administration of a topical imidacloprid/moxidectin spot-on solution and a 20-day course of oral fenbendazole were reported to be 85.2% and 91.3%, respectively (Willesen et al. 2007).

#### 4.5.3 Current biosecurity measures

There are no specific biosecurity measures for CPA. Current biosecurity measures for internal parasites are:

- Within four days before export, dogs and cats must be treated with an approved anthelmintic that is effective against nematodes and cestodes. The active ingredients and dose rate must be recorded on the veterinary certificate.

#### 4.5.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by CPA:

- CPA is not an OIE-listed disease and is not a nationally notifiable disease in Australia.
- *A. vasorum* has a wide geographic distribution and is known to occur in dog populations in Africa, Europe, and North and South America.
- There is a long history of importation of dogs from countries where CPA is endemic. One case of CPA was reported in Australia in 2007.
- Prevalence estimates in endemically infected countries are not readily available, but are likely to vary considerably between regions and within a population based on exposure to infected intermediate or paratenic hosts.
- The anthelmintic treatment specified in current import requirements is aimed at parasites of the gastrointestinal tract (hookworm, roundworm, tapeworm, whipworm,) and is unlikely to eliminate infection with *A. vasorum*.
- Although acute fatal infection has been reported, infection is typically subclinical for a prolonged period of time (months to years) before clinical signs appear.
- Recommended diagnostic testing (Baermann technique) has a relatively low sensitivity of detection (i.e. a high number of false negative results) making it unreliable as a screening test.
- There is limited information on the efficacy of treatment options in dogs in the general population. Macrocyclic lactones appear to be reasonably effective in reducing parasitic burdens and clinical manifestations.

#### 4.5.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for CPA are not warranted for dogs or their semen.

#### References

- Chapman PS, Boag AK, Guitian J, Boswood A (2004). *Angiostrongylus vasorum* infection in 23 dogs (1999-2002). *Journal of Small Animal Practice* 45:435–440.
- Conboy G (2004). Natural infections of *Crenosoma vulpis* and *Angiostrongylus vasorum* in dogs in Atlantic Canada and their treatment with milbemycin oxime. *Veterinary Record* 155:16–18.
- Conboy GA (2011). Canine angiostrongylosis: the French heartworm: an emerging threat in North America. *Veterinary Parasitology* 176:382–389.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011), National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Ferdushy T, Hasan M (2010). *Angiostrongylus vasorum*: the ‘French heartworm’. *Parasitology Research* 107:765–771.
- Helm JR, Morgan ER, Jackson MW, Wotton P, Bell R (2010). Canine angiostrongylosis: an emerging disease in Europe. *Journal of Veterinary Emergency and Critical Care* 20:98–109.
- Jeffery RA, Lankester MW, McGrath MJ, Whitney HG (2004). *Angiostrongylus vasorum* and *Crenosoma vulpis* in red foxes (*Vulpes vulpes*) in Newfoundland, Canada. *Canadian Journal of Zoology* 82:66–74.
- Koch J, Willesen JL (2009). Canine pulmonary angiostrongylosis: an update. *The Veterinary Journal* 179:348–359.
- Morgan E, Shaw S (2010). *Angiostrongylus vasorum* infection in dogs: continuing spread and developments in diagnosis and treatment. *Journal of Small Animal Practice* 51:616–621.
- Morgan ER, Shaw SE, Brennan SF, De Waal TD, Jones BR, Mulcahy G (2005). *Angiostrongylus vasorum*: a real heartbreaker. *Trends in Parasitology* 21:49–51.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Roberts FHS (1940). Notes on some helminths infesting domestic animals in Queensland. *Australian Veterinary Journal* 16:30–33.

Schnyder M, Fahrion A, Ossent P, Kohler L, Webster P, Heine J, Deplazes P (2009). Larvicidal effect of imidacloprid/moxidectin spot-on solution in dogs experimentally inoculated with *Angiostrongylus vasorum*. *Veterinary Parasitology* 166:326–332.

Taubert A, Pantchev N, Vrhovec MG, Bauer C, Hermosilla C (2009). Lungworm infections (*Angiostrongylus vasorum*, *Crenosoma vulpis*, *Aelurostrongylus abstrusus*) in dogs and cats in Germany and Denmark in 2003–2007. *Veterinary Parasitology* 159:175–180.

Tebb AI, Johnson VS, Irwin PJ (2007). *Angiostrongylus vasorum* (French heartworm) in a dog imported into Australia. *Australian Veterinary Journal* 85:23–28.

Thrusfield M (2005). *Veterinary epidemiology*, 3rd edn, Blackwell Science Ltd, Ames, Iowa.

Verzberger-Epshtein I, Markham RJF, Sheppard JA, Stryhn H, Whitney H, Conboy GA (2008). Serologic detection of *Angiostrongylus vasorum* infection in dogs. *Veterinary Parasitology* 151:53–60.

Willesen JL, Kristensen AT, Jensen AL, Heine J, Koch J (2007). Efficacy and safety of imidacloprid/moxidectin spot-on solution and fenbendazole in the treatment of dogs naturally infected with *Angiostrongylus vasorum* (Baillet, 1866). *Veterinary Parasitology* 147:258–264.

## 4.6 Chagas' disease

### 4.6.1 Background

Chagas' disease (American trypanosomiasis) is caused by *Trypanosoma cruzi*, a haemoflagellate protozoan parasite. *T. cruzi* infects humans and a wide range of domestic and wild animals. Dogs and cats are common and important hosts of the organism (Barr 1991). Disease may be subclinical or include clinical signs of right-sided heart failure and/or neurological deficits (Meurs et al. 1998).

Chagas' disease is widespread throughout Central and South America. Cases have also been reported in the southern United States in both humans and dogs (Meurs et al. 1998). There have not been any reports of feline trypanosomiasis in North America (Barr 2006).

Chagas' disease is not an OIE-listed disease (OIE 2012), but it is a nationally notifiable disease in Australia (DAFF 2011).

### 4.6.2 Technical information

#### Epidemiology

*T. cruzi* is transmitted by haematophagous insects of the family Reduviidae (subfamily Triatominae). These insects ingest circulating trypomastigotes in the blood meal they obtain from a vertebrate host. Humans and domestic animals (mainly dogs, cats and guinea pigs) are the main reservoirs of infection. Sylvatic

reservoir hosts of *T. cruzi* include armadillos, opossums and raccoons, as well as various mouse, rat and squirrel species (Barr 2009). The host becomes infected when trypomastigotes in the insect's faeces invade the skin or mucous membranes through bite wounds or abrasions (Zeledon 1974). Dogs might also become infected by eating contaminated meat or infected insects (Chapman, Jr. and Hanson 1984). Other modes of transmission include transplacental and blood transfusion (Zeledon 1974).

Although there are many triatomine species that feed on humans, a close association (either direct or indirect—e.g. via contaminated food or beverages) between triatomines and humans (and domestic animals) is required for transmission of *T. cruzi*. The most domestically adapted vector species is *Triatoma infestans* and it is the principal vector across much of South America (Zeledon 1974). Other vectors include *Tr. dimidiata* and *Rhodnius prolixus*, which also display the appropriate behaviour for effective transmission of *T. cruzi*. These parasites feed on blood from both people and domestic reservoir mammals, reproduce prolifically and defecate soon after taking a blood meal, often on the host near the bite wound (Zeledón and Rabinovich 1981). Infection prevalence of 100% has been reported in *Tr. infestans* in equatorial regions of Central America (Barr 2009).

A low infection prevalence of approximately 20% has been reported for the two principal vectors in the United States (*Tr. protracta* and *Tr. sanguisuga*). Both display different feeding habits to *Tr. infestans*, and defecate about 20 minutes after feeding, often after leaving the host (Barr 2009). Most cases of Chagas' disease in the United States are in people that have emigrated from Latin America (Kirchhoff 1993). Other than a few instances reported in Texas (Beard et al. 2003), domestic transmission cycles are uncommon.

*Tr. leopoldi* is the only triatomine to have been identified in Australia. It is located in a remote region in the Iron Range National Park, Cape York, Queensland (Monteith 1974). The host range and preference of *Tr. leopoldi* is unknown, but might include birds and bats. No information on its feeding behaviour was found in the published scientific literature. The remote location and habitat preference of this triatomine suggest it is unlikely to provide an effective pathway for establishment and spread of *T. cruzi* from an infected dog.

No evidence for venereal routes of transmission in dogs or cats was found in the scientific.

### **Clinical signs**

Acute disease occurs mainly in young dogs less than one year of age with clinical signs referable to right-sided heart failure and cardiac arrhythmias (Barr 2006). Some animals may also show neurological signs referable to meningoencephalitis. Survivors of acute disease can become aparasitaemic without clinical signs and may later develop chronic myocarditis and cardiac dilatation. Many persistently infected dogs remain subclinically affected for life (Barr 2009).

Little is known about the occurrence of feline trypanosomiasis (Barr 2006).

## Diagnosis

During acute disease, trypomastigotes may be detected on examination of a blood smear. In most dogs, circulating trypomastigotes are evident as early as three days post-infection, reaching maximum levels during the peak period of the acute disease (two to three weeks post-infection) (Barr 1991).

Serological tests include indirect fluorescent antibody tests, direct haemagglutination and complement fixation tests (Chapman and Hanson 1984). These tests confirm the presence of antibodies to *T. cruzi*, although there is cross-reactivity with antibodies to leishmania (Barr 2009). As antibody levels remain markedly elevated throughout infection, serology is the preferred method for diagnosing canine Chagas' disease (Barr 1991).

## Treatment

Antiprotozoal agents are not highly effective against *T. cruzi*. Benznidazole, a nitroimidazole derivative, has been reported to be effective in reducing clinical signs associated with acute canine disease, but does not appear to be effective in eliminating infection (Barr 2009; Viotti et al. 1994).

### 4.6.3 Current biosecurity measures

There are no specific biosecurity measures for Chagas' disease.

### 4.6.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by Chagas' disease:

- Chagas' disease is not OIE-listed, but is a nationally notifiable disease in Australia.
- Chagas' disease is endemic in Central and South America where it is a significant human health problem; it also occurs in the southern United States.
- Dogs and cats are recognised reservoir hosts of *T. cruzi*, as are humans.
- Treatment does not reliably eliminate infection.
- At least one triatomine (*Tr. leopoldi*) has been identified in Australia; however, its remote location and habitat preference suggest it is unlikely to provide an effective pathway for establishment and spread of *T. cruzi* from an infected dog.

### 4.6.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for Chagas' disease are not warranted for dogs, cats or their semen.

## References

Barr SC (1991). American trypanosomiasis in dogs. *Compendium on Continuing Education for the Practicing Veterinarian* 13:745–755.

- Barr SC (2006). Trypanosomiasis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 676–685.
- Barr SC (2009). Canine Chagas' disease (American trypanosomiasis) in North America. *The Veterinary Clinics of North America. Small Animal Practice* 39:1055–1064.
- Beard CB, Pye G, Steurer FJ, Rodriguez R, Campman R, Peterson AT, Ramsey J, Wirtz RA, Robinson LE (2003). Chagas' disease in a domestic transmission cycle, southern Texas, USA. *Emerging Infectious Diseases* 9:103–105.
- Chapman WL, Jr., Hanson WL (1984). American trypanosomiasis. In *Clinical microbiology and infectious diseases of the dog and cat*. Greene CE (ed.), Saunders Elsevier, Philadelphia, pp. 757–763.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Kirchhoff LV (1993). American trypanosomiasis (Chagas' disease)—a tropical disease now in the United States. *New England Journal of Medicine* 329:639–644.
- Meurs KM, Anthony MA, Slater M, Miller MW (1998). Chronic *Trypanosoma cruzi* infection in dogs: 11 cases (1987–1996). *Journal of the American Veterinary Medical Association* 213:497–500.
- Monteith GB (1974). Confirmation of the presence of Triatominae (Hemiptera: Reduviidae) in Australia, with notes on Indo-Pacific species. *Journal of the Australian Entomological Society* 13:89–94.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Viotti R, Vigliano C, Armenti H, Segura E (1994). Treatment of chronic Chagas' disease with benznidazole: clinical and serologic evolution of patients with long-term follow-up. *American Heart Journal* 127:151–162.
- Zeledon R (1974). Epidemiology, modes of transmission and reservoir hosts of Chagas' disease. *Ciba Foundation Symposium* 20:51–85.
- Zeledón R, Rabinovich JE (1981). Chagas' disease: an ecological appraisal with special emphasis on its insect vectors. *Annual Review of Entomology* 26:101–133.

## 4.7 Hepatozoonosis

### 4.7.1 Background

Hepatozoonosis is caused by haemoprotozoans of the phylum Apicomplexa, family Hepatozoidae. Canine hepatozoonosis is caused by *Hepatozoon canis* and *H. americanum* (Baneth et al. 2003; Baneth 2006). *Hepatozoon* spp. that infect cats have not been clearly documented, with authors attributing disease in cats to *H. felis* (Baneth 2011) and *H. canis*, or species closely related to *H. canis* (Jittapalapong et al. 2006; Rubini et al. 2006).

Infection in dogs is described as accidental, dogs becoming infected by swallowing infected ticks either when grooming themselves or ingesting tick-infested prey (Ewing and Panciera 2003; Johnson et al. 2009). *H. canis* has been found in Africa, Asia, Europe, the Middle East and South America, and generally produces mild disease in dogs. *H. canis* and *H. canis*-like organisms are present in dogs in southern areas of the United States (Allen et al. 2008; Li et al. 2008). *H. americanum* is an emerging disease, spreading from the south-eastern United States where it was originally identified (Li et al. 2008). Infection can cause severe disease in dogs and may be fatal if untreated (Macintyre et al. 2006).

Hepatozoonosis is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

### 4.7.2 Technical information

#### Epidemiology

*Rhipicephalus sanguineus* becomes infected with *H. canis* by feeding on a parasitaemic dog, and dogs can become infected by ingesting the tick (Forlano et al. 2005). Intra-uterine transmission from dam to pups can occur (Baneth 2011; Murata et al. 1993).

The life cycle of *H. canis* including dog and tick stages takes approximately 81 days. Gametocytes appear in the dog's blood around 28 days following experimental infection (Baneth et al. 2001; Baneth 2011).

A number of wild canids have been reported as infected with *H. canis* or a *H. canis*-like species, including the red fox (*Vulpes vulpes*), the crab-eating fox (*Ceronyon thous*), the black-backed jackal (*Canis mesomelas*), the golden jackal (*Canis aureus*), the African wild dog (*Lycaon pictus*) and the hyena (*Crocuta crocuta*) (Baneth 2011).

Transmission of *H. americanum* is similar to that of *H. canis* except the tick vector is *A. maculatum*. Intra-uterine transmission has not been demonstrated and infection can also occur by predation on small animals (Baneth 2011; Ewing et al. 2002; Johnson et al. 2009). *Amblyomma ovale* has also been implicated as a host for *Hepatozoon* spp. in Brazil (Forlano et al. 2005). The tick vector for feline hepatozoonosis is unknown. *Hepatozoon* infection has been reported from cats in France, India, Israel, Nigeria and South Africa.

In dogs infected experimentally with *H. americanum*, parasites were found in blood leukocytes 28–50 days post-exposure, histological lesions in skeletal muscle biopsies within 21 days and clinical signs observed 4–5 weeks post-exposure (Pancieria et al. 1999). *H. americanum* has been reported in dogs in numerous southern states of the United States mainland. (Cummings et al. 2005; Li et al. 2008). Coyotes may be the primary vertebrate host for *H. americanum*. Ground-dwelling birds and small rodents could be involved in the cycle as hosts for larval and nymphal stages of *A. maculatum*, with large herbivores reported to be the preferred host for adult *A. maculatum*. Infection is inferred to be transmitted trans-stadially (Ewing et al. 2002).

Infection by *Hepatozoon* spp. in both dogs and cats can be influenced or reactivated by co-infections with other disease agents. Co-infection with parvovirus, *Ehrlichia canis*, *Toxoplasma gondii* and *Leishmania infantum* has been reported in dogs. Infection in cats is often associated with immunosuppressive viral diseases such as feline leukaemia virus (FeLV) or feline immunodeficiency virus (FIV) (Baneth 2002).

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

*H. canis* infection generally produces a subclinical to mild disease, with a low level parasitaemia. In dogs with severe infection, significant parasitaemia occurs, with up to 100% of peripheral blood neutrophils infected (Baneth 2011). Infected dogs may show anaemia and dogs with a high parasitaemic load may develop hepatitis, pneumonia, glomerulonephritis and weight loss.

*H. americanum* produces a severe disease that frequently leads to death. The disease may be acute, or have a waxing and waning pattern. A marked neutrophilia is present. Pyrexia, depression, anaemia and lethargy have been observed in infected dogs. Muscle wasting and gait abnormalities such as limb stiffness, recumbency or inability to rise can occur due to periosteal bone proliferation and myositis. There can be a copious mucopurulent ocular discharge (Baneth 2011; Ewing and Panciera 2003). Chronic wasting can be followed by death within one year if the dog is untreated (Macintire et al. 1997).

*H. americanum* usually produces a relatively low parasitaemia compared to *H. canis*, typically parasitising less than 0.1% of leukocytes (Baneth 2011).

### **Diagnosis**

Hepatozoonosis can be diagnosed by microscopic examination of blood smears for *H. canis* gamonts, which have been found in neutrophils and monocytes at 28 days post-infection (Baneth 2002). Examination of blood smears is less suitable for detecting *H. americanum* due to the lower level of parasitaemia associated with *H. americanum* infection. An indirect fluorescent antibody test or enzyme-linked immunosorbent assay (ELISA) can detect *H. canis* antibodies (Baneth 2002). An ELISA for *H. americanum* antibodies developed by Oklahoma State University has a sensitivity of 93% and specificity of 96% (Baneth 2011; Mathew et al. 2001).

Standard polymerase chain reaction (PCR) is more sensitive than microscopy for diagnosis of hepatozoonosis (Li et al. 2008) but is not highly sensitive and more suitable for application in clinical diagnosis than as a screening test to detect a subclinical carrier status (Criado-Fornelio et al. 2007). A quantitative PCR assay has been developed that appears to be particularly useful for detecting co-infection in dogs, infection in cats and detection of carrier animals (Criado-Fornelio et al. 2007).

#### **Treatment**

Imidocarb dipropionate is used for the treatment of *H. canis*, but the parasite may not be completely eliminated (Sasanelli et al. 2010).

Prolonged treatment with a combination of clindamycin, pyrimethamine and trimethoprim-sulphadiazine is required for treatment of *H. americanum*. Post-treatment relapses have been prevented by the administration of the coccidiostat decoquinate given every 12 hours for two years (Potter and Macintire 2010). Decoquinate prevents development of the parasites in the definitive host but may not be effective against all developmental stages (Macintire et al. 2001).

#### **Vector management**

Tick control provides the best method of prophylaxis against hepatozoonosis. Effective tick prevention depends on regular treatment of dogs and cats with a suitable parasiticide in accordance with the manufacturer's recommendations. For dogs and cats visiting tick endemic regions, tick prevention should be practised. Owners should search animals daily for ticks, and physically remove and dispose of any ticks detected.

### **4.7.3 Current biosecurity measures**

There are no specific biosecurity measures for hepatozoonosis. Current import conditions for external parasites have the following requirements.

#### **Vector management**

- At the time of blood sampling for ehrlichiosis, dogs must be treated with an acaricide effective against ticks on contact; the acaricide must be applied according to the manufacturer's instructions by a government-approved veterinarian.
- Within five days immediately before export, dogs and cats must be treated with an acaricide effective against ticks on contact; the acaricide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within five days immediately before export, dogs and cats must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks.

#### 4.7.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by hepatozoonosis:

- Hepatozoonosis is not an OIE-listed disease and is not a nationally notifiable disease in Australia.
- Canine hepatozoonosis is exotic to Australia.
- Dogs are definitive hosts for *H. canis* and *H. americanum*.
- *H. canis* infection is typically subclinical or results in mild disease. *H. americanum* produces severe disease that is frequently fatal.
- *R. sanguineus*, the tick vector for *H. canis*, is present in Australia. *A. maculatum*, the principal tick vector for *H. americanum* is not known to be present in Australia.
- Prevention of exposure to infective ticks is the best method of prevention of hepatozoonosis.
- Treatment cannot be relied upon to eliminate infection with either *H. canis* or *H. americanum*.

#### 4.7.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for hepatozoonosis are warranted for dogs. In addition, it was concluded that risk management measures for hepatozoonosis are not warranted for cats, or for dog or cat semen.

The following measures would provide appropriate risk management.

##### Pre-export measures (dogs)

- As for pre-export measures for external parasite control (see 4.13.5).

##### Post-arrival measures (dogs)

- As for post-arrival measures for external parasite control (see 4.13.5).
- To manage the biosecurity risks of hepatozoonosis, dogs may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### References

Allen KE, Li Y, Kaltenboeck B, Johnson EM, Reichard MV, Panciera RJ, Little SE (2008). Diversity of Hepatozoon species in naturally infected dogs in the southern United States. *Veterinary Parasitology* 154:220–225.

Baneth G (2002). Hepatozoonosis. In *Arthropd-borne diseases*, Coles G (ed.), Quedgeley, England.

- Baneth G (2006). Hepatozoonosis: Hepatozoon canis infection. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 698–705.
- Baneth G (2011). Perspectives on canine and feline hepatozoonosis. *Veterinary Parasitology* 181:3–11.
- Baneth G, Mathew S, Shkap V, Macintyre DK, Barta JR, Ewing SA (2003). Canine hepatozoonosis: two disease syndromes caused by separate *Hepatozoon* spp. *Trends in Parasitology* 19:27–31.
- Baneth G, Samish M, Alekseev E, Aroch I, Shkap V (2001). Transmission of *Hepatozoon canis* to dogs by naturally fed or percutaneously injected *Rhipicephalus sanguineus* ticks. *The Journal of Parasitology* 87:606–611.
- Criado-Fornelio A, Buling A, Cunha-Filho NA, Ruas JL, Farias NAR, Rey-Valeiron C, Pingret JL, Etievant M, Barba-Carretero JC (2007). Development and evaluation of a quantitative PCR assay for detection of *Hepatozoon* sp. *Veterinary Parasitology* 150:352–356.
- Cummings CA, Panciera RJ, Kocan KM, Mathew JS, Ewing SA (2005). Characterisation of stages of *Hepatozoon americanum* and of parasitised canine host cells. *Veterinary Pathology* 42:788–796.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Ewing SA, DuBois JG, Mathew JS, Panciera RJ (2002). Larval Gulf Coast ticks (*Amblyomma maculatum*) [Acari: Ixodidae] as host for *Hepatozoon americanum* [Apicomplexa: Adeleorina]. *Veterinary Parasitology* 103:43–51.
- Ewing SA, Panciera RJ (2003). American canine hepatozoonosis. *Clinical Microbiology Reviews* 16:688–697.
- Forlano M, Scofield A, Elisei C, Fernandes KR, Ewing SA, Massard CL (2005). Diagnosis of *Hepatozoon* spp. in *Amblyomma ovale* and its experimental transmission in domestic dogs in Brazil. *Veterinary Parasitology* 134:1–7.
- Jittapalapong S, Rungphisutthipongse O, Maruyama S, Schaefer JJ, Stich RW (2006). Detection of *Hepatozoon canis* in stray dogs and cats in Bangkok, Thailand. *Annals of the New York Academy of Sciences* 1081:479–488.
- Johnson EM, Panciera RJ, Allen KE, Sheets ME, Beal JD, Ewing SA, Little SE (2009). Alternate pathway of infection with *Hepatozoon americanum* and the epidemiologic importance of predation. *Journal of Veterinary Internal Medicine* 23:1315–1318.

Li Y, Wang C, Allen KE, Little SE, Ahluwalia SK, Gao D, Macintire DK, Blagburn BL, Kaltenboeck B (2008). Diagnosis of canine *Hepatozoon* spp. infection by quantitative PCR. *Veterinary Parasitology* 157:50–58.

Macintire DK, Vincent-Johnson N, Dillon AR, Blagburn B, Lindsay D, Whitley EM, Banfield C (1997). Hepatozoonosis in dogs: 22 cases (1989–1994). *Journal of the American Veterinary Medical Association* 210:916–922.

Macintire DK, Vincent-Johnson NA, Kane CW, Lindsay DS, Blagburn BL, Dillon AR (2001). Treatment of dogs infected with *Hepatozoon americanum*: 53 cases (1989–1998). *Journal of the American Veterinary Medical Association* 218:77–82.

Macintyre DK, Vincent-Johnson NA, Craig TM (2006). Hepatozoonosis: *Hepatozoon americanum* infection. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders, Elsevier, St. Louis, pp. 705–711.

Mathew JS, Saliki JT, Ewing SA, Lehenbauer TW, Panciera RJ, Malayer JR, Cummings CA, Kocan AA (2001). An indirect enzyme-linked immunosorbent assay for diagnosis of American canine hepatozoonosis. *Journal of Veterinary Diagnostic Investigation* 13:17–21.

Murata T, Inoue M, Tateyama S, Taura Y, Nakama S (1993). Vertical transmission of *Hepatozoon canis* in dogs. *Journal of Veterinary Medical Science* 55:867–868.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Panciera RJ, Ewing SA, Mathew JS, Lehenbauer TW, Cummings CA, Woods JP (1999). Canine hepatozoonosis: comparison of lesions and parasites in skeletal muscle of dogs experimentally or naturally infected with *Hepatozoon americanum*. *Veterinary Parasitology* 82:261–272.

Potter TM, Macintire DK (2010). *Hepatozoon americanum*: an emerging disease in the south-central/southeastern United States. *Journal of Veterinary Emergency and Critical Care* 20:70–76.

Rubini AS, dos Santos Paduan K, Perez RR, Ribolla PEM, O'Dwyer LH (2006). Molecular characterization of feline *Hepatozoon* species from Brazil. *Veterinary Parasitology* 137:168–171.

Sasanelli M, Paradies P, Greco B, Eyal O, Zaza V, Baneth G (2010). Failure of imidocarb dipropionate to eliminate *Hepatozoon canis* in naturally infected dogs based on parasitological and molecular evaluation methods. *Veterinary Parasitology* 171:194–199.

## 4.8 Leishmaniasis

### 4.8.1 Background

*Leishmania* spp. are intracellular trypanosomatid parasites that cause a spectrum of clinical diseases, all termed 'leishmaniasis'<sup>9</sup>. Significant disease is seen in dogs, humans and some species of rodents. The clinical presentation varies from focal cutaneous disease to disseminated visceral disease, and the severity varies from subclinical to fatal.

Leishmaniasis is caused by numerous different *Leishmania* spp. In the New World<sup>10</sup>, leishmaniasis is caused by *L. braziliensis* complex, *L. mexicana* complex, *L. peruviana* and *L. chagasi*, the latter widely accepted to be a synonym of *L. infantum*. In the Old World, leishmaniasis is caused by *L. donovani*, *L. infantum*, *L. tropica*, *L. major* and *L. aethiopica*. The disease agents are zoonotic with a few exceptions (Gramiccia and Gradoni 2005). Canine leishmaniasis (CanL) is a chronic viscerocutaneous disease caused by *L. infantum*, of which the dog acts as the reservoir host. In some instances, *L. braziliensis* complex, *L. major* and *L. tropica* species have been isolated from canids (Dantas-Torres 2007).

Leishmaniasis in humans and dogs is widespread throughout Africa, parts of Asia, southern Europe, and in South and Central America. Major epidemics occur in the Middle East and South America. Endemic foci are found in the Mediterranean basin countries and Africa, as well as India, parts of China and other areas of Asia (CFSPH 2009).

Leishmaniasis is an OIE-listed disease (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011). There is a chapter in the *Manual of diagnostic tests and vaccines* relating to the disease, (OIE 2008) but there are no recommendations in the *Terrestrial animal health code* (OIE 2011b) for the importation of animals with respect to leishmaniasis.

Biosecurity Australia reviewed leishmaniasis (*Biosecurity Australia Policy Memorandum 2006/04*) following the detection of a novel *Leishmania* species in Australian wildlife in 2001 (Rose et al. 2004).

The review found that:

- infected dogs can appear healthy and not be detected during pre-export and post-arrival examinations, and these dogs can be infective to vectors
- there was an increasing incidence of leishmaniasis and significant expansion of the endemic range overseas
- there was uncertainty due to the unknown vector competence of Australian insects and the distribution of potential vectors

<sup>9</sup> The OIE uses the term 'leishmaniosis'. Most of the scientific literature refers to it as leishmaniasis, and this is the term used in this policy review.

<sup>10</sup> New World refers to the Americas; Old World refers to Africa, Asia and Europe.

Based on these findings of the 2006 review, biosecurity measures were developed for *L. infantum*.

#### 4.8.2 Technical information

##### Epidemiology

Disease caused by *Leishmania* spp. is most common in humans and dogs. The zoonotic potential of different *Leishmania* spp. varies. Dogs infected by *L. infantum* and *L. braziliensis* are considered sources of human visceral and cutaneous leishmaniasis, respectively (Dantas-Torres 2007). Throughout the world, rodents, small mammals and canids are common reservoirs of *Leishmania* infection in endemic countries. Domestic dogs also play a significant role in transmission in the urban environment (OIE 2008). *L. infantum* is the most common species reported in domestic animals; the reported incubation period in dogs varies from three months to seven years (CFSPH 2009). The distinction between species responsible for visceral and cutaneous disease observed in humans is not observed in animals (CFSPH 2009).

In 2001 a novel *Leishmania* spp. was isolated from the skin lesions of a group of captive red kangaroos (*Macropus rufus*) in the Northern Territory (Rose et al. 2004). Leishmaniasis has since been observed in eight captive northern wallaroos (*M. robustus woodwardi*), one black wallaroo (*M. bernardus*) and two agile wallabies (*M. agilis agilis*) (Dougall et al. 2009).

It is widely accepted that phlebotomine sand flies are vectors of *Leishmania* spp. (Saridomichelakis 2009). However, other vectors such as ticks and fleas may also play a role in transmission (Coutinho et al. 2005). In Australia, there is some evidence that the biting midge *Forcipomyia* (*Lasiohelea*) spp. – possibly *F. (L.) peregrinator* – may be transmitting the *Leishmania* species detected in macropods in the Northern Territory (Dougall et al. 2011).

There is evidence of genital lesions and shedding of *Leishmania* species in the semen of dogs with visceral leishmaniasis (Diniz et al. 2005). There is also evidence of sexual transmission of *L. chagasi* from naturally infected, serologically positive dogs to susceptible bitches (Pedersoli et al. 2010; Silva et al. 2009) – indicating that semen may be another source of infection. It is unclear from the scientific literature whether infection can be transmitted via semen from subclinically infected dogs.

Epidemiological studies using molecular diagnostic techniques in areas where leishmaniasis is endemic have shown that the prevalence of canine *Leishmania* spp. infection may be considerably higher than indicated by estimates of seroprevalence or the prevalence of clinical disease (Baneth et al. 2008). *L. infantum* infection in dogs is endemic in approximately 50 countries across Africa, the Americas, Asia and Europe. Infection prevalence varies depending on ecological and climatic conditions that determine the abundance of vectors (Solano-Gallego et al. 2009). Infection with *L. infantum* is endemic in countries of the Mediterranean basin; an increasing incidence of clinical leishmaniasis in humans was reported in Spain between 2009 and 2012 (Promed Mail 2012). In North America, limited foci of infection have been

reported in dog populations in Canada and the United States with leishmaniasis caused by *L. infantum* occurring mainly in foxhounds (CFSPH 2009).

Cats are susceptible to infection but are considered an unusual host for *L. infantum*. Visceral leishmaniasis is rare in cats while feline cutaneous leishmaniasis has been documented in numerous countries (Nasereddin et al. 2008). Further investigations are required to evaluate the role of cats as secondary hosts.

### **Clinical signs**

Animals are often infected subclinically with *Leishmania* spp. The clinical features of leishmaniasis vary widely due to the numerous pathogenic mechanisms of the disease process, the different organs affected and the diversity of immune responses. The main clinical findings of classical CanL include skin lesions, generalised lymphadenomegaly, progressive weight loss, decreased appetite, lethargy, polyuria and polydypsia, ocular lesions, epistaxis, onychogryphosis, lameness, vomiting and diarrhoea. Disease is usually fatal if left untreated (Solano-Gallego et al. 2009).

Feline cutaneous leishmaniasis typically presents as alopecia, with nodular crusty lesions of the nose, ears, lips and eyelids (Nasereddin et al. 2008).

### **Diagnosis**

The diagnosis of leishmaniasis is often difficult because of the variability of clinical presentation. In animals with evident clinical signs, detection of amastigotes by microscopic examination of stained smears from skin lesions, bone marrow smears or aspirates from enlarged lymph nodes provides the simplest method of diagnosis. The sensitivity of detection via microscopy is poor—ranging from about 30% (lymph nodes) to 60% (bone marrow) of infections (Miró et al. 2008).

Serological testing is the preferred method for diagnosis of CanL, even during the early stages of the disease (OIE 2008). An indirect fluorescent antibody test (IFAT) is widely used. The IFAT for *L. infantum* has a sensitivity of 96% and specificity of 98%, similar to the enzyme-linked immunosorbent assay (ELISA). However, due to cross-reactivity with *Trypanosoma cruzi* (the cause of Chagas' disease), the IFAT is less reliable in Chagas' disease-affected regions. The ELISA is useful for the diagnosis of both Old World and New World leishmaniases. There is little or no cross-reaction with other diseases and, according to the *Leishmania* spp. strain used, sensitivity can range from 86% to 99% (OIE 2011a).

Polymerase chain reaction (PCR) testing of lymph nodes and bone marrow is highly sensitive for diagnosis of early stage infection with *Leishmania* spp., but sampling from these sites is considered too invasive for routine import screening of dogs. Although PCR testing performed on blood is not considered to be highly sensitive, it increases the overall sensitivity of *Leishmania* spp. testing when used in conjunction with serology, especially if real-time PCR is used (OIE 2011a).

## Treatment

Several drugs used for therapy of the disease, including meglumine antimoniate and allopurinol, are able to improve clinical signs temporarily or cure dogs clinically, but none of these treatments reliably eliminates the infection (Miró et al. 2008).

### 4.8.3 Current biosecurity measures

Within 30 days before export, a blood sample must be obtained from the dog by a government-approved veterinarian for testing for serological evidence of *L. infantum* infection by IFAT or ELISA. The test result must be negative.<sup>11</sup>

### 4.8.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by leishmaniasis:

- Leishmaniasis is an OIE-listed disease and is a nationally notifiable disease in Australia.
- Leishmaniasis due to *L. infantum* is endemic in many countries and causes severe viscerocutaneous disease in dogs and humans. Disease is usually fatal if left untreated.
- Dogs and humans are a recognised reservoir host of *L. infantum*. Cats are susceptible to infection but considered an unusual host.
- *Leishmania* spp. have been isolated from the skin lesions of a group of captive red kangaroos (*Macropus rufus*) in Australia.
- There is evidence that biting midges can transmit Australian *Leishmania* spp. in the Northern Territory. It remains uncertain whether a competent vector capable of transmitting *L. infantum* is present in Australia.
- Transmission via semen is reported to occur in clinically affected dogs. It is not known whether the disease agent is transmitted in semen of dogs with subclinical infection.
- Treatment does not eliminate infection.

### 4.8.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for leishmaniasis caused by *L. infantum* continue to be warranted for dogs. It was also concluded that risk management for *L. infantum* is warranted for dog semen. In addition, it was concluded that risk management measures for leishmaniasis are not warranted for cats or their semen.

The current biosecurity measure of pre-export serology with a negative result<sup>12</sup> would provide appropriate risk management for dogs.

---

<sup>11</sup> Dogs continuously resident in New Zealand, Norfolk Island and/or Cocos (Keeling) Islands since either birth, or importation from Australia (whichever is applicable) do not require testing.

For dog semen, the following measures would provide appropriate risk management:

#### Pre-export measures (dog semen)

*Donor dogs—serology:*

- Between 30 to 45 days following the last collection of semen in the consignment for export, a blood sample must be collected from the donor dog and tested for serological evidence of *L. infantum* infection by IFAT or ELISA. The test must produce a negative result.

#### References

Baneth G, Koutinas AF, Solano-Gallego L, Bourdeau P, Ferrer L (2008). Canine leishmaniosis—new concepts and insights on an expanding zoonosis: part one. *Trends in Parasitology* 24:324–330.

CFSPH (Center for Food Security and Public Health) (2009). Leishmaniasis (cutaneous and visceral). CFSPH, Iowa State University, Ames. <http://www.cfsph.iastate.edu/Factsheets/pdfs/leishmaniasis.pdf> (accessed 15 April 2011)

Coutinho MTZ, Bueno LL, Sterzik A, Fujiwara RT, Botelho JR, De Maria M, Genaro O, Linardi PM (2005). Participation of *Rhipicephalus sanguineus* (Acari: Ixodidae) in the epidemiology of canine visceral leishmaniasis. *Veterinary Parasitology* 128:149–155.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Dantas-Torres F (2007). The role of dogs as reservoirs of *Leishmania* parasites, with emphasis on *Leishmania (Leishmania) infantum* and *Leishmania (Viannia) braziliensis*. *Veterinary Parasitology* 149:139–146.

Diniz SA, Melo MS, Borges AM, Bueno R, Reis BP, Tafuri WL, Nascimento EF, Santos RL (2005). Genital lesions associated with visceral leishmaniasis and shedding of *Leishmania* sp. in the semen of naturally infected dogs. *Veterinary Pathology* 42:650–658.

Dougall A, Shilton C, Low Choy J, Alexander B, Walton S (2009). New reports of Australian cutaneous leishmaniasis in Northern Australian macropods. *Epidemiology and Infection* 137:1516–1520.

Dougall AM, Alexander B, Holt D, Harris T, Sultan AH, Bates PA, Rose K, Walton SF (2011). Evidence incriminating midges (Diptera: Ceratopogonidae) as potential vectors of *Leishmania* in Australia. *International Journal for Parasitology* 41:271–579.

---

<sup>12</sup> Dogs continuously resident in New Zealand, Norfolk Island and/or Cocos (Keeling) Islands since either birth, or importation from Australia (whichever is applicable) do not require testing.

- Gramiccia M, Gradoni L (2005). The current status of zoonotic leishmaniases and approaches to disease control. *International Journal for Parasitology* 35:1169–1180.
- Miró G, Cardoso L, Pennisi MG, Oliva G, Baneth G (2008). Canine leishmaniosis—new concepts and insights on an expanding zoonosis: part two. *Trends in Parasitology* 24:371–377.
- Nasereddin A, Salant H, Abdeen Z (2008). Feline leishmaniasis in Jerusalem: serological investigation. *Veterinary Parasitology* 158:364–369.
- OIE (World Organisation for Animal Health) (2008). Leishmaniosis. In *Manual of diagnostic tests and vaccines for terrestrial animals 2011*. OIE, Paris.  
[http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.01.08\\_LEISHMANIOSIS.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.08_LEISHMANIOSIS.pdf) (accessed 08 May 2012)
- OIE (World Organisation for Animal Health) (2011b). *Terrestrial animal health code*. OIE, Paris.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Pedersoli AV, Ribeiro VM, Rachid MA, Castro ACS, Valle GR (2010). Dogs with *Leishmania chagasi* infection have semen abnormalities that partially revert during 150 days of Allopurinol and Amphotericin B therapy. *Animal Reproduction Science* 117:183–186.
- Promed Mail (2012). Leishmaniasis—Spain, Madrid. ProMED Mail.  
<http://www.promedmail.org> (accessed 9 November 2012).
- Rose K, Curtis J, Baldwin T, Mathis A, Kumar B, Sakthianandeswaren A, Spurck T, Low Choy J, Handman E (2004). Cutaneous leishmaniasis in red kangaroos: isolation and characterisation of the causative organisms. *International Journal for Parasitology* 34:655–664.
- Saridomichelakis MN (2009). Advances in the pathogenesis of canine leishmaniosis: epidemiologic and diagnostic implications. *Veterinary Dermatology* 20:471–489.
- Silva FL, Oliveira RG, Silva TMA, Xavier MN, Nascimento EF, Santos RL (2009). Venereal transmission of canine visceral leishmaniasis. *Veterinary Parasitology* 160:55–59.
- Solano-Gallego L, Koutinas A, Miró G, Cardoso L, Pennisi MG, Ferrer L, Bourdeau P, Oliva G, Baneth G (2009). Directions for the diagnosis, clinical staging, treatment and prevention of canine leishmaniosis. *Veterinary Parasitology* 165:1–18.

## 4.9 Leptospirosis

### 4.9.1 Background

Leptospirosis is an infectious bacterial disease of mammals, including humans, due to serovars of *Leptospira interrogans*. Dogs are the only species recognised as the maintenance host for *L. interrogans* sv. *Canicola*, which has a worldwide distribution, but is considered exotic to Australia and New Zealand. Exposure to some serovars of *L. interrogans* normally maintained by other host animals—for example, Icterohaemorrhagiae, Grippotyphosa and Bratislava—can produce clinical disease and a carrier state in dogs (Ellis 2010).

Leptospirosis is not an OIE-listed disease (OIE 2012) and is not nationally notifiable in animals in Australia (DAFF 2011), but clinical disease in humans is nationally notifiable (Communicable Diseases Network Australia 2011).

Leptospiral disease was briefly reviewed by the Bureau of Resource Sciences (BRS) in its *Review of quarantine policy for dogs and cats, with particular reference to rabies* (BRS 1993). The review:

- noted that *L. Canicola* is the serovar of concern and is exotic to Australia
- recommended that pre-export testing and treatment requirements against canine leptospirosis be limited to *L. Canicola*
- proposed that dogs must test negative (less than 50% agglutination at a serum dilution of 1:100) to a single serum agglutination test within 30 days of export.

Accordingly, import conditions based on the BRS review were developed for *L. Canicola*.

### 4.9.2 Technical information

#### Epidemiology

Leptospirosis is not a highly infectious disease. Direct or indirect contact with infected urine is a common mode of transmission. Common sources of exposure include contaminated surface water, mud and soil (Faine 1998). Exposure to risk factors such as high rainfall in the warmer time of year (late summer/autumn) may lead to outbreaks (Ward 2002). Studies examining the zoonotic risk of *L. Canicola* have concluded that the bacteria have low infectivity for humans (McIntyre and Seiler 1953). Human cases of *L. Canicola* have occurred where human association with dogs could not be established (Lawson and Michna 1966).

Infection typically occurs directly through mucous membranes or through abraded or water-softened skin. Leptospire appear in blood four to ten hours after infection, remaining detectable from only a few hours to seven days. Clinical signs are not always evident, but pyrexia is typical with acute leptospirosis (Greene et al. 2006).

Recovered animals can become subclinical carriers (maintenance hosts) in which leptospire multiply in renal tubules for periods of days to years and are excreted

intermittently in the urine (Faine et al. 1994). There is no relationship between the severity of the infection and the subsequent development of a carrier status. (Rojas et al. 2010).

*L. Canicola* has not been isolated from dogs in Australia, but seroreactivity to leptospira in the *Canicola* serogroup was detected in coastal areas of North Queensland, with rainforest animals such as rats and bandicoots believed to be the main carriers (Queensland Government 2008).

Transmission can occur during breeding through infectious semen or infectious urine residual in the genito-urinary tract (Faine et al. 1999; Hudson 1978).

### **Clinical signs**

Clinical signs depend on the organs affected. Kidneys are generally the target organ for *L. Canicola*, with oliguria, anuria or polyuria, and chronic renal failure a common sequel to infection. This contrasts with *L. Grippotyphosa*, another serovar that can infect dogs but generally targets the liver, resulting in hepatic disease (Sessions and Greene 2004a). Age at infection may influence clinical presentation as clinical signs observed in 2–4 month-old dogs challenged with *L. Canicola* and *L. Icterohaemorrhagiae* were similar, and included depression, anorexia, haemorrhagic diarrhoea, vomiting, icterus and haematuria (Minke et al. 2009).

### **Diagnosis**

Numerous serological tests are available, including the microscopic agglutination test (MAT), indirect fluorescent antibody test (IFAT), and enzyme-linked immunosorbent assay (ELISA).

The sensitivity and specificity of the standard MAT can vary widely between laboratories due to the subjective nature of test interpretation. The level of exposure, the immune status of an exposed animal and vaccination are other key factors that can influence an animal's immune response (Adler and de la Peña Moctezuma 2010; Bolin 1996; Burr et al. 2009; Gautam et al. 2010).

Despite its limitations, the MAT remains the most frequently used test for diagnosing canine leptospirosis. MAT titres are usually negative for the first seven to ten days of infection and the level of antibody response may be reduced in dogs that have received antibiotic treatment (Greene et al. 2006).

A MAT result from a single blood specimen is of limited value for determining the disease status of an animal, including infective carriers (André-Fontaine 2006). Where two blood specimens are collected at least two weeks apart, the MAT may be useful in diagnosing acute infection, through detection of seroconversion and/or a significant increase in circulating antibodies (Greene et al. 2006).

The ELISA is of value for detection of early leptospiral infections and has the advantage of distinguishing between infection and vaccine-induced immunity (Greene et al. 2006).

Identification of leptospires is by culture, microscopy, fluorescent antibody or polymerase chain reaction (PCR) techniques. Culture of leptospires is both difficult and time consuming, and few laboratories can provide this service. Microscopy has poor sensitivity and specificity. PCR can be used to identify leptospiral shedding in urine (Harkin et al. 2003), but is not readily available.

### Treatment

Leptospirosis can be treated with antibiotics and supportive therapy (Sessions and Greene 2004b). Penicillins are the treatment of choice for clinical disease. Leptospires that are located intracellularly may evade the effects of antibiotics so a prolonged course of treatment is recommended to eliminate a carrier state. Treatment with tetracyclines is recommended to eliminate the carrier state in dogs—doxycycline administered at a dose rate of 5mg/kg twice daily for 14 days is the recommended treatment regimen (Greene et al. 2006).

### Vaccination

Vaccines for dogs are generally suspensions of one or more serovars of *Leptospira* spp. inactivated such that immunogenic activity is retained. The protection provided by the current vaccines is restricted to the serogroups used for their production (André-Fontaine 2006). Vaccines containing *L. Canicola* and administered according to manufacturer's recommendations can provide immune protection that lasts at least 12 months against both clinical leptospirosis and the renal carrier stage (Gueguen et al. 2000; Klaasen et al. 2003; Minke et al. 2009). However, efficacy of vaccination has been estimated at about 70% and regular annual vaccination is required to maintain immunity. Vaccination is not recommended in toy breeds due to a reported increased risk of adverse reaction (Day et al. 2010).

There is a lack of association between vaccine-induced immunity and MAT titres against leptospirosis. The MAT response following vaccination is usually transient—lasting up to 4–5 months (Gueguen et al. 2000; Klaasen et al. 2003). Post-vaccinal MAT titres are usually low (1:100 to 1:400), although high titres (1:3200) have been reported (Greene et al. 2006).

While vaccination does not provide a sterile immunity, studies have shown that it considerably mitigates the risk that dogs will shed leptospires in their urine as well as the risk of animals becoming subclinical carriers (André-Fontaine et al. 2003). The efficacy of prophylaxis against infection appears to be dependent on the vaccine used and the serovar of *Leptospira* to which the dog is exposed (André-Fontaine et al. 2003; Klaasen et al. 2003; Schreiber et al. 2005). Vaccination is considered useful in decreasing zoonotic risk (Klaasen et al. 2003).

#### 4.9.3 Current biosecurity measures

##### *Dogs*

- Within 30 days before export, dogs must be tested for evidence of *L. interrogans* sv. *Canicola* infection using a MAT. Dogs that record a negative result (less than 50% agglutination at a serum dilution of 1:100) are eligible for export to Australia.

For vaccinated dogs that record a result of positive at 1:100 or more, but negative at 1:800, must be retested 14 days or more after the first date of sampling. To be eligible for export to Australia, the second test must also show a negative result at 1:800.

Dogs that record a result positive at 1:800 or more are ineligible for export to Australia.

#### *Semen donors*

Within 30 days before collection of semen for export:

- The donor must be tested for evidence of *L. Canicola* infection using a MAT. Semen collected from donors that record a negative result (less than 50% agglutination at a serum dilution of 1:100) is eligible for export to Australia.

OR

- For vaccinated donors with a positive MAT result at 1:400 (or less) for *L. Canicola*, a blood sample must be collected from the dog between 14 and 30 days after the collection of the first blood sample and be subjected to a MAT that shows no increase above the titre of the first MAT.

Donor dogs must not be naturally mated between blood collection and the last collection of semen for export to Australia.

#### **4.9.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by leptospirosis:

- Due to the widespread international distribution of leptospires, leptospirosis was removed from the OIE list of diseases in 2012. Leptospirosis is not a nationally notifiable animal disease in Australia.
- Leptospirosis is not a highly infectious disease.
- *Canicola* serovars occur in Australia. It is not certain that *L. Canicola* is exotic, although it has not been detected in dogs in Australia.
- Dogs are the recognised maintenance host for *L. Canicola*. Severe disease due to *L. Canicola*, including fatal infection, is more likely to occur in young dogs.
- There are a number of endemic leptospiral serovars in Australia that cause severe and sometimes fatal infection in dogs.
- Vaccination against *L. Canicola* is effective in preventing clinical disease, but does not provide a sterile immunity. Vaccination mitigates the risk of dogs establishing a carrier status and shedding *L. Canicola*.
- Vaccination of toy breeds against *L. Canicola* is not recommended, because these breeds are reported to be at an increased risk of adverse vaccination reactions.

- Treatment of dogs with doxycycline for 14 days at a dose rate of 5 mg/kg is recommended to eliminate latent infection (carrier status) with *L. Canicola*.
- Serology is a useful but not highly sensitive diagnostic tool for determining infection status. Dogs that are not vaccinated against *L. Canicola* and return negative serology results can carry and shed *L. Canicola*.
- *L. Canicola* can be transmitted via the semen of infected dogs.

#### 4.9.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures continue to be warranted for leptospirosis caused by *L. Canicola*. It was also concluded that risk management measures for *L. Canicola* are warranted for dog semen.

The following biosecurity measures would provide appropriate risk management.

##### **Pre-export measures (dogs)**

###### *Vaccination*

- Dogs must be fully vaccinated with an approved vaccine against *L. Canicola* in accordance with the manufacturer's recommendations.
- The annual revaccination or final vaccination of the initial course must be administered between 12 months and 14 days before export to Australia.

OR

###### *Serology*

- Within 45 days of export, dogs must be tested for *L. Canicola* infection by the MAT and have a negative result.
- A negative result is defined as less than 50% agglutination at a serum dilution of 1:100.

OR

###### *Treatment*

- Within 45 days of export, dogs must be treated with doxycycline at a therapeutic dose rate of at least 5 mg/kg twice daily for 14 consecutive days.

##### **Pre-export measures (dog semen)**

###### *Donor dogs—vaccination*

- Donor dogs must be fully vaccinated with an approved vaccine against *L. Canicola* in accordance with the manufacturer's recommendations.
- Vaccination status must be current, at the time of each collection of semen for export to Australia.

OR

Donor dogs—serology (single sample)

- Between 30 and 45 days following the final collection of semen in the consignment for export, a blood sample must be collected from the donor dog and tested by MAT for *L. Canicola* with a negative result. A negative result is defined as less than 50% agglutination at a serum dilution of 1:100.

## References

- Adler B, de la Peña Moctezuma A (2010). *Leptospira* and leptospirosis. *Veterinary Microbiology* 140:287–296.
- Alt DP, Bolin CA (1996). Preliminary evaluation of antimicrobial agents for treatment of *Leptospira interrogans* serovar Pomona in hamsters and swine. *American Journal of Veterinary Research* 57:59–62.
- André-Fontaine G (2006). Canine leptospirosis: Do we have a problem? *Veterinary Microbiology* 117:19–24.
- André-Fontaine G, Branger C, Gray AW, Klaasen HLBM (2003). Comparison of the efficacy of three commercial bacterins in preventing canine leptospirosis. *The Veterinary Record* 153:165–169.
- Barocchi MA, Ko AI, Reis MG, McDonald KL, Riley LW (2002). Rapid translocation of polarized MDCK cell monolayers by *Leptospira interrogans*, an invasive but nonintracellular pathogen. *Infection and Immunity* 70:6926–6932.
- Bolin CA (1996). Diagnosis of leptospirosis: a reemerging disease of companion animals. *Seminars in Veterinary Medicine and Surgery (Small Animal)* 11:166–171.
- BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper. BRS, Canberra.
- Burr P, Lunn K, Yam P (2009). Current perspectives on canine leptospirosis. *In Practice* 31:98–102.
- Communicable Diseases Network Australia (2011). Australian national notifiable diseases case definitions. Australian Government Department of Health and Ageing, Canberra.  
<http://www.health.gov.au/internet/main/publishing.nsf/content/cdna-casedefinitions.htm#dislist> (accessed 29 April 2011)
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra,  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Day MJ, Horzinek MC, Schultz RD (2010). WSAVA guidelines for the vaccination of dogs and cats. *Journal of Small Animal Practice* 51:e1–e32.

- Ellis WA (2010). Control of canine leptospirosis in Europe: time for a change? *Veterinary Record* 167:602–605.
- Faine S (1998). Leptospirosis. In *Topley and Wilson's microbiology and microbial infections*, 9th edn, Collier L, Balows A, Sussman M (eds), Arnold, London, pp. 849–869.
- Faine S, Adler BB, Bolin C, Perolat P (1994). A brief overview of the disease, leptospirosis. In *Leptospira and Leptospirosis*, 1st edn, MediSci Writing, Melbourne, pp. 145–152.
- Faine S, Adler B, Bolin C, Perolat P (1999). Sources, transmission and spread of leptospirosis. In *Leptospira and Leptospirosis*, 2nd edn, MediSci Writing, Melbourne, p. 132.
- Gautam R, Wu C-C, Guptill LF, Potter A, Moore GE (2010). Detection of antibodies against *Leptospira* serovars via microscopic agglutination tests in dogs in the United States, 2000–2007. *Journal of the American Veterinary Medical Association* 237:293–298.
- Greene CE, Sykes JE, Brown CA, Hartmann K (2006). Leptospirosis. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 402–417.
- Gueguen S, Mähl P, Martin V, Lecoutre C, Aubert A (2000). Duration of immunity and clinical protection against canine leptospirosis with a multivalent vaccine. In Anclivepa Congress, Rio de Janeiro, Brazil, 9 June 2000, pp. 1–16.
- Harkin KR, Roshto YM, Sullivan JT, Purvis TJ, Chengappa MM (2003). Comparison of polymerase chain reaction assay, bacteriologic culture, and serologic testing in assessment of prevalence of urinary shedding of leptospires in dogs. *Journal of the American Veterinary Medical Association* 222:1230–1233.
- Hudson DB (1978). G78-417 Leptospirosis of domestic animals. Historical Materials from University of Nebraska-Lincoln Extension. <http://digitalcommons.unl.edu/extensionhist/227> (accessed 15 March 2012)
- Klaasen HLBM, Molkenboer MJCH, Vrijenhoek MP, Kaashoek MJ (2003). Duration of immunity in dogs vaccinated against leptospirosis with a bivalent inactivated vaccine. *Veterinary Microbiology* 95:121–132.
- Lawson JH, Michna SW (1966). Canicola fever in man and animals. *British Medical Journal* 2:336–340.
- McIntyre WIM, Seiler HE (1953). Epidemiology of Canicola fever. *Journal of Hygiene* 51:330–339.
- Minke JM, Bey R, Tronel JP, Latour S, Colombet G, Yvorel J, Cariou C, Guiot AL, Cozette V, Guigal PM (2009). Onset and duration of protective immunity against

clinical disease and renal carriage in dogs provided by a bivalent inactivated leptospirosis vaccine. *Veterinary Microbiology* 137:137–145.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Queensland Government (2008). *Leptospira serovar* data sheet: serovar canicola. WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis. <http://www.health.qld.gov.au/> (accessed 2008)

Rojas P, Monahan A, Schuller S, Miller I, Markey B, Nally J (2010). Detection and quantification of leptospire in urine of dogs: a maintenance host for the zoonotic disease leptospirosis. *European Journal of Clinical Microbiology and Infectious Diseases* 29:1305–1309.

Schreiber P, Martin V, Najbar W, Sanquer A, Gueguen S, Lebreux B (2005). Prevention of renal infection and urinary shedding in dogs by a *Leptospira* vaccination. *Veterinary Microbiology* 108:113–118.

Sessions JK, Greene CE (2004a). Canine leptospirosis: epidemiology, pathogenesis, and diagnosis. *Compendium on Continuing Education for the Practicing Veterinarian* 26:606–622.

Sessions JK, Greene CE (2004b). Canine leptospirosis: treatment, prevention, and zoonosis. *Compendium on Continuing Education for the Practicing Veterinarian* 26:700–706.

Ward MP (2002). Seasonality of canine leptospirosis in the United States and Canada and its association with rainfall. *Preventive Veterinary Medicine* 56:203–213.

## 4.10 Lyme disease

### 4.10.1 Background

Lyme disease is an emerging zoonotic disease caused by the tick-borne spirochaete *Borrelia burgdorferi* sensu lato (*B. burgdorferi* s.l.) (Burgdorfer et al. 1982; Ogden et al. 2010). Within the species *B. burgdorferi* s.l., there are several genospecies that vary in geographical distribution, host and vector preferences, tissue tropism, pathogenicity and clinical presentation (Burgdorfer 1991; Kawabata et al. 1993; Ryffel et al. 1999; Wang et al. 1999).

Infection with *B. burgdorferi* s.l. has been reported in humans (Parker and White 1992), cats (Magnarelli et al. 1990), dogs, deer (Madigan and Teitler 1988), cattle (Burgess et al. 1993), sheep (Fridriksdottir et al. 1992; Ogden et al. 1997), horses (Divers 2007; Imai et al. 2011) and zoo animals (Stoebel et al. 2003). Infected birds, rabbits, sheep and wildlife serve as reservoirs for tick infection and introduce the disease to a wider geographical area (Anderson et al. 1985; Brinkerhoff et al. 2009;

Greene et al. 1998; Lane and Regnery 1989; Ogden et al. 1997; Olsen et al. 1993; Olsen et al. 1995; Telford and Spielman 1989).

During the 1980s, the disease incidence in both dogs and humans increased significantly; in 2004, Lyme disease was the most commonly reported tick-borne disease in humans in Asia, Europe and the United States (Steere et al. 2004). In endemic regions, seroprevalence estimates of exposure of dogs to *B.burgdorferi* s.l range from 40% to 89% (Bushmich 1994; DAFF 2008).

Serosurveillance and attempts at isolation from potential tick vectors have failed to reveal conclusive evidence of Lyme disease in Australia (Baldock et al. 1993; Doggett et al. 1997; Hudson et al. 1998; Rothwell et al. 1989; Russell 1995).

Lyme disease is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

#### **4.10.2 Technical information**

##### **Epidemiology**

Prevalence of *B. burgdorferi* s.l. infection in vector-competent ticks varies geographically and is a good predictor of Lyme disease incidence. In endemic areas, prevalence in adult ticks averages 50%, in part because an adult has two chances of acquiring an infectious blood meal, having fed as both a larva and a nymph (Barbour and Fish 1993). Vector ticks harbour and transmit *B. burgdorferi* s.l. to humans and animals, with *Ixodes* spp. commonly implicated in transmission (Bushmich 1994). Other ticks, which do not bite humans, may play a key role in maintaining enzootic life cycles of *B. burgdorferi* s.l. in nature as vectors to reservoir hosts (Xu et al. 2003). Reporting requirements for Lyme disease in humans vary internationally. Some European countries have mandatory notification and surveillance programs; others do not (Coumou et al. 2011; Sood et al. 2011). Transmission of *B. burgdorferi* s.l. is unlikely to occur until the tick has been attached for at least 24 hours (Berger et al. 1995). There is evidence that *I. holocyclus* nymphs infected with *B. burgdorferi* s.l. do not retain infection after moulting (Piesman and Stone 1991). Direct modes of transmission have been demonstrated experimentally in dogs and mice via oral, intramuscular and subcutaneous routes (Burgess et al. 1986). Iatrogenic transmission is also possible (Parker and White 1992).

There is no evidence that infected dogs and cats pose a threat to humans; however, they do provide a means by which infected ticks can be carried into the domestic environment (Straubinger 2000). In endemic areas, dogs are at an equal or greater risk of becoming infected than humans (Eng et al. 1988), and have a role as a sentinel species (Hamer et al. 2009).

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

The incubation period is 2–5 months (CFSPH 2005). Lyme disease in dogs is primarily an acute or subacute arthritis (Appel et al. 1993). Approximately 5% of naturally infected and 75% of experimentally infected dogs show clinical signs of disease, which include anorexia and lethargy. Rarely, signs can include heart block, fatal kidney failure and neurological changes (Appel et al. 1993; Levy et al. 1993; Straubinger 2000). In experimental infections, cats may develop pyrexia, lethargy, stiffness and arthritis, or remain subclinically affected. Cases of naturally occurring disease have not been published in cats, although 5–47% of cats are seropositive in surveys (CFSPH 2011).

### **Diagnosis**

There are no specific clinical, haematological or biochemical changes that are pathognomonic for Lyme disease. The serological tests, enzyme-linked immunosorbent assay (ELISA) and indirect fluorescent antibody test (IFAT), are both available to detect antibodies to *B. burgdorferi* s.l. However, inconsistent results among laboratories, false positives due to cross-reactivity, and the inability to distinguish between infection and vaccination can complicate interpretation of results. PCR is sensitive and specific, but does not allow the differentiation between live and dead organisms, and false negatives can occur (Straubinger 2000).

### **Treatment**

Corticosteroids and other anti-inflammatory drugs are sometimes used for treatment of Lyme disease in dogs; however, persistent subclinical infection with *B. burgdorferi* s.l. can be reactivated by a two-week course of prednisone (Straubinger 2000). Seronegative Lyme disease has been reported in humans following early antibiotic treatment, which possibly interferes with antibody production (Dattwyler et al. 1988). False seronegatives can occur with the ELISA during the first weeks after infection (Cohen et al. 1992). In addition, because *B. burgdorferi* s.l. has the ability to convert and reconvert to cystic forms both *in vivo* and *in vitro*, infections can reactivate (Brorson and Brorson 2004).

Treatment with doxycycline is frequently recommended due to the drug being inexpensive, having anti-inflammatory properties and being appropriate for the treatment of possible co-infections. Experimental results indicate that antibiotic treatment is not reliable in eliminating infection with *B. burgdorferi* s.l. An optimal dose and duration of treatment is unknown. Doxycycline is typically administered at 10mg/kg daily, with the duration of treatment between 14 days and 4 weeks. (Littman et al. 2006).

### **Vector management**

The best prevention for Lyme disease is tick control. Effective tick prevention depends on regular treatment of dogs and cats with a suitable parasiticide in accordance with the manufacturer's recommendations. For dogs and cats visiting tick endemic regions, tick prevention should be practised. Owners should search animals daily for ticks and physically remove and dispose of any ticks detected.

### 4.10.3 Current biosecurity measures

There are no specific biosecurity measures for Lyme disease. Current biosecurity measures for the management of external parasites that may act as disease vectors are provided below.

#### Vector management

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a parasiticide effective against ticks on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be treated with a parasiticide effective against ticks on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks.

### 4.10.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by Lyme disease:

- Lyme disease is the most commonly reported tick-borne disease in humans in Asia, Europe and the United States.
- In endemic areas, dogs have an equal or greater likelihood of becoming infected to that of humans.
- Lyme disease in dogs is primarily an acute or subacute arthritis. The incubation period is 2–5 months, most infections are subclinical and diagnosis is not straightforward. Clinical signs have not been reported in cats.
- Exotic ticks infected with *B. burgdorferi* s.l. may be present on imported dogs and cats. Local ticks feeding on infected animals may become infected and act as vectors.
- If potential tick vectors in Australia became infected, they may spread infection to other animals, which may then act as reservoir hosts.
- Birds and small mammals may disseminate the organism across a wide area.
- Transmission via semen is not a recognised mode of spread.

### 4.10.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for Lyme disease continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for Lyme disease are not warranted for dog or cat semen.

The following measures would provide appropriate risk management options.

#### **Pre-export measures (dogs and cats)**

##### *Vector management*

- As for pre-export measures for external parasite control (see 4.13.5).

#### **Post-arrival measures (dogs and cats)**

##### *Vector management*

- As for post-arrival measures for external parasite control (see 4.13.5).
- To manage the biosecurity risks of Lyme disease, dogs and cats may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### **References**

Anderson JF, Johnson RC, Magnarelli LA, Hyde FW (1985). Identification of endemic foci of Lyme disease: isolation of *Borrelia burgdorferi* from feral rodents and ticks (*Dermacentor variabilis*). *Journal of Clinical Microbiology* 22:36–38.

Appel MJG, Allan S, Jacobson RH, Lauderdale TL, Chang YF, Shin SJ, Thomford JW, Todhunter RJ, Summers BA (1993). Experimental Lyme disease in dogs produces arthritis and persistent infection. *Journal of Infectious Diseases* 167:651–654.

Baldock FC, Yamane I, Gardner I (1993). Pilot survey for Lyme disease antibodies in Brisbane dogs. *Australian Veterinary Journal* 70:356–357.

Barbour AG, Fish D (1993). The biological and social phenomenon of Lyme disease. *Science* 260:1610–1616.

Berger BW, Johnson RC, Kodner C, Coleman L (1995). Cultivation of *Borrelia burgdorferi* from human tick bite sites: a guide to the risk of infection. *Journal of the American Academy of Dermatology* 32:184–187.

Brinkerhoff RJ, Folsom-O'Keefe CM, Tsao K, Diuk-Wasser MA (2009). Do birds affect Lyme disease risk? Range expansion of the vector-borne pathogen *Borrelia burgdorferi*. *Frontiers in Ecology and the Environment* 9:103–110.

Brorson Ø, Brorson S-V (2004). An *in vitro* study of the susceptibility of mobile and cystic forms of *Borrelia burgdorferi* to tinidazole. *International Microbiology* 7:139–142.

Burgdorfer W (1991). Lyme borreliosis: ten years after discovery of the etiologic agent, *Borrelia burgdorferi*. *Infection* 19:257–262.

Burgdorfer W, Barbour AG, Hayes SF, Benach JL, Grunwaldt E, Davis JP (1982). Lyme disease: a tick-borne spirochetosis? *Science* 216:1317–1319.

- Burgess EC, Amundson TE, Davis JP, Kaslow RA, Edelman R (1986). Experimental inoculation of *Peromyscus* spp. with *Borrelia burgdorferi*: evidence of contact transmission. *The American Journal of Tropical Medicine and Hygiene* 35:355–359.
- Burgess EC, Wachal MD, Cleven TD (1993). *Borrelia burgdorferi* infection in dairy cows, rodents, and birds from four Wisconsin dairy farms. *Veterinary Microbiology* 35:61–77.
- Bushmich SL (1994). Lyme borreliosis in domestic animals. *Journal of Spirochetal and Tick-borne Diseases* 1:1–6.
- CFSPH (Center for Food Security and Public Health) (2005). Lyme disease. CFSPH, Iowa State University, Ames.  
<http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm> (accessed 25 February 2009)
- CFSPH (Center for Food Security and Public Health) (2011) Lyme disease. CFSPH, Iowa State University, Ames.  
[http://www.cfsph.iastate.edu/Factsheets/pdfs/lyme\\_disease.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/lyme_disease.pdf) (accessed 31 May 2011)
- Cohen ND, Heck FC, Heim B, Flad DM, Bosler EM, Cohen D (1992). Seroprevalence of antibodies to *Borrelia burgdorferi* in a population of horses in central Texas. *Journal of the American Veterinary Medical Association* 201:1030–1034.
- Coumou J, van der Poll T, Speelman P, Hovius JWR (2011). Tired of Lyme borreliosis: Lyme borreliosis in the Netherlands. *The Netherlands Journal of Medicine* 69:101–111.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2008). Dogs and ticks. Exotic Animal Disease Newsletter 2:2, DAFF, Canberra.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National notifiable diseases list of terrestrial animals, December 2010. DAFF, Canberra.  
[http://www.daff.gov.au/\\_\\_data/assets/pdf\\_file/0019/1015075/notifiable-diseases.pdf](http://www.daff.gov.au/__data/assets/pdf_file/0019/1015075/notifiable-diseases.pdf) (accessed 1 July 2011)
- Dattwyler RJ, Volkman DJ, Luft BJ, Halperin JJ, Thomas J, Golightly MG (1988). Seronegative Lyme disease: dissociation of specific T- and B-lymphocyte responses to *Borrelia burgdorferi*. *New England Journal of Medicine* 319:1441–1446.
- Divers TJ (2007). Lyme disease. In *Equine infectious diseases*, 1st edn, Sellon DC, Long MT (eds), Saunders Elsevier, St. Louis, pp. 310–312.
- Doggett SL, Russell RC, Lawrence R, Dickeson D (1997). Lyme disease. Department of Medical Entomology, University of Sydney.  
<http://medent.usyd.edu.au/fact/lyme%20disease.htm#intro> (accessed 24 February 2009)

- Eng TR, Wilson ML, Spielman A, Lastavica CC (1988). Greater risk of *Borrelia burgdorferi* infection in dogs than in people. *Journal of Infectious Diseases* 158:1410–1411.
- Fridriksdottir V, Nesse LL, Gudding R (1992). Seroepidemiological studies of *Borrelia burgdorferi* infection in sheep in Norway. *Journal of Clinical Microbiology* 30:1271–1277.
- Greene CE, Appel MJ, Straubinger RK (1998). Lyme borreliosis. In *Infectious diseases of the dog and cat*, 2nd edn, Greene CE (ed.), WB Saunders, Philadelphia, pp. 282–293.
- Hamer SA, Tsao JI, Walker ED, Mansfield LS, Foster ES, Hickling GJ (2009). Use of tick surveys and serosurveys to evaluate pet dogs as a sentinel species for emerging Lyme disease. *American Journal of Veterinary Research* 70:49–56.
- Hudson BJ, Stewart M, Lennox VA, Fukunaga M, Yabuki M, Macorison H, Kitchener-Smith J (1998). Culture-positive Lyme borreliosis. *Medical Journal of Australia* 168:500–502.
- Imai DM, Barr BC, Daft B, Bertone JJ, Feng S, Hodzic E, Johnston JM, Olsen KJ, Barthold SW (2011). Lyme neuroborreliosis in 2 horses. *Veterinary Pathology* 48:1151–1157.
- Kawabata H, Masuzawa T, Yanagihara Y (1993). Genomic analysis of *Borrelia japonica* sp. nov. isolated from *Ixodes ovatus* in Japan. *Microbiology and Immunology* 37:843–848.
- Lane RS, Regnery DC (1989). Lagomorphs as sentinels for surveillance of borreliosis in the far western United States. *Journal of Wildlife Diseases* 25:189–193.
- Levy SA, Lissman BA, Ficke CM (1993). Performance of a *Borrelia burgdorferi* bacterin in borreliosis-endemic areas. *Journal of the American Veterinary Medical Association* 202:1834–1838.
- Littman MP, Goldstein RE, Labato MA, Lappin MR, Moore GE (2006) ACVIM small animal consensus statement on Lyme disease in dogs: diagnosis, treatment, and prevention. *Journal of Veterinary Internal Medicine* 20: 422-434.
- Madigan JE, Teitler J (1988). *Borrelia burgdorferi* borreliosis. *Journal of the American Veterinary Medical Association* 192:892–904.
- Magnarelli LA, Anderson JF, Levine HR, Levy SA (1990). Tick parasitism and antibodies to *Borrelia burgdorferi* in cats. *Journal of the American Veterinary Medical Association* 197:63–66.
- Ogden NH, Bouchard C, Kurtenbach K, Margos G, Lindsay LR, Trudel L, Nguon S, Milord F (2010). Active and passive surveillance and phylogenetic analysis of *Borrelia burgdorferi* elucidate the process of Lyme disease risk emergence in Canada. *Environmental Health Perspectives* 118:909–914.

- Ogden NH, Nuttall PA, Randolph SE (1997). Natural Lyme disease cycles maintained via sheep by co-feeding ticks. *Parasitology* 115:591–599.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Olsen B, Duffy DC, Jaenson TG, Gylfe A, Bonnedahl J, Bergstrom S (1995). Transhemispheric exchange of Lyme disease spirochetes by seabirds. *Journal of Clinical Microbiology* 33:3270–3274.
- Olsen B, Jaenson TG, Noppa L, Bunikis J, Bergstrom S (1993). A Lyme borreliosis cycle in seabirds and *Ixodes uriae* ticks. *Nature* 362:340–342.
- Parker JL, White KK (1992). Lyme borreliosis in cattle and horses: a review of the literature. *The Cornell Veterinarian* 82:253–274.
- Piesman J, Stone BF (1991). Vector competence of the Australian paralysis tick, *Ixodes holocyclus*, for the Lyme disease spirochete *Borrelia burgdorferi*. *International Journal for Parasitology* 21:109–111.
- Rothwell JT, Christie BM, Williams C, Walker K (1989). Suspected Lyme disease in a cow. *Australian Veterinary Journal* 66:296–298.
- Russell RC (1995). Lyme disease in Australia: still to be proven! *Emerging Infectious Diseases* 1:29–31.
- Ryffel K, Peter O, Rutti B, Suard A, Dayer E (1999). Scored antibody reactivity determined by immunoblotting shows an association between clinical manifestations and presence of *Borrelia burgdorferi sensu stricto*, *B. garinii*, *B. afzelii* and *B. valaisiana* in humans. *Journal of Clinical Microbiology* 37:4086–4092.
- Sood SK, O'Connell S, Weber K (2011). The emergence and epidemiology of Lyme borreliosis in Europe and North America. In *Lyme borreliosis in Europe and North America: epidemiology and clinical practice*, 1st edn, Sood SK (ed.), John Wiley & Sons, Inc., Hoboken, pp. 1–35.
- Steere AC, Coburn J, Glickstein L (2004). The emergence of Lyme disease. *Journal of Clinical Microbiology* 113:1093–1101.
- Stoebel K, Schoenberg A, Streich WJ (2003). The seroepidemiology of Lyme borreliosis in zoo animals in Germany. *Epidemiology and Infection* 131:975–983.
- Straubinger RK (2000). Lyme borreliosis in dogs. Recent advances in canine infectious diseases. [http://www.ivis.org/advances/Infect\\_Dis\\_Carmichael/straubinger/chapter\\_frm.asp?LA=1](http://www.ivis.org/advances/Infect_Dis_Carmichael/straubinger/chapter_frm.asp?LA=1) (accessed 31 May 2011)

Telford SR, III, Spielman A (1989). Enzootic transmission of the agent of Lyme disease in rabbits. *The American Journal of Tropical Medicine and Hygiene* 41:482–490.

Wang G, van Dam AP, Schwartz I, Dankert J (1999). Molecular typing of *Borrelia burgdorferi sensu lato*: taxonomic, epidemiological, and clinical implications. *Clinical Microbiology Reviews* 12:633–653.

Xu G, Fang QQ, Keirans JE, Durden LA (2003). Molecular phylogenetic analyses indicate that the *Ixodes ricinus* complex is a paraphyletic group. *The Journal of Parasitology* 89:452–457.

## 4.11 Nagana

### 4.11.1 Background

Nagana is a generic term for a trypanosomal disease of animals in tropical and subtropical Africa (Barrowman et al. 1994). Nagana occurs in a wide range of domestic animals including cats, dogs, cattle, goats, sheep, pigs, horses and donkeys. Several species of laboratory animals can be infected experimentally, but human infections are rare.

The three trypanosome species causing nagana—*Trypanosoma brucei brucei*, *T. congolense* and *T. vivax*—are usually transmitted by *Glossina* spp. (tsetse fly).

Dogs and cats are considered to be refractory to infection with *T. vivax* and therefore do not develop an infective parasitaemia (Soulsby 1982).

*T. vivax* was introduced into South America with the importation of cattle from Africa where the disease mainly affects cattle and buffalo, but has been reported to infect wild antelopes and capybara (Osorio et al. 2008). *T. vivax* has become extensively established in regions beyond the range of the tsetse fly, in Central and South America and parts of the Caribbean, and is transmitted mechanically by tabanid flies. Australia has suitable vectors of the genera *Tabanus* and *Stomoxys* in some regions.

Trypanosomosis (tsetse transmitted) is an OIE-listed disease of cattle (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011). There are no recommendations in the *Terrestrial animal health code* (OIE 2011) for the importation of animals for tsetse-transmitted trypanosomosis. There is a chapter in the *Manual of diagnostic tests and vaccines* relating to the disease in cattle (OIE 2008).

### 4.11.2 Technical information

#### Epidemiology

The epidemiology of nagana depends primarily on the species of the various tsetse fly vectors, and their distribution, abundance and host-feeding preferences. Dogs and cats are generally resistant to infection and do not play a role in transmission (Soulsby 1982). Trypanosomes may be present in the bloodstream between 5 and 20

days post-infection and can survive for long periods in the mammalian host, maximising opportunity for vector transmission. It is not likely that either *T. brucei* or *T. congolense* would establish outside the range of their usual tsetse vectors.

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

In dogs and cats, the disease caused by *T. congolense* infection is typically acute with clinical signs including icterus, anaemia, enlarged lymph nodes, keratitis, ulcerative stomatitis, gastroenteritis and subcutaneous oedema. *T. brucei* infection also produces acute disease in dogs and cats, and typically includes pyrexia and oedema of the eyelids and thorax. Ocular and central nervous signs may also occur (Soulsby 1982).

### **Diagnosis**

A variety of diagnostic tests are available that vary in sensitivity and specificity. Parasite detection techniques, using microscopic examination of wet and stained thick or thin blood films, are highly specific but their sensitivity is relatively low. Several antibody detection techniques have been developed to detect trypanosomal antibodies for the diagnosis of trypanosomiasis, primarily in cattle. The methods of choice are the indirect fluorescent antibody test and the trypanosomal antibody-linked immunosorbent assay (OIE 2008).

### **Treatment**

Some chemotherapeutic agents are available to treat trypanosomiasis, but their use is largely limited to the treatment of disease in humans. Treatment is expensive and efficacy is variable. Severe toxicity, as well as drug resistance are recognised as limitations that can be associated with treatment.

#### **4.11.3 Current biosecurity measures**

There are no specific biosecurity measures for nagana.

#### **4.11.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by nagana:

- Nagana is widespread in tropical and subtropical Africa, and transmitted by tsetse flies.
- Dogs and cats are susceptible to infection with *T. brucei* or *T. congolense*, but these parasites are only transmitted by tsetse fly vectors. Tsetse flies are not present in Australia and, therefore, an infected imported animal would not present a risk of the disease agent establishing.
- Dogs and cats are resistant to infection with *T. vivax* and do not play a role in transmission.

#### 4.11.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for nagana are not warranted for dogs, cats or their semen.

#### References

Barrowman P, Stoltz WH, van der Lugt JJ, Williamson CC (1994). Dourine. In *Infectious diseases of livestock with special reference to southern Africa*, 1st edn, Coetzer JAW, Thomson GR, Tustin RC (eds), Oxford University Press, Cape Town, pp. 206–212.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National notifiable diseases list of terrestrial animals, December 2010. DAFF, Canberra.

[http://www.daff.gov.au/\\_\\_data/assets/pdf\\_file/0019/1015075/notifiable-diseases.pdf](http://www.daff.gov.au/__data/assets/pdf_file/0019/1015075/notifiable-diseases.pdf) (accessed 1 July 2011)

OIE (World Organisation for Animal Health) (2008). Trypanosomosis (tsetse-transmitted). *Manual of diagnostic tests and vaccines for terrestrial animals 2011*. OIE, Paris.

[http://web.oie.int/eng/normes/mmanual/2008/pdf/2.04.18\\_TRYPANOSOMOSIS.pdf](http://web.oie.int/eng/normes/mmanual/2008/pdf/2.04.18_TRYPANOSOMOSIS.pdf) (accessed 27 August 2010)

OIE (World Organisation for Animal Health) (2011). *Terrestrial animal health code*. OIE, Paris.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Osorio ALAR, Madruga CR, Desquesnes M, Soares CO, Ribeiro LRR, da Costa SCG (2008). *Trypanomoma (Duttonella) vivax*: its biology, epidemiology, pathogenesis, and introduction in the new world- a review. *Memoirs of Instituto Oswaldo Cruz* (Rio de Janeiro) 103:1–13.

Soulsby EJJ (1982). Protozoa. In *Helminths, arthropods and protozoa of domesticated animals*, 7th edn, Baillière Tindall, London, pp. 507–759.

## 4.12 Nipah virus encephalitis

### 4.12.1 Background

Nipah virus (NiV) is closely related to Hendra virus and is a member of the Henipavirus genus in the family Paramyxoviridae (Lamb et al. 2005). NiV was first described in Malaysia in 1998 as a cause of pyrexia associated with respiratory disease in weaner and growing pigs. Infection in exposed pig farm workers and abattoir workers resulted in an often fatal encephalitic disease (Chua et al. 2000).

Since then, there have been outbreaks in humans in Bangladesh (Luby et al. 2006), India (Promed Mail 2007) and Singapore (Paton et al. 1999).

NiV spreads readily between pigs and from pigs to humans. There is also serological evidence of natural infection in cats, dogs, horses and goats (Chua 2003).

Experimental infection has been demonstrated in guinea pigs, hamsters and ferrets (Williamson and Torres-Velez 2010).

NiV infection is an OIE-listed disease of pigs (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011). There is a chapter in the *Manual of diagnostic tests and vaccines* (OIE 2010b) relating to the disease, but there are no recommendations in the *Terrestrial animal health code* (OIE 2011) for the importation of animals with respect to NiV infection.

During the initial Nipah virus encephalitis (NVE) outbreak in Malaysia, very little was known about the disease. In addition, there were reports of dogs testing positive for NiV (Promed Mail 1999). Accordingly, import conditions for NiV infection were introduced by the Australian Government Department of Agriculture.

#### **4.12.2 Technical information**

##### **Epidemiology**

Fruit bats of the genus *Pteropus* are the main reservoir hosts. The distribution of *Pteropus* species extends from the western Indian Ocean islands of Mauritius, Madagascar and Comoro, along the sub-Himalayan region of Pakistan and India, through South-East Asia, the Philippines, Indonesia, New Guinea, south-west Pacific Islands as far east as the Cook Islands, and Australia excluding Tasmania (Field et al. 2001).

Illness or death due to NiV infection has not been reported in any bat species. In an experimental study, NiV infection in *Pteropus poliocephalus* did not result in overt clinical disease (Middleton et al. 2007). However, NiV is intermittently shed in urine, which may be sufficient to maintain the virus in the bat population (Chua et al. 2002).

NiV is readily transmissible from pig-to-pig and from pig-to-human. The virus is transmitted directly or indirectly from bats to pigs, with pigs acting as amplifying hosts that can then transmit the virus by direct contact with other animals and humans. In NVE outbreaks in Bangladesh, direct and indirect bat-to-human transmission appears to have been responsible for spread of infection. In addition, human-to-human spread has been reported (Luby et al. 2006).

Other domesticated animals can be infected by contact with pigs. Although there was serological evidence of infection of dogs following close association with infected pigs during the NVE outbreak in Malaysia in 1998, the results of a serological survey suggest that the virus did not spread horizontally in dogs (Mills et al. 2009). Cats have been experimentally infected by intranasal and oral inoculation. Horizontal transmission has not been demonstrated between cats but it is theoretically possible (Epstein et al. 2006). NiV has been found in feline respiratory

secretions, urine, the placenta and embryonic fluids (Middleton et al. 2002). In addition, in utero transmission has been demonstrated in this species (Mungall et al. 2007).

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

Serological surveys in Malaysia around the time of the outbreak demonstrated seroprevalence ranging from 15 to 55% in dogs, 4 to 6% in cats and 1.5% in goats (CFSPH 2007; Chua 2003). However, following an extensive culling program of infected pigs, subsequent serosurveillance of domestic animals from October 1999 to December 2000 demonstrated the absence of exposure to NiV. Effective from 1 June 2001, Malaysia declared itself free from NiV infection in domestic animals (OIE 2001). As of April 2011, no further outbreaks of NiV infection have been reported in Malaysia (OIE 2010a).

During the outbreaks in Bangladesh and India, infections in pigs and other domesticated species were not reported (OIE 2010a). Infections in nonporcine domesticated species appear to be uncommon or non-existent in the absence of infected pigs.

#### **Clinical signs**

Natural infections in dogs and cats appear to be rare with limited information regarding the incubation period. Clinical signs in dogs include pyrexia, respiratory distress, conjunctivitis, and mucopurulent ocular and nasal discharges (Hooper et al. 2001). In experimentally infected cats, incubation periods of six to eight days have been reported (Mungall et al. 2007; Njaa 2008). In a confirmed case in a cat, severe dyspnoea due to pulmonary oedema was reported (Hooper et al. 2001).

#### **Diagnosis**

NiV infections can be diagnosed by viral isolation, the detection of antigens or nucleic acids and serology (CFSPH 2007; OIE 2010b). The currently accepted reference procedure is the virus neutralisation test (OIE 2010b).

#### **Treatment**

Treatment of NiV infection is supportive and agents effective in preventing the progression of disease have not been identified (CFSPH 2007).

#### **4.12.3 Current biosecurity measures**

All dogs and cats from Malaysia must be tested for NiV by the serum neutralisation test. The blood sample must be collected within 30 days before export and record a negative result.

#### 4.12.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by Nipah virus encephalitis:

- NiV infection is not present in domestic animals in any approved country and Malaysia was declared free from NiV infection in domestic animals in 2001.
- Fruit bats of the genus *Pteropus* are the main reservoir hosts of NiV.
- Pigs are the main amplifying hosts of NiV.
- Dogs and cats are not recognised as amplifying hosts of NiV.
- There is no evidence of horizontal spread of NiV infection in either dogs or cats.

#### 4.12.5 Conclusion

Based on the preceding key points it was concluded that risk management measures are not warranted for NiV encephalitis in dogs, cats or their semen. In the event of an outbreak of NiV infection in an approved country during the 12 months before export, adoption of a serological test requirement, with a negative result, would provide an appropriate risk management option for dogs and cats.

#### References

- CFSPH (Center for Food Security and Public Health) (2007). Nipah virus infection. CFSPH, Iowa State University, Ames.  
<http://www.cfsph.iastate.edu/Factsheets/pdfs/nipah.pdf> (accessed 3 December 2008)
- Chua KB (2003). Nipah virus outbreak in Malaysia. *Journal of Clinical Virology* 26:265–275.
- Chua KB, Bellini WJ, Rota PA, Harcourt BH, Tamin A, Lam SK, Ksiazek TG, Rollin PE, Zaki SR, Shieh W-J, Goldsmith CS, Gubler DJ, Roehrig JT, Eaton B, Gould AR, Olson J, Field H, Daniels P, Ling AE, Peters CJ, Anderson LJ, Mahy BWJ (2000). Nipah virus: a recently emergent deadly paramyxovirus. *Science* 288:1432–1435.
- Chua KB, Koh CL, Hooi PS, Wee KF, Khong JH, Chua BH, Chan YP, Lim ME, Lam SK (2002). Isolation of Nipah virus from Malaysian Island flying foxes. *Microbes and Infection* 4:145–151.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Epstein JH, Rahman SA, Zambriski JA, Halpin K, Meehan G, Jamaluddin AA, Hassan SS, Field HE, Hyatt AD, Daszak P, Henipavirus Ecology Research Group (2006). Feral cats and risk for Nipah virus transmission. *Emerging Infectious Diseases* 12:1178–1179.

- Field H, Young P, Yob JM, Mills J, Hall L, Mackenzie J (2001). The natural history of Hendra and Nipah viruses. *Microbes and Infection* 3:307–314.
- Hooper P, Zaki S, Daniels P, Middleton D (2001). Comparative pathology of the diseases caused by Hendra and Nipah virus. *Microbes and Infection* 3:315–322.
- Lamb RA, Collins PL, Kolakofsky D, Melero JA, Nagai Y, Oldstone MBA, Pringle CR, Rima BK (2005). Paramyxoviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses*, Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds), Elsevier, San Diego, pp. 655–668.
- Luby SP, Rahman M, Hossain MJ, Blum LS, Husain MM, Gurley E, Khan R, Ahmed B-N, Rahman S, Nahar N, Kenah E, Comer JA, Ksiazek TG (2006). Foodborne transmission of Nipah virus, Bangladesh. *Emerging Infectious Diseases* 12: 888–1894.
- Middleton DJ, Morrissy CJ, van der Heide BM, Russell GM, Braun MA, Westbury HA, Halpin K, Daniels PW (2007). Experimental Nipah virus infection in pteropid bats (*Pteropus poliocephalus*). *Journal of Comparative Pathology* 136:266–272.
- Middleton DJ, Westbury HA, Morrissy CJ, van der Heide BM, Russell GM, Braun MA, Hyatt AD (2002). Experimental Nipah virus infection in pigs and cats. *Journal of Comparative Pathology* 126:124–136.
- Mills JN, Alim ANM, Bunning ML, Lee OB, Wagoner KD, Amman BR, Stockton PC, Ksiazek TG (2009). Nipah virus infection in dogs, Malaysia, 1999. *Emerging Infectious Diseases* 15:950–952.
- Mungall BA, Middleton D, Crameri G, Halpin K, Bingham J, Eaton BT, Broder CC (2007). Vertical transmission and fetal replication of Nipah virus in an experimentally infected cat. *Journal of Infectious Diseases* 196:812–816.
- Njaa BL (2008). Emerging viral encephalitides in dogs and cats. *The Veterinary Clinics of North America. Small Animal Practice* 38:863–878.
- OIE (World Organisation for Animal Health) (2001). Malaysia: declaration of freedom from Nipah virus in domestic animals from 1 June 2001. *Bulletin de l'Office International des Epizooties* 113:514–515.
- OIE (World Organisation for Animal Health) (2010a). Disease timelines: nipah virus encephalitis. WAHID Interface: Animal Health Information, OIE, Paris. [http://web.oie.int/wahis/public.php?page=disease\\_timelines&disease\\_type=Terrestrial&disease\\_id=190&empty=999999](http://web.oie.int/wahis/public.php?page=disease_timelines&disease_type=Terrestrial&disease_id=190&empty=999999) (accessed 7 April 2011)
- OIE (World Organisation for Animal Health) (2010b). Hendra and Nipah virus diseases. *Manual of diagnostic tests and vaccines for terrestrial animals 2011*, OIE, Paris. [http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.09.06\\_HENDRA\\_&\\_NIPAH\\_FINAL.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.09.06_HENDRA_&_NIPAH_FINAL.pdf) (accessed 15 March 2012)

OIE (World Organisation for Animal Health) (2011). *Terrestrial animal health code*. OIE, Paris.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Paton NI, Leo YS, Zaki SR, Auchus AP, Lee KE, Ling AE, Chew SK, Ang B, Rollin PE, Umapathi T, Sng I, Lee CC, Lim E, Ksiazek TG (1999). Outbreak of Nipah-virus infection among abattoir workers in Singapore. *Lancet* 354:1253–1256.

Promed Mail (1999). Nipah virus, dogs positive. ProMED Mail. <http://www.promedmail.org> (accessed 6 May 2011)

Promed Mail (2007). Nipah virus, fatal—India (West Bengal). ProMED Mail. [http://www.promedmail.org/pls/otn/f?p=2400:1202:411279077062146::NO::F2400\\_P1202\\_CHECK\\_DISPLAY,F2400\\_P1202\\_PUB\\_MAIL\\_ID:X,37338](http://www.promedmail.org/pls/otn/f?p=2400:1202:411279077062146::NO::F2400_P1202_CHECK_DISPLAY,F2400_P1202_PUB_MAIL_ID:X,37338) (accessed 5 April 2011)

Williamson MM, Torres-Velez FJ (2010). Henipavirus: a review of laboratory animal pathology. *Veterinary Pathology Online* 47:871–880.

## 4.13 Parasites—external

### 4.13.1 Background

External parasites of dogs and cats (primarily ticks and fleas) can excrete toxins that can cause illness in the hosts and/or harbour disease agents such as viruses, bacteria and protozoa which can infect animals and humans. External parasites, with the exception of screw-worm flies, *Cochliomyia hominivorax* and *Chrysomya bezziana*, are generally not listed as notifiable diseases. However, some tick-borne diseases; for example, Crimean Congo haemorrhagic fever and tularaemia are OIE-listed diseases (OIE 2012) and are nationally notifiable diseases in Australia (DAFF 2011). Screw-worm fly is reviewed elsewhere in this report, although myiasis is included in this section as a general hazard of biosecurity concern. The following vector-borne diseases are also reviewed in this report: canine monocytic ehrlichiosis, Chagas' disease, hepatozoonosis, leishmaniasis, Lyme disease, nagana, piroplasmosis, Rift Valley fever, surra, tularaemia and yersiniosis.

External parasites were briefly reviewed by the Bureau of Resource Sciences (BRS) in its *Review of quarantine policy for dogs and cats, with particular reference to rabies* (BRS 1993). The review:

- listed external parasites (ticks, fleas, lice, mites) as infectious or contagious diseases transmissible to other animals during post-arrival quarantine
- noted current strategy was treatment before entry with an external or topical chemical (insecticide etc.)

- proposed that clinical examination and treatment with topical chemicals (insecticides etc.) would be adequate to prevent the introduction of exotic external parasites.

Accordingly, import conditions based on the BRS review were developed for external parasites.

#### 4.13.2 Technical information

##### Epidemiology

Of 899 tick species identified (Barker and Murrell 2004), only a few have been identified as a principal or occasional vector of tick-borne diseases (Table 4). Similarly, of over 2000 flea species, only a few parasitise dogs and cats, and are implicated in the transmission of important zoonoses such as plague, murine typhus (Rolain et al. 2005) and tularaemia (Shaw et al. 2004).

Tick and flea species show a degree of preferential parasitism for specific host species; however, many opportunistically parasitise a wide range of host species. Fleas are capable of completing their life cycle on a single host, in contrast to ticks that, depending on the species, may require more than one host to complete their life cycle (Soulsby 1982b).

Table 4 Examples of significant tick-transmitted diseases of dogs

Tick vector	Potential tick-borne disease agent	Geographical distribution of ticks
<i>Rhipicephalus sanguineus</i>	<i>Babesia canis vogeli</i> <i>Babesia gibsoni</i> <i>Hepatozoon canis</i> <i>Ehrlichia canis</i> <i>Rickettsia conorii</i>	Tropical/semitemperal worldwide
<i>Dermacentor reticulatus</i> <i>Dermacentor marginatus</i>	<i>Babesia canis canis</i>	Tropical/semitemperal worldwide
<i>Dermacentor variabilis</i>	<i>Ehrlichia chaffeensis</i> <i>Rickettsia rickettsii</i>	United States
<i>Dermacentor andersoni</i>	<i>Rickettsia rickettsii</i>	United States
<i>Haemaphysalis elliptica</i>	<i>Babesia canis rossi</i>	Southern Africa
<i>Haemaphysalis bispinosa</i>	<i>Babesia gibsoni</i>	Africa, Asia, Southern Europe, United States, Middle East
<i>Amblyomma americanum</i>	<i>Hepatozoon americanum</i> <i>Ehrlichia chaffeensis</i> <i>Ehrlichia ewingii</i>	United States (southern)
<i>Amblyomma maculatum</i>	<i>Hepatozoon canis</i>	Africa, Asia, Middle East, Southern Europe
<i>Haemaphysalis longicornis</i>	<i>Hepatozoon canis</i>	Africa, Asia, Middle East, Southern Europe
<i>Ixodes</i> spp. (excluding the Australasian subgroup)	<i>Anaplasma phagocytophilum</i> genogroup <i>Borrelia burgdorferi</i> genogroup	Asia, Europe, Middle East, United States

Source: Adapted from Shaw et al. (2001)

Fleas have four development stages: egg, larva, pupae and adult—only adult fleas parasitise the host. The female flea can lay up to 50 eggs each day and 400–500 eggs in a lifetime.

Each tick species and life cycle stage is adapted to particular hosts, temperature and moisture levels (Soulsby 1982a). The female tick lays only one batch of eggs, but each batch may contain several thousand eggs. Ticks generally undergo four developmental stages: egg, larva, nymph and adult. Each stage usually feeds only once; however, some adult males may move to another host in search of a female and feed on that host. Depending on tick species and environmental conditions, the life cycle ranges from two months to a few years. To complete the life cycle, tick larvae, nymphs and adults feed on the protein rich blood of their host, usually impairing the host immune response to the blood-feeding activity.

For both fleas and ticks, feeding generally involves alternate periods of sucking blood, excreting saliva and sometimes regurgitating blood. The process of salivating and regurgitating blood provides a pathway for transmission of disease agents between the ticks and their hosts (Shaw et al. 2001; Soulsby 1982a).

Tick-borne disease agents (e.g. piroplasms, viruses, rickettsia, bacteria) may be maintained in ticks to the next development stage trans-stadially, transmitted to offspring transovarially and/or sexually transmitted (Thrusfield 1995).

Some ticks spend up to 95% of their time off a host in the environment. The brown dog tick, *Rhipicephalus sanguineus*, can live in both rural and urban areas and become established indoors, including in localities where it may not be able to survive in the external environment (Dantas-Torres 2010). Similar to ticks, most fleas spend a relatively short part of their life cycle on a host (Soulsby 1982b).

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

Most ticks and fleas cause only a mild skin irritation or an allergic reaction at the site of attachment to a host. A few species of ticks worldwide are associated with paralysis in animals (Soulsby 1982a).

### **Detection**

A thorough examination of the animal is required to detect ticks. If detected, the tick should be removed and submitted for identification.

THE DEPARTMENT OF AGRICULTURE post-arrival quarantine (PAQ) data for 2009 and 2010 show that exotic ticks were identified on 59/7430 (0.79%) dogs and 3/3873 (0.08%) cats imported, notwithstanding that certification accompanies the animals confirming that pre-export treatments and examinations have been performed. The introduced ticks were detected within ten days of entry of a dog or cat into PAQ.

## Treatment

Controlling and eradicating ticks requires an understanding of the tick life cycle and properties. Elimination of ticks will typically require the adoption of a combination of tactics, including direct application of approved parasiticides to animals and their environment, as well as reducing the potential for reinfestation of the yard, kennel and home environment (Stafford 2007).

A diverse range of parasiticial preparations presented in a variety of formulations (e.g. shampoos, rinses, spot-on formulations, impregnated collars, sprays and dips) are available to treat and control ectoparasites. However, the frequency of parasiticide treatment required varies with the duration of effectiveness of the parasiticide. No parasiticide treatment or combination of treatments is known to be 100% effective and resistance to parasiticides is a recognised problem (Soulsby 1982a). Effective flea control requires treatment of the animal and its habitat to reduce the presence of eggs and juvenile life cycle stages (Soulsby 1982b).

Some products require the parasites to attach and feed on the animal host before the active constituents can be effective. Such a mode of action is unlikely to reduce or limit the transmission of vector-borne diseases. Other products have active constituents that are available on the surface of the skin and haircoat to deter or promote detachment of ectoparasites before they feed on the host and transmit disease agents. Amitraz and permethrin are examples of active constituents that promote a rapid knockdown effect on external parasites and decrease the opportunity for disease agent transmission.

A combination of amitraz and fipronil in a topical formulation has been shown to achieve a significantly increased acaricidal effect on the brown dog tick (*Rhipicephalus sanguineus*) when compared to a topical application containing either fipronil or amitraz alone (Prullage et al. 2011). Pre-export application of an acaricidal product with enhanced acaricidal activity is likely to reduce the opportunity for dogs to be exposed to disease agents via tick vectors in the period before export and therefore reduce the likelihood that an animal is in the incubation phase of infection when imported.

### 4.13.3 Current biosecurity measures

Current biosecurity measures to manage external parasites are as follows:

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian. (Note: This treatment is not required for dogs and cats being imported from New Zealand due to the absence of external parasites of dogs and cats that are exotic to Australia.)

- Within four days immediately before export, dogs and cats must be subject to a thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks and fleas.

#### **4.13.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by external parasites:

- Ticks and fleas can harbour diseases of biosecurity concern for both animals and humans; measures to prevent the introduction of these external parasites, irrespective of species, are necessary.
- Tick species and life cycle stage vary in their host, geographical and environmental preferences.
- Detection of ticks and fleas can be difficult. Immature stages of ticks may take up to 10 days to detect.
- The detection of exotic ticks in PAQ is not uncommon.
- In 2009 and 2010, ticks were identified on 59/7430 (0.79%) dogs and 3/3873 (0.08%) cats imported, despite pre-export treatments and examinations.
- The detections in PAQ confirm the importance of a PAQ period sufficient to enable detection of exotic ticks that have not been eliminated by pre-export treatments or detected at examination.
- A tick or flea species may be present in Australia, but if introduced with an imported dog or cat can act as a vector to enable the introduction of an exotic disease agent.
- A single treatment with an approved parasiticide cannot be relied upon to eliminate ticks and fleas. Effective external parasite control requires an integrated management strategy to minimise the risk of infestation by ticks and fleas.

#### **4.13.5 Conclusion**

Based on the preceding key points, it was concluded that risk management measures for external parasites continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for external parasites are not warranted for dog or cat semen.

The following biosecurity measures would provide appropriate risk management.

##### **Pre-export measures (dogs and cats)**

- For at least the 21-day period immediately before export, dogs and cats must be treated with a parasiticide effective against ticks and fleas on contact, to provide continual protection against infestation until the day of export and during transport to Australia.

- The parasiticide must be applied by a government-approved veterinarian and in accordance with the manufacturer's recommendations.
- Within the five days immediately before export, dogs and cats must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from external parasites—ticks and fleas.
- If a tick is detected at any pre-export examination then the preparation is to recommence and sampling for ehrlichiosis to be rescheduled (see risk review for canine monocytic ehrlichiosis).

#### **Post-arrival measures (dogs and cats)**

- Dogs and cats must be subject to a minimum PAQ period of ten days.
- Both dogs and cats must be thoroughly examined by a Department of Agriculture veterinary officer for external parasites—ticks and fleas—as soon as practical after entry into PAQ and subject to regular monitoring during the period of PAQ.
- Both dogs and cats must be thoroughly examined by a Department of Agriculture veterinary officer for external parasites—ticks and fleas—before release from PAQ.
- If a tick is detected on imported dogs or cats in PAQ, the tick must be removed and submitted for identification, and the animal must be subject to treatment with a parasiticide effective on contact against external parasites.
- To manage the biosecurity risks of vector-borne disease, dogs and cats may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### **References**

Barker SC, Murrell A (2004). Systematics and evolution of ticks with a list of valid genus and species names. *Parasitology* 129:S15–S36.

BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper. BRS, Canberra.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Dantas-Torres F (2010). Biology and ecology of the brown dog tick, *Rhipicephalus sanguineus*. *Parasites and Vectors* 3:N/A.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.  
<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Prullage JB, Tran HV, Timmons P, Harriman J, Chester ST, Powell K (2011). The combined mode of action of fipronil and amitraz on the motility of *Rhipicephalus sanguineus*. *Veterinary Parasitology* 179 :302–310.

Rolain J-M, Bourry O, Davous B, Raoult D (2005). *Bartonella quintana* and *Rickettsia felis* in Gabon. *Emerging Infectious Diseases* 11:1742–1744.

Shaw SE, Day MJ, Birtles R, Breitschwerdt EB (2001). Tick-borne infectious diseases of dogs. *Trends in Parasitology* 17:74–80.

Shaw SE, Kenny MJ, Tasker S, Birtles RJ (2004). Pathogen carriage by the cat flea *Ctenocephalides felis* (Bouché) in the United Kingdom. *Veterinary Microbiology* 102:183–188.

Soulsby E JL (1982a). Family: Ixodidae Murray, 1877. In *Helminths, arthropods and protozoa of domesticated animals*, 7th edn, Baillière Tindall, London, pp. 456–475.

Soulsby E JL (1982b). Order: Siphonaptera Latreille, 1825. In *Helminths, arthropods and protozoa of domesticated animals*, 7th edn, Baillière Tindall, London, pp. 378–384.

Stafford KC, III (2007). *Tick management handbook: an integrated guide for homeowners, pest control operators, and public health officials for the prevention of tick-associated disease*, revised edn. The Connecticut Agricultural Experiment Station, New Haven.

Thrusfield M (1995). *Veterinary epidemiology*, 2nd edn, Blackwell Science, Oxford.

## 4.14 Parasites—internal

### 4.14.1 Background

Dogs and cats have the potential to harbour a diverse range of internal parasites. The majority of internal parasites of dogs and cats are located in the gastrointestinal tract, although parasitism of other internal organs, e.g. the respiratory and cardiovascular systems, also occurs. Although, many of the internal parasites of dogs and cats are endemic in Australia, there are a large number of parasites that remain exotic to Australia. Infestation by internal parasites can cause clinical signs of disease in susceptible hosts and, in some instances, life cycle stages of parasites carried by dogs and cats are zoonotic, which presents public health concerns.

### 4.14.2 Technical information

#### Life-cycles

Helminths form three main life-cycle stages: eggs, larvae and adults. Adult worms infect definitive hosts (those in which sexual development occurs) whereas larval stages may be free-living or parasitize invertebrate vectors, intermediate or paratenic hosts. Nematodes produce eggs that embryonate in utero or outside the host. The emergent larvae undergo 4 metamorphoses (moult) before they mature as adult male or female worms. Cestode eggs released from gravid segments embryonate to produce 6-hooked embryos (hexacanth oncospheres) which are ingested by

intermediate hosts. The oncospheres penetrate host tissues and become metacestodes (encysted larvae). When eaten by definitive hosts, the larvae excyst and form adult tapeworms. Trematodes have more complex life-cycles where 'larval' stages undergo asexual amplification in snail intermediate hosts. Eggs hatch to release free-swimming miracidia which actively infect snails and multiply in sac-like sporocysts to produce numerous rediae. These stages mature to cercariae which are released from the snails and either actively infect new definitive hosts or form encysted metacercariae on aquatic vegetation which is eaten by definitive hosts.

### **Clinical signs**

Internal parasitism results in a variety of non-specific clinical signs that vary according to animal susceptibility, the organ systems and tissues affected by infestation and the pathogenicity of the particular parasite. Clinical signs of parasitic infestation of the gastrointestinal tract can include weight loss, lethargy, altered appetite, anaemia, diarrhoea and/or vomiting.

### **Diagnosis**

Detection of infestation by internal parasites is typically conducted by direct examination of faecal smears or by applying sedimentation, flotation or centrifugation techniques to faecal specimens. However, detection of infestations by internal parasites via faecal specimens is not always reliable, particularly following recent infestation when parasites are in the prepatent phase of their life cycle.

### **Treatment**

Management and eradication of internal parasites requires an understanding of the parasitic life cycle, including intermediate and reservoir hosts where appropriate. Attention must be given not only to treatment of the principal host, but also to prevent reinfestation by eliminating dormant life cycle stages from the environment, and controlling an animal's activities and diet to reduce the likelihood of exposure.

In general anthelmintic treatment is effective against adult helminths in the gastrointestinal tract but not against encysted or migratory somatic larvae of ascarid parasites (Overgaauw 1997). The prepatent period for a number of parasites of dogs and cats (e.g. *Ancylostoma* spp., *Toxocara* spp.) is short (e.g. three weeks) and therefore an inter-treatment interval of about 14 days with an efficacious anthelmintic is generally recommended to reduce the risk of reinfestation by encysted or migratory larvae and environmental contamination with parasite eggs (Novartis 2010).

There are numerous anthelmintic preparations available for administration by different routes (e.g. oral, topical and injectable formulations). The spectrum of efficacy of anthelmintic agents against internal parasites is usually taxa-specific. For example, praziquantel is a highly effective parasiticide against flatworms (Class Trematoda and Class Cestoda), but has little efficacy against nematode parasites (Class Nematoda). The efficacy of anthelmintic treatment depends on the dose administered, the duration of parasiticide action, the severity of infestation as well as the ability to prevent re-infestation (Campbell and Graham 1999). For the majority of

internal parasites, there is no single anthelmintic treatment or combination of treatments that can be regarded as 100% effective (Hong et al. 2003; Rim 2005). The development of anthelmintic resistance due to prolonged, indiscriminate and/or inappropriate application of certain anthelmintic agents is recognised worldwide as a significant problem but is outside the scope of this policy review.

#### **4.14.3 Current biosecurity measures**

- Within 4 days before export, dogs and cats must be treated by a government-approved veterinarian with approved anthelmintics effective against nematodes and cestodes.

#### **4.14.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by internal parasites:

- Dogs and cats have the potential to introduce a diverse range of internal parasites of biosecurity concern to Australia.
- Requiring a specific diagnostic procedure for each parasite of biosecurity concern is impractical.
- As different developmental stages of parasites differ in their susceptibility to anthelmintic treatment, a single anthelmintic treatment is not a reliable measure to eliminate infestation with internal parasites.
- To minimise the risk of infestation with internal parasites in dogs and cats, it is best practice to administer two treatments at an interval of 14 days.
- Praziquantel is the drug of choice for a number of flatworms (such as cestodes and trematodes) of biosecurity concern.

#### **4.14.5 Conclusion**

Based on the preceding key points, it was concluded that risk management measures for internal parasites continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for internal parasites are not warranted for dog or cat semen.

The following biosecurity measures would provide appropriate risk management.

##### **Pre-export measures**

- In preparation for export, dogs and cats must be treated twice by a government-approved veterinarian with an approved anthelmintic (or combination of anthelmintics) effective against nematodes and cestodes at the manufacturer's recommended dose (the product/s used must include praziquantel).
- The first treatment must be administered within the 45 days immediately before export and at an interval of not less than 14 days before the second treatment.

The second treatment must be administered within the five days immediately before export.

## References

Campbell KL, Graham JC (1999). *Physaloptera* infection in dogs and cats. *Compendium on Continuing Education for the Practicing Veterinarian* 21:299–314.

Hong S-T, Lee SH, Lee S-J, Kho W-G, Lee M, Li S, Chung B-S, Seo M, Choi M-H (2003). Sustained-release praziquantel tablet: pharmacokinetics and the treatment of clonorchiasis in beagle dogs. *Parasitology Research* 91:316–320.

Novartis Animal Health (2010) Internal parasites of dogs and cats. <http://www.students.novartis.us/pdf/InternalParasiteManual.pdf> (Accessed 28 May 2013).

Overgaaauw PAM (1997) General introduction: Aspects of *Toxocara* epidemiology, Toxocarosis in dogs and cats. *Clinical Reviews in Microbiology* 23: 233-251.

Rim H-J (2005). Clonorchiasis: an update. *Journal of Helminthology* 79:269–281.

## 4.15 Piropalmsis

### 4.15.1 Background

Piropalmsis is caused by intra-erythrocytic protozoan parasites. The majority of piropalmsid parasites are tick-borne with additional modes of transmission proposed for some species. It is one of the most common infections of animals worldwide and can cause severe disease in domesticated dogs and cats, wild canids (dingoes, foxes, jackals and wolves) and felids (leopards and lions), horses, pigs and ruminants. Some piropalmsids are zoonotic. Susceptibility to infection varies with the species of piropalmsid, presence of competent tick vectors, age and immune status of the animal, and the presence or absence of co-infections. Acute, chronic and carrier states exist.

Piropalmsid parasites are found in dogs and cats on all continents and comprise two main genera, *Babesia* and *Theileria*. Piropalmsid spp. are largely dependent on specific tick vectors for transmission, although the identification of tick vectors is yet to be confirmed for many piropalmsid parasites.

Feline piropalmsis is less common and has not been as well researched as canine piropalmsis. Canine and feline piropalmsis are not OIE-listed diseases (OIE 2012) and are not nationally notifiable diseases (DAFF 2011). However, canine piropalmsids in particular are capable of causing severe clinical disease and their detection in temperate regions appears to have increased significantly in the past twenty years. Carrier states, transovarial transmission in ticks and translocation of dogs and cats are likely to promote the establishment of tick vectors and disease in new areas and countries (Boozer and Macintire 2003; Shaw and Irwin 2001; Trotz-Williams and Trees 2003).

The increased occurrence in temperate regions may be linked to the establishment of ticks in previously non-enzootic regions and increasing international pet trade, or to the advancement of molecular diagnostic techniques leading to improved diagnostic capacity, or possibly both (Shaw et al. 2001).

*B. canis vogeli* was the only *Babesia* spp. regarded as endemic in dogs in Australia until 2001 when three dogs infected with *B. gibsoni* were detected in Victoria (Muhlnickel et al. 2002). The dogs lived in the same household and were traced to contact with a single import from the United States. As the infected dogs were American pit bull terriers and no ticks were detected on the premises, it was concluded that the three dogs were most likely infected horizontally through biting and that the detection did not represent establishment of the disease in Australia. A further three cases were confirmed in Victoria in 2004, all of which were linked to the animals in the first isolation (Jubb 2004).

A subsequent study conducted in Victoria in two locations in 2004 and 2005 demonstrated that 14 American pit bull terriers were positive for *B. gibsoni* and that biting was likely to have been the mode of transmission (Jefferies et al. 2007).

*Rhipicephalus sanguineus* (brown dog tick) is the recognised vector for *B. canis vogeli* in dogs (Battsetseg et al. 2002). *Haemaphysalis longicornis* (the bush tick) is considered a likely vector for *B. gibsoni* in Australia.

#### **Global distribution**

The most pathogenic strain of canine babesiosis, *B. canis rossi*, occurs on the African continent with reported occurrence in Nigeria, the Republic of South Africa and Sudan. The associated tick vector, *Haemaphysalis elliptica*, occurs widely across the African mainland. Other *Babesia* spp. – *B. canis vogeli* and *B. gibsoni* – have also been reported in the Republic of South Africa (Irwin 2010).

*B. canis canis* is endemic in central and southern Europe. *B. canis* infection associated with the establishment of *D. reticulatus* ticks has also been reported in previously piroplasmid-free European countries including Belgium, the Netherlands and Switzerland (Matjila et al. 2005). The United Kingdom was generally considered to be free from piroplasmid parasites although tick vectors are present (Trees and Shaw 1999). In 2006, a dog that had never travelled outside the United Kingdom was diagnosed with babesiosis caused by a species closely related to *B. canis vogeli* suggesting piroplasmid parasite establishment in tick vectors in the United Kingdom (Holm et al. 2006).

*B. canis vogeli* is found in the Middle East, North Africa, Europe, Asia, and Australia.

A summary of piroplasmid parasites isolated from dogs and cats and their known geographical distribution is provided in Table 5.

#### 4.15.2 Technical information

##### Epidemiology

*Babesia* and *Theileria* spp. are intra-erythrocytic tick-borne parasites of the canidae and to a lesser extent, the felidae. They are transmitted to vertebrate hosts via the saliva of tick vectors, which have ingested infected erythrocytes. Needles containing infected blood can transmit parasites inadvertently during blood transfusions or deliberately during experimental studies.

Dogs that have recovered from piroplasmosis remain subclinically infected and may relapse or serve as point sources for the further spread of disease (Birkenheuer et al. 1999).

Transmission to ticks via ingestion of blood from an infected host can occur at low levels of parasitaemia (Chauvin et al. 2009).

*Babesia* and *Theileria* spp. are transmitted trans-stadially (de Waal and van Heerden 2004; Ikadai et al. 2007). Transovarial transmission occurs in ticks infected with *Babesia* spp., but is not thought to occur with *Theileria* spp. (Mehlhorn and Schein 1998).

Vertical transmission, blood-borne transmission and possible transmission via bite wounds have been reported for *B. gibsoni* (Jefferies et al. 2007; Jubb 2004). Infected pregnant bitches can spread *Babesia* spp. to their unborn puppies. For example, *B. gibsoni* and *B. canis* have been found in puppies as young as 36 hours old (Boozer and Macintire 2003; Fukumoto et al. 2005). Transplacental transmission resulting in a fading puppy syndrome, and infection as a result of blood transfusion has been reported with *B. canis vogeli* (Irwin 2003; Taboada and Lobetti 2006).

Piroplasmid parasites are largely host-specific, which may reflect the host preference of tick vectors, although parasitaemia has been reported in atypical host species. *B. canis* subsp. have rarely been reported in cats; typically in association with co-existing infection with an immunosuppressive agent (e.g. feline immunodeficiency virus) (Baneth et al. 2004; Criado-Fornelio et al. 2003).

The three subspecies of *B. canis* are considered to be vector-specific. The recognised vectors are *R. sanguineus* for *B. canis vogeli*, *D. reticulatus* for *B. canis canis* (Goodfellow and Shaw 2005; Hauschild and Schein 1996; Uilenberg et al. 1989) and *H. elliptica* for *B. canis rossi* (Apanaskevich et al. 2007). Knowledge of vectors involved in the transmission of some virulent species (e.g. *T. annae*, *B. conradae*) remains uncertain.

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

Table 5 Summary of piroplasmid spp. reported in dogs and cats

Species	Host	Distribution	Tick vector <sup>b</sup>	Virulence
<i>B. canis canis</i>	Dog	Central and Southern Europe	<i>Dermacentor reticulatus</i>	Moderate to severe <sup>d</sup>
<i>B. canis canis</i>	Cat	Portugal and Spain	Unknown	Unknown
<i>B. canis rossi</i>	Dog	Nigeria, Republic of South Africa, Sudan	<i>Haemaphysalis elliptica</i>	Severe <sup>d</sup>
<i>B. canis vogeli</i>	Dog	Australia, Africa, Argentina, Brazil, Europe, Japan, United States	<i>Rhipicephalus sanguineus</i>	Mild to moderate <sup>d</sup>
<i>B. canis presentii</i>	Cat	Israel	Unknown	Unknown
<i>B. gibsoni</i>	Dog	Australia, Asia, Europe, Middle East, North and East Africa, United States (northern)	Possibly <i>Haemaphysalis longicornis</i> , <i>H. bispinosa</i> and <i>R. sanguineus</i>	Moderate to severe <sup>d</sup>
<i>B. conradae</i>	Dog	United States (California)	Unknown	Moderate to severe <sup>d</sup>
<i>T. annae</i>	Dog	Spain	Possibly <i>Ixodes hexagonus</i>	Moderate to severe <sup>d</sup>
<i>T. annae</i>	Cat	Portugal	Unknown	Unknown
<i>T. equi</i>	Dog	Spain	Unknown	Usually subclinical
<i>B. cati</i>	Indian wild cat	India, Republic of South Africa	Unknown	Mild to moderate
<i>B. felis</i>	Cat	Africa	Unknown	Mild to moderate
<i>B. herpailurfi</i>	Wild felids	Africa	Unknown	Usually subclinical
<i>B. leo</i> <sup>c</sup>	Wild felids	Southern Africa	Unknown	Unknown
<i>B. pantherae</i> <sup>c</sup>	Wild felids	Africa	Unknown	Usually subclinical

*B.* = *Babesia*; *T.* = *Theileria*

<sup>a</sup> Large = 2 µm x 5 µm; small = 1 µm x 2.25–2.5 µm

<sup>b</sup> Tick species can vary with geographical location

<sup>c</sup> Detected in wild felids; experimental transmission in domestic cats

<sup>d</sup> Adapted from: Irwin (2005) and Schoeman (2008)

### Pathogenesis

The pathogenicity of piroplasmid parasites is determined primarily by the species and strain of parasite. Host factors such as age and the immunological response are also important. Clinical disease tends to be more severe in puppies than adult dogs. *B. canis rossi* is considered the most virulent form of canine babesiosis, while the virulence of *B. gibsoni*, *B. canis canis*, *B. conradae* and *T. annae* in dogs is described as moderate to severe (Irwin 2005).

## Clinical signs

The incubation period of canine piroplasmosis varies from 10–28 days. Female ticks feed on their host for about one week and are likely to have left the host by the time clinical signs of disease develop (Schoeman 2008).

Peracute onset of clinical signs is a feature of *B. canis rossi* infection with signs that include collapse, hypotensive shock, cyanotic mucous membranes, tachycardia, severe intravascular haemolysis, widespread organ dysfunction and death. There are also acute and chronic forms of the disease, which may vary in clinical presentation.

Signs of infection with other *Babesia* spp. or *Theileria* spp. generally reflect a haemolytic disorder and are less severe than those observed due to infection with *B. canis rossi*. However, infection with *B. canis canis*, *B. conradae* and *T. annae* can cause severe clinical disease with high mortality rates in untreated dogs.

Clinical signs of acute lethargy, anorexia, pyrexia, icterus and haematuria have been reported in cats from which *B. canis presentii* was isolated (Baneth et al 2004). *B. canis canis* has only been reported in three cats: two immunocompromised, subclinically infected cats from Portugal (one cat also had a concurrent *T. annae* infection) and one cat from Spain (Criado-Fornelio et al. 2003). The cat from Spain showed clinical signs consistent with *Babesia* spp. infection.

Infection of cats with *B. felis* is often subclinical and persistent (Irwin 2005).

## Diagnosis

A number of techniques may be used to diagnose piroplasmosis. These include microscopic examination of blood smears, serology—e.g. indirect fluorescent antibody test (IFAT) or enzyme-linked immunosorbent assay (ELISA)—and polymerase chain reaction (PCR).

Serological testing has limitations in the diagnosis of acute infection because seroconversion may be delayed until 14 days after exposure (Comazzi et al. 1999). Also, serology cannot be used reliably in domestic cats because reference intervals for babesiosis-related serological titres have not been established. Poor specificity is attributed to cross-reactions between *Babesia* spp. and other apicomplexan parasites unless species-specific antigens are used (Aboqe et al. 2007).

PCR is both sensitive and specific for disease diagnosis as it can distinguish between different piroplasmid spp. However, this test is restricted to relatively few laboratories and due to transient or low levels of parasitaemia, it may take several tests on peripheral blood to obtain a positive result in a subclinically infected dog (Irwin 2009).

## Treatment

There are few drugs that have been shown to be both safe (without side effects) and effective in eliminating piroplasmid parasites. Dogs treated with specific antibabesial drugs are unlikely to be cured of their infection (Irwin 2010).

A single treatment with imidocarb dipropionate at a rate of 7.5 mg/kg body weight by subcutaneous injection is the recommended single-dose treatment against infection with the large (2 µm x 5 µm) *Babesia* piroplasms (*B. canis* subspp.). Alternatively, repeat treatments with imidocarb dipropionate at rates ranging from 5.0 to 6.6 mg/kg body weight by subcutaneous or intramuscular injection administered two weeks apart have been recommended as effective (Penzhorn et al. 1995; Taboada and Lobetti 2006).

Single-dose administration of imidocarb dipropionate at a rate of 6.6 mg/kg body weight was shown to provide prophylactic protection for two weeks against *B. canis* (assumed *B. canis canis*) infection in a small study (Vercammen et al. 1996a). This contrasted with the manufacturer's claim of three to four weeks protection against infection. Side effects such as pain at the injection site, vomiting, diarrhoea and muscle tremors can occur. A dose of 10 mg/kg body weight is toxic (Birkenheuer et al. 1999).

Imidocarb dipropionate was reported to be less effective in dogs infected with small (1 µm x 2.25–2.5 µm) piroplasmid parasites, such as *T. annae* (García 2006).

Doxycycline and doxycycline/imidocarb combinations have been used for prophylaxis but have not necessarily prevented infection (Vercammen et al. 1996b).

Drugs used to treat experimental infections of *B. felis* in domestic cats have given variable results (Penzhorn et al. 2000; Potgeiter 1981). It is not uncommon for chronic (or repeated) chemotherapy to be required to treat feline babesiosis. Cats have been treated with primaquine phosphate but it does not eliminate the infection (Penzhorn et al. 2000).

### **Vaccination**

Vaccines developed using soluble parasite antigens (SPA) derived from cell culture have been effective in providing immunity against *B. canis* parasites. Effective protection against challenge by homologous strains of *B. canis canis* has been demonstrated but not protection against heterologous challenge by other *B. canis* subspp (Irwin 2003).

A vaccine that contain SPA of *B. canis canis* and *B. canis rossii* has been reported to provide up to six months protection against disease due to heterologous subspecies of *B. canis*, including the severe clinical effects associated with infection by *B. canis rossii* (Schetters et al. 2007). Vaccination does not provide a sterile immunity as infection with *Babesia* spp. still occurs (Schetters et al. 2007).

### **Vector management**

The best prevention for canine and feline piroplasmosis is tick control. Effective tick prevention depends on regular treatment of dogs and cats with a suitable parasiticide in accordance with the manufacturer's recommendations. Kennels should be frequently treated since some ticks (e.g. *R. sanguineus*) can survive off the host for prolonged periods. For dogs and cats visiting tick-endemic regions, tick prevention should be practised. General tick prevention measures should include

preventive treatment with an appropriate acaricide and a daily search of animals for the presence of ticks, and removal of any ticks detected.

#### 4.15.3 Current biosecurity measures

For dogs that have ever resided in Africa, the dog must be treated for *B. canis rossi* within 28 days of export:

##### Treatment

- Option 1: A government-approved veterinarian must treat the dog once with imidocarb dipropionate at a rate of 7.5 mg/kg body weight by subcutaneous injection.

OR

- Option 2: A government-approved veterinarian must treat the dog twice with imidocarb dipropionate at a rate of 6.6 mg/kg body weight by subcutaneous injection given two weeks apart.

##### Vector management

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a parasiticide effective against ticks on contact; the parasiticide must be applied in accordance with the manufacturer's recommendations by a government-approved veterinarian.
- Within four days immediately before export, dogs must be treated with a parasiticide effective against ticks on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks.

#### 4.15.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by piroplasmiasis:

- *B. canis canis*, *B. canis rossi*, *B. conradae* and *T. annae* are exotic to Australia and are primarily parasites of dogs. *B. canis rossi* is recognised as being the most virulent species, occurs on the African continent and causes acute fatal haemolytic disease.
- There have been rare reports of infection with *B. canis canis* and *T. annae* in apparently immunocompromised cats; therefore, cats appear to be atypical hosts.
- A subclinical carrier state occurs in dogs that recover from infection.
- The parasites are tick-borne and largely vector-specific. Infected ticks are a key potential source of infection.

- The known vectors of *B. canis canis* (*D. reticulatus*) and *B. canis rossi* (*H. elliptica*) are not present in Australia; neither is *I. hexagonus*, the possible tick vector for *T. annae*. A known vector for *B. conradae* has not been identified.
- A number of tick species in the genus *Haemaphysalis* are present in Australia; their ability to act as a competent vector for *B. canis rossi* is unknown.
- Establishment of infection in competent tick vectors would be expected to enable generalised establishment or spread.
- Exposure of naïve dogs may also occur iatrogenically (e.g. via blood transfusion from an infected dog).
- Modes of direct (non-vector) transmission (e.g. via biting) appear to exist for some piroplasmid parasites (e.g. *B. gibsoni*), but have not been reported in association with other piroplasms. Direct transmission may enable localised establishment and spread within discrete dog populations.

#### 4.15.5 Conclusion

Based on the preceding key points it was concluded that risk management measures for piroplasmidosis caused by *B. canis rossi* continue to be warranted for dogs. In addition, it was concluded that risk management measures for piroplasmidosis caused by *B. canis rossi* are not warranted for cats, or for dog or cat semen.

The following biosecurity measures would provide appropriate risk management for dogs.

##### Pre-export measures (dogs)

As for current pre-export measures for dogs that have ever resided in Africa.

##### *Vector management*

- As for pre-export measures for external parasite control (see 4.13.5).

##### Post-arrival measures (dogs)

##### *Vector management*

- As for post-arrival measures for external parasite control (see 4.13.5).
- To manage the biosecurity risks of piroplasmidosis, dogs may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### References

Aboge GO, Jia H, Terkawi MA, Goo Y, Kuriki K, Nishikawa Y, Igarashi I, Suzuki H, Xuan X (2007). A novel 57-kDa merozoite protein of *Babesia gibsoni* is a prospective antigen for diagnosis and serosurvey of canine babesiosis by enzyme-linked immunosorbent assay. *Veterinary Parasitology* 149:85–94.

Apanaskevich DA, Horak IG, Camicas J-L (2007). Redescription of *Haemaphysalis* (*Rhipistoma*) *elliptica* (Koch, 1844), an old taxon of the *Haemaphysalis* (*Rhipistoma*) *leachi* group from East and southern Africa, and of *Haemaphysalis* (*Rhipistoma*) *leachi* (Audouin, 1826) (Ixodida, Ixodidae). *Onderstepoort Journal of Veterinary Research* 74:181–208.

Baneth G, Kenny MJ, Tasker S, Anug Y, Shkap V, Levy A, Shaw SE (2004). Infection with a proposed new subspecies of *Babesia canis*, *Babesia canis* subsp. *presentii*, in domestic cats. *Journal of Clinical Microbiology* 42:99–105.

Battsetseg B, Lucero S, Xuan X, Claveria FG, Inoue N, Alhassan A, Kanno T, Igarashi I, Nagasawa H, Mikami T, Fujisaki K (2002). Detection of natural infection of *Boophilus microplus* with *Babesia equi* and *Babesia caballi* in Brazilian horses using nested polymerase chain reaction. *Veterinary Parasitology* 107:351–357.

Birkenheuer AJ, Levy MG, Savary KC, Gager RB, Breitschwerdt EB (1999). *Babesia gibsoni* infections in dogs from North Carolina. *Journal of the American Animal Hospital Association* 35:125–128.

Boozer AL, Macintire DK (2003). Canine babesiosis. *Veterinary Clinics of North America: Small Animal Practice* 33:885–904.

Chauvin A, Moreau E, Bonnet S, Plantard O, Malandrin L (2009). *Babesia* and its hosts: adaptation to long-lasting interactions as a way to achieve efficient transmission. *Veterinary Research* 40:1–18.

Comazzi S, Paltrinieri S, Manfredi MT, Agnes F (1999). Diagnosis of canine babesiosis by Percoll gradient separation of parasitized erythrocytes. *Journal of Veterinary Diagnostic Investigation* 11:102–104.

Criado-Fornelio A, Martinez-Marcos A, Buling-Saraña A, Barba-Carretero JC (2003). Presence of *Mycoplasma haemofelis*, *Mycoplasma haemominutum* and piroplasmids in cats from southern Europe: a molecular study. *Veterinary Microbiology* 93:307–317.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

de Waal DT, van Heerden J (2004). Equine piroplasmosis. In *Infectious diseases of livestock*, Coetzer JAW, Tustin RC (eds.), Oxford University Press, Oxford, pp. 425–434.

Fukumoto S, Suzuki H, Igarashi I, Xuan X (2005). Fatal experimental transplacental *Babesia gibsoni* infections in dogs. *International Journal for Parasitology* 35:1031–1035.

García ATC (2006). Piroplasma infection in dogs in northern Spain. *Veterinary Parasitology* 138:97–102.

- Goodfellow M, Shaw S (2005). Exotic diseases of dogs and cats at risk of importation to Ireland. *Irish Veterinary Journal* 58:271–277.
- Gothe R, Wegerdt S (1991), Die Babesiosen des hundes in Deutschland: epidemiologische fallanalysen (*Babesia* infections of dogs in Germany: epidemiological surveys). *Tierärztliche Praxis* 19:17–173.
- Hauschild S, Schein E (1996). Zur artspezifität von *Babesia canis* (The subspecies specificity of *Babesia canis*). *Berliner und Münchener Tierärztliche Wochenschrift* 109:216–219. (Abstract only)
- Holm LP, Kerr MG, Trees AJ, McGarry JW, Munro ER, Shaw SE (2006). Fatal babesiosis in an untravelled British dog. *The Veterinary Record* 159:179–180.
- Ikadai H, Sasaki M, Ishida H, Matsuu A, Igarashi I, Fujisaki K, Oyamada T (2007). Molecular evidence of *Babesia equi* transmission in *Haemaphysalis longicornis*. *The American Journal of Tropical Medicine and Hygiene* 76:694–697.
- Irwin PJ (2002). Companion animal parasitology: a clinical perspective. *International Journal for Parasitology* 32:581–593.
- Irwin PJ (2003). Babesiosis in dogs and cats. The 28th Congress of the World Small Animal Veterinary Association Proceedings Online.  
<http://www.vin.com/proceedings/Proceedings.plx?CID=WSAVA2003&Category=1008&PID=6634&O=Generic> (Accessed 13 January 2009)
- Irwin PJ (2005). Babesiosis and cytauxzoonosis. In *Arthropod-borne infectious diseases of the dog and cat* Shaw S, Day MJ (eds), Manson, London, pp. 63–77.
- Irwin PJ (2009). Canine babesiosis: from molecular taxonomy to control. *Parasites and Vectors* 2: S4.
- Irwin PJ (2010). Canine Babesiosis. *Veterinary Clinics of North America: Small Animal Practice* 40:1141–1156.
- Jefferies R, Ryan U, Jardine J, Broughton DK, Robertson ID, Irwin PJ (2007). Blood, bull terriers and babesiosis: further evidence for direct transmission of *Babesia gibsoni* in dogs. *Australian Veterinary Journal* 85:459–463.
- Jubb T (2004). *Babesia gibsoni* in a pit bull terrier. *Animal Health Surveillance: Quarterly Report* 9:14.
- Kahl O, Janetzki C, Gray JS, Stein J, Bauch RJ (1992). Tick infection rates with *Borrelia: Ixodes ricinus* versus *Haemaphysalis concinna* and *Dermacentor reticulatus* in two locations in eastern Germany. *Medical and Veterinary Entomology* 6:363–366.
- Matjila TP, Nijhof AM, Taoufik A, Houwers D, Teske E, Penzhorn BL, de Lange T, Jongejan F (2005). Autochthonous canine babesiosis in The Netherlands. *Veterinary Parasitology* 131:23–29.

- Mehlhorn H, Schein E (1998). Redescription of *Babesia equi* Laveran 1901, as *Theileria equi* Mehlhorn, Schein 1998. *Parasitology Research* 84:467–475.
- Muhlnickel CJ, Jefferies R, Morgan-Ryan UM, Irwin PJ (2002). *Babesia gibsoni* infection in three dogs in Victoria. *Australian Veterinary Journal* 80:606–610.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Penzhorn BL, Lewis BD, de Waal DT, López Rebollar LM (1995). Sterilisation of *Babesia canis* infections by imidocarb alone or in combination with diminazene. *Journal of the South African Veterinary Association* 66:157–159.
- Penzhorn BL, Lewis BD, López-Rebollar LM, Swan GE (2000). Screening of five drugs for efficacy against *Babesia felis* in experimentally infected cats. *Journal of the South African Veterinary Association* 71:53–57.
- Perkins SCB (2000). *Babesia* and the pet travel scheme. *The Veterinary Record* 147:460.
- Potgeiter FT (1981). Chemotherapy of *Babesia felis* infection: efficacy of certain drugs. *Journal of the South African Veterinary Association* 52:289–293.
- Schettters TPM, Kleuskens J, Carcy B, Gorenflot A, Vermeulen A (2007). Vaccination against large *Babesia* species from dogs. *Parassitologia* 49:13–17.
- Schoeman JP (2008). Canine babesiosis: an update. The 33rd Congress of the World Small Animal Veterinary Association Proceedings [Online]. <http://www.vin.com/proceedings/Proceedings.plx?CID=WSAVA2008&Category=&PID=24062&O=Generic> (accessed 22 June 2010)
- Shaw SE, Day MJ, Birtles R, Breitschwerdt EB (2001) Tick-borne infectious diseases of dogs. *Trends in Parasitology* 17: 74-80.
- Shaw SE, Irwin PJ (2001). The consequences of tick infestation in dogs and cats. *WALTHAM Focus* 11:16–23.
- Taboada J, Lobetti R (2006). Babesiosis. In *Infectious diseases of the dog and cat*, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 722-736.
- Trees A, Shaw S (1999). Imported diseases in small animals. *In Practice* 21:482–491.
- Trotz-Williams LA, Trees AJ (2003). Systematic review of the distribution of the major vector-borne parasitic infections in dogs and cats in Europe. *The Veterinary Record* 152:97–105.
- Uilenberg G, Franssen FFJ, Perié NM, Spanjer AAM (1989). Three groups of *Babesia canis* distinguished and a proposal for nomenclature. *Veterinary Quarterly* 11:33–40.

Vercammen F, de Deken R, Maes L (1996a). Prophylactic activity of imidocarb against experimental infection with *Babesia canis*. *Veterinary Parasitology* 63: 95–198.

Vercammen F, de Deken R, Maes L (1996b). Prophylactic treatment of experimental canine babesiosis (*Babesia canis*) with doxycycline. *Veterinary Parasitology* 66:251–255.

## 4.16 Rabies

### 4.16.1 Background

Rabies is a zoonotic, almost invariably fatal viral encephalomyelitis caused by rabies virus genotype 1 (hereafter referred to as rabies virus) of the genus *Lyssavirus* in the family *Rhabdoviridae* (ICTV 2009). All lyssaviruses can cause fatal encephalitic disease in mammals. Rabies virus causes at least 55 000 human deaths each year, mainly in Africa and Asia (Knobel et al. 2005). Most human infections in these regions result from exposure to infected dogs (Bingham 2005; Cliquet and Picard-Meyer 2004).

Reservoir host species for rabies virus are mammalian species from the orders Carnivora or Chiroptera (bats) for which particular biotypes of the virus have adapted; for example, the rabies virus that has adapted to the domestic dog. Spill over infection to other domestic and wild mammals can occur from the maintenance host species.

Where rabies virus is endemic, biotypes may be maintained in domestic and/or wild animal species. In urban areas domesticated dogs, and to a minor extent cats, play a role in disease spread.

Due to the serious public health implications, rabies is listed in Australia's Emergency Animal Disease Response Agreement as a Category 1 disease. If the emergency agreement is invoked, the government will provide 100% of the funding necessary for an emergency response.

Pre-exposure vaccines are available for humans, all domestic animal species and many species of wildlife (Bowen-Davies and Lowings 2000).

Rabies is an OIE-listed multiple species disease (OIE 2012) and is nationally notifiable in Australia (DAFF 2011).

Before 1993, only certain designated rabies-free countries were eligible to export dogs and cats to Australia. The *Review of quarantine policy for dogs and cats, with particular reference to rabies* (BRS review) (BRS 1993):

- provided an extensive description of rabies, including its epidemiology, ecology, pathogenesis, transmission and the efficacy of vaccination as a control measure
- recommended importation of dogs and cats from countries in which rabies occur be permitted provided the proposed quarantine requirements were met

- proposed a policy for importing dogs and cats from rabies-free countries, and a policy for importing dogs and cats from countries in which rabies occurs.

This section updates technical information about rabies virus based on a review of the scientific literature, including risk factors relevant to the introduction, establishment and spread of rabies virus via imported dogs and cats.

### **Global distribution**

Rabies occurs in most countries of the world except for Australia, Denmark Ireland, Japan, New Zealand, Sweden, the United Kingdom and many small island nations. In contrast, other lyssaviruses have limited distribution (Rupprecht et al. 2001). The geographical distribution of rabies, based on international reporting of human and domestic animal rabies cases, is considered to be unreliable unless free-ranging wildlife rabies cases are also included (Rupprecht et al. 2001).

#### *Africa*

Rabies virus is endemic in many countries in Africa, with dogs acting as a reservoir host and principal vector (Cliquet and Picard-Meyer 2004). In southern Africa, rabies virus is maintained and transmitted by different host species (see Table 6), and remains a serious public and animal health threat (Ngoepe et al. 2009).

#### *Asia*

The domestic dog is considered the most significant reservoir of rabies virus throughout Asia, with wildlife believed to play a lesser role (Rupprecht et al. 2001). The disease is generally not notifiable and largely uncontrolled in most Asian countries, and thousands of human cases are reported annually (Cliquet and Picard-Meyer 2004). An outbreak of canine rabies on the Indonesian island of Bali in 2008 resulted in 157 officially reported deaths in 2011 (most recent OIE data) (Promed Mail 2013).

Information about prevalence, incidence, geographical distribution and biotypes present in this region is limited. Singapore, Japan and Hong Kong have reportedly been free from rabies since 1953, 1956 and 1987, respectively.

#### *Australia*

There is no evidence of rabies virus being present in Australia. Rabies was last reported in animals in Australia in 1867 (Animal Health Australia 2011a), though there are occasional imported cases in humans who were infected overseas.

Australian bat lyssavirus (ABL) was discovered in 1995. ABL isolates represent a new Lyssavirus spp., genotype 7 (Gould et al. 1998). No other lyssaviruses have been identified in Australia.

#### *Central and South America*

Rabies virus is endemic in most countries in Central and South America, including the Caribbean islands (Cliquet and Picard-Meyer 2004). Rabies virus circulates as distinct biotypes in dogs and bats. Spill over of both biotypes to other hosts occurs.

## Europe

Rabies virus in domestic animals and wild carnivores is rare in western Europe. In 2005 and 2006, a few cases were reported in foxes in Germany (Wandeler 2008). Vaccination via oral baits was adopted as a key method of rabies control in reservoir populations of dogs and wildlife (Cliquet et al. 2007) and enabled the eradication of rabies virus from the red fox population throughout much of Europe (Wilsmore et al 2006).

In eastern Europe, rabies virus infection is reported in small numbers of dogs, bats and other wildlife species (Matouch 2008). The reservoir host is the red fox (*Vulpes vulpes*) with the raccoon dog (*Nyctereutes procyonoides*) being another significant host (Cliquet and Picard-Meyer 2004). Turkey is the only European country where dogs are the maintenance host of rabies virus (Cliquet and Aubert 2004).

## North America

Molecular, phylogenetic and epidemiological evidence indicate that the domestic canine rabies virus biotype is no longer endemic in domestic dogs in the United States (Velasco-Villa et al. 2008). However, several rabies virus biotypes are maintained in mammalian wildlife reservoirs, predominantly raccoons (*Procyon lotor*), skunks (*Mephitis mephitis*), several species of bats, red foxes (*Vulpes vulpes*) and gray foxes (*Urocyon cinereoargenteus*) (Cliquet and Picard-Meyer 2004). The causal agent of rabies cases in domestic dogs and cats is typically found to be the common rabies virus biotype that occurs in local wildlife (McQuiston et al. 2001).

In 2011, just over 5% of all reported rabies cases in the United States were in domestic dogs and cats (Blanton et al. 2012).

### 4.16.2 Technical information

#### Taxonomy

Lyssaviruses are categorized into eleven species—of which seven are characterised into genotypes: rabies virus (genotype 1), Lagos bat virus (genotype 2), Mokola virus (genotype 3), Duvenhage virus (genotype 4), European bat lyssaviruses 1 and 2 (genotypes 5 and 6) and Australian bat lyssavirus (genotype 7) (ICTV 2009). Four additional species (Aravan virus, Irkut virus, Khujand virus, and West Caucasian bat virus), not yet characterised into genotypes, were recently identified. Current research suggests Shimoni bat virus to be a potential new species in the Lyssavirus genus (Kuzmin et al. 2010). The taxonomy of lyssaviruses and their geographic range is summarised in Table 6. Since the BRS review (BRS 1993), rabies-related viruses in invertebrate hosts are no longer in the Lyssavirus genus.

Rabies virus has distinctive biotypes adapted to single maintenance host species in which infection and transmission is highly efficient. Infection of other species, referred to as spill over hosts, may also occur but these hosts may be inefficient as vectors or may not be numerous enough to maintain a transmission cycle. All genotypes, except genotype 2 (Lagos bat virus), cause human and/or animal deaths (Cliquet and Picard-Meyer 2004).

### Agent properties

Rabies virus is fragile and does not persist for long outside a host animal. The virus is inactivated by ultraviolet radiation, many lipid solvents, most detergents and disinfectants, sunlight, heat, and extremes of pH (Rupprecht et al. 2001; Swanepoel 1994).

### Host range

All warm-blooded animals are considered susceptible to rabies. However, susceptible animals are classified as either maintenance or spill over hosts. Maintenance hosts are those that primarily sustain, and are highly susceptible to, a given rabies virus biotype (Table 7). Spill over hosts are species that can be infected by the biotype, but are epidemiologically insignificant in sustaining an epidemic. Spill over hosts are often, but not always, dead-end hosts. They may transmit infection to other hosts, although such events are uncommon. Spill over hosts include humans and other primates, cats, horses, cattle, sheep, pigs, birds and some wildlife species.

Only a few species within the orders Carnivora and Chiroptera are maintenance hosts of rabies virus (Table 6) (Rupprecht et al. 2002). Of the Carnivora, these include canids (e.g. coyotes, dogs, foxes and raccoon dogs), herpestids/viverrids (mongooses), mephitids (skunks) and procyonids (raccoons) (Bowen-Davies and Lowings 2000; Niezgodna et al. 2002).

Some species show more resistance to rabies infection than others. Species regarded as moderately susceptible include felids, mustelids (badgers, ferrets and minks), primates and ungulates (Niezgodna et al. 2002). Cats are effective vectors for transmission; however, there are no known rabies virus biotypes adapted to felids (Rupprecht et al. 2006).

Potential wild or feral hosts (foxes, wild dogs and feral cats) are widespread in Australia. In some places, foxes and wild dogs are in sufficient densities to become maintenance hosts for rabies virus. It was estimated that foxes (*Vulpes vulpes*) and wild dogs inhabit about 76% and 83% of Australia, respectively (West 2008). Feral cats are potential spill over hosts.

Insectivores, lagomorphs, marsupials, monotremes and rodents are regarded as species at low risk of, or less susceptible to, rabies virus infection (Niezgodna et al. 2002). The susceptibility of Australian native mammals is unknown. However, it is likely that if infected, Australian native mammals in the orders Chiroptera, Carnivora (dingoes) and Dasyuromorphia (antechinus, dunnart, quoll, Tasmanian devil) would contribute to the maintenance of a wildlife cycle of rabies virus.

Birds can be infected with rabies virus experimentally, but the virus is restricted to central nervous tissue and is apparently overcome by host defences. There are no substantiated reports of natural cases of rabies in birds (Niezgodna et al. 2002).

Table 6 Species in the Lyssavirus genus and their host species

Genotype	Species	Abbreviation <sup>a</sup>	Ecology	Natural hosts	Spill over hosts reported
Genotype 1	Rabies virus	RABV	Widespread	Carnivores; Bats in the Americas	All mammals
Genotype 2	Lagos bat virus	LBV	Sub-Saharan Africa	Frugivorous and insectivorous bats	Domestic dog and cat; experimentally in mouse, water mongoose and monkey
Genotype 3	Mokola virus	MOKV	Sub-Saharan Africa	Not known. Was isolated from shrews. Insectivorous rodents possible	Humans, domestic dog and cat, shrews
Genotype 4	Duvenhage virus	DUVV	Southern Africa	Insectivorous bats	Humans
Genotype 5	European bat lyssavirus 1	EBL 1 (a and b)	Europe	Insectivorous bats ( <i>Eptesicus serotinus</i> and other <i>Eptesicus</i> spp.)	Humans, cats, sheep, stone martens
Genotype 6	European bat lyssavirus 2	EBL 2	Europe	Insectivorous bats ( <i>Myotis</i> spp.)	Humans, experimentally; also , domestic cat, fox and mouse
Genotype 7	Australian bat lyssavirus	ABL	Australia	Frugivorous and insectivorous bats ( <i>Megachiroptera</i> and <i>Microchiroptera</i> spp.)	Humans, experimentally; also domestic cat and mouse
Undesignated Proposed new genotype	Aravan virus	ARAV	Central Asia	Lesser mouse-eared bat ( <i>Myotis blythi</i> )	Fruit bats, insectivorous bats, experimentally in <i>Pipistrellus pipistrellus</i> ,
	Khujand virus	KHUV	Central Asia	Insectivorous bats ( <i>Myotis mystacinus</i> )	Fruit bats in Thailand
	Irkut virus	IRKV	East Siberia	Insectivorous bats ( <i>Murina leucogaster</i> )	Fruit bats in Thailand
	West Caucasian bat virus	WCBV	Caucasia	Insectivorous bats ( <i>Miniopterus schreibersi</i> )	

<sup>a</sup> Abbreviations from ICTV 2009

Sources: Arai et al. (2003); Bowen-Davies and Lowings (2000); Kuzmin et al. (2008); McColl et al. (2000); WHO (2006a)

## Epidemiology

Although all warm-blooded animals are susceptible to rabies, many are considered dead-end hosts and relatively few species are effective vectors and reservoirs. In reservoir host species, infection can result from small infectious doses and typically leads to an increase in biting behaviour. A significant viral load is usually shed in saliva which facilitates disease spread. In contrast, infection of spill over hosts by a given biotype usually requires a larger infectious dose and virus may be excreted in saliva in only small amounts. Consequently, rabies virus is readily maintained in species to which the biotype is adapted, but infection does not spread readily to other species (Bowen-Davies and Lowings 2000).

The rabies virus biotype determines the epidemiology of the disease, such as reservoir host species. The biotype's geographical distribution is directly related to that of the reservoir host (Table 7). Behavioural traits can also influence virus transmission and disease occurrence. Territorial behaviour and size are also risk factors in transmission of rabies virus (Bowen-Davies and Lowings 2000).

Table 7 Known reservoir hosts of rabies virus biotypes

Maintenance host	Geographic location
Domestic dog ( <i>Canis familiaris</i> )	Africa, Asia, the Caribbean and Central and South America
Red fox ( <i>Vulpes vulpes</i> )	Europe, Middle East, North America and the countries of the former Soviet Union
Raccoon dog ( <i>Nyctereutes procyonoides</i> )	Eastern Europe
Arctic fox ( <i>Alopex lagopus</i> )	Arctic Circle
Common raccoon ( <i>Procyon lotor</i> )	North America
Striped skunk ( <i>Mephitis mephitis</i> )	North America
Coyote ( <i>Canis latrans</i> )	North America
Gray fox ( <i>Urocyon cinereoargenteus</i> )	North America
Small Indian mongoose ( <i>Herpestes auropunctatus</i> )	the Caribbean
Black-backed jackal ( <i>Canis mesomelas</i> )	Southern Africa
Bat-eared fox ( <i>Otocyon megalotis</i> )	Republic of South Africa
Yellow mongoose ( <i>Cynictis penicillata</i> )	Southern Africa
Grey wolf ( <i>Canis lupus</i> )	Eastern Europe, Mexico, Middle East and North America
Haematophagous bats	the Caribbean and South America
Insectivorous bats	Africa, the Caribbean, and Central, North and South America

Source: Adapted from Bowen-Davies and Lowings (2000)

Despite the public health importance of rabies, little is known about the distribution of rabies virus in dog populations (Bourhy et al. 2008). There are few studies on the incidence of rabies virus infection in unvaccinated dog populations, while active and/or passive surveillance systems underestimate the incidence of rabies (Kitala et al. 2000). Studies conducted in Chad, Ecuador, Kenya and the Philippines reported annual incidences ranging between 0.04 and 0.86 per cent (Kayali et al. 2003).

## Transmission

Rabies virus is generally transmitted in saliva through the bite of an infected animal (Bingham 2005). Inquisitive species such as cats, cattle and humans are more likely to be bitten than timid species. Territorial species may be more involved in fighting and are therefore more likely to transmit the virus. Small species rarely survive physical attack by a rabid animal, whereas larger species might survive an attack and be more likely to transmit rabies virus before succumbing to the disease (Bowen-Davies and Lowings 2000). The infective period in domestic carnivores can start up to 15 days before the onset of clinical signs (Fekadu et al. 1982; Fekadu 1988; Greene and Rupprecht 2006; OIE 2011b).

Transmission can occur via licking and abrasions contaminated by infected saliva (Rupprecht et al. 2002). Rarely, oral exposure can result in clinical rabies or immunity, depending on the dose of virus (Niezgoda et al. 2002). Indirect transmission can occur but is rare because rabies virus does not readily survive in the environment (Rupprecht et al. 2002). Rabies virus transmission by aerosols has been shown to occur in bat caves and laboratories (Bowen-Davies and Lowings 2000).

### *Dogs*

Dogs can be infected with non-canine biotypes of rabies virus (Velasco-Villa et al. 2008). Even though rabies in dogs is well controlled in the United States, vaccination is still considered important to guard against reintroduction or spill over of rabies virus biotypes from wildlife maintenance hosts to dogs (Krebs et al. 2004; Rupprecht et al. 2006).

There are reports of a subclinical carrier state in the scientific literature (Fekadu et al. 1981, 1983; Fekadu 1988; Fekadu and Baer 1980; Warner et al. 1996). However, these reports could not be verified and a subclinical carrier state is not considered to be an epidemiological feature of rabies virus infection in dogs (Rupprecht et al. 2002; Wu et al. 2009; Zhang et al. 2008).

### *Dog semen*

There is no report of dog semen as a source of natural rabies infection, despite the vast amount of scientific study of rabies virus.

### *Cats*

Despite being effective vectors for transmission, cats (domestic or wild) are not known to be maintenance hosts (Rupprecht et al. 2002). Rabid cats are usually spill over hosts infected with the dominant geographic biotype from wild or domestic maintenance host species. Cats excrete virus in saliva and are therefore able to transmit infection to other animals or humans (Trimarchi et al. 1986).

### *Humans*

Rabies is almost invariably fatal in humans (Greene and Rupprecht 2006).

Most cases of human rabies infection are transmitted by dogs, especially in Africa, Asia and Latin America (Cliquet and Picard-Meyer 2004).

Rabid cats also pose a significant risk to humans, particularly in countries where canine rabies has been controlled but rabies virus biotypes are present in wildlife, such as in North America and Europe (Kihm et al. 1982; Rupprecht et al. 2006). Presumed transmission of bat rabies from bat to cat to human has been reported (Tesoro-Cruz et al. 2008).

#### *Interspecies transmission*

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).

Spill over infections are uncommonly linked to secondary transmission to other animals, but species barriers are not entirely predictable (McQuiston et al. 2001). Dogs appear to be the main vector for interspecies rabies virus transmission (Bourhy et al. 2008). There is molecular and virological evidence that independent rabies endemics occurred in wild terrestrial carnivores (skunks in California and north-central United States, gray foxes in Texas and Arizona, coyotes in Texas and mongooses in Puerto Rico) as a result of spill over of rabies virus from dogs (Velasco-Villa et al. 2008).

Interspecies transmission includes infection of dogs with wildlife rabies virus biotypes. For example, Texas gray fox rabies virus biotype was detected in dogs and coyotes (Velasco-Villa et al. 2008). Maintaining vaccination of dog populations, even with moderate rates of compliance, reduces the likelihood of reintroducing a canine rabies virus biotype by translocation (Rupprecht et al. 2006).

Bats are hosts for six of the seven Lyssavirus genotypes described, including rabies virus (Rupprecht et al. 2002). They are also the natural hosts of the newly discovered species of lyssaviruses. Despite this, transmission of bat lyssaviruses, or rabies virus biotypes maintained in bats, to dogs or cats is unlikely, because only a few spill over infections have been documented in mammals other than bats (Kuzmin et al. 2011).

#### **Pathogenesis**

The site of infection is usually a bite wound. Rapid onset and development of disease is associated with proximity of the wound to the head or neck, and whether it is in an area of high sensory innervation (Baer 1975). Viral replication in muscular and connective tissue may occur at the infection site before virus moves along nerve cell axoplasm to the central nervous system (Jackson 1994; Swanepoel 1994). Spread within the brain is rapid. Rabies virus then spreads from the central nervous system to peripheral nerves and a range of non-neural tissues, including salivary glands. Infection of salivary glands is variable and depends on the stage of infection.

Before entering nerve endings, virus can be exposed to immune effectors, explaining the efficacy of pre-exposure vaccination and post-exposure immunoglobulin treatment in humans (Lafon 2002). If infection is cleared before it enters the central

nervous system, seroconversion may occur without disease and this could explain an abortive infection.

Studies have shown that some virus remains at or near the site of entry for most of the incubation period (Baer and Cleary 1972). The respiratory system, especially the nasal mucosa, may serve as a route of entry for aerosolised virus.

### **Incubation period**

For international movement of mammals, the OIE defines the rabies virus incubation period as up to six months (OIE 2011b).

In natural infections, the incubation period of rabies varies. 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 biotype of the rabies virus involved (Kaplan 1969; Niezgoda et al. 2002). In dogs, the incubation period is generally two to eight weeks but can range from ten days after severe head wounds, to a year or longer in very rare cases (Kaplan 1969; Swanepoel 1994).

A study into a series of naturally contracted rabies infections in cats estimated the median incubation period to be from four to six weeks (Fogelman et al. 1993). One case developed clinical signs 80 days after being bitten by an infected raccoon. However, it is generally agreed that the incubation period in cats is shorter than in dogs (Swanepoel 2004).

Experimental studies in dogs and cats have estimated the rabies incubation periods to be within the range of nine to sixty days (Fekadu et al. 1982; Jones et al. 2005; Soulebot et al. 1981; Tesoro-Cruz et al. 2008; Trimarchi et al. 1986; Vaughn et al. 1963).

The literature strongly supports the OIE definition. Cases in dogs and cats with incubation periods over six months are rare.

### **Clinical signs**

Clinical signs are not pathognomonic, and can vary in domestic or wild animals (Niezgoda et al. 2002). After entering the central nervous system via peripheral nerves, rabies virus causes encephalitis leading to fulminant, progressive neurological disease. The most consistent clinical signs are acute behavioural change and unexplained, progressive paralysis (Rupprecht 2005). The disease is rapidly progressive, with death within eight to ten days of clinical signs appearing (Niezgoda et al. 2002; Tepsumethanon et al. 2004).

Details of clinical signs are available in the AUSVETPLAN for rabies (Animal Health Australia 2011b).

### **Pathology**

Severe clinical signs occur with little gross pathology in the brain other than congestion and less frequently, mild cerebral oedema. Microscopic changes include perivascular mononuclear inflammatory cell infiltration and vascular congestion in

the central nervous system (Perl 1975). Intracytoplasmic inclusion (Negri) bodies in neurons are diagnostic, but may only be present in about 75% of cases, and vary in size and morphology with the species affected. When present, they may occur throughout the central nervous system or be concentrated in particular regions of the brain, but usually in inflamed regions (Perl 1975).

### **Diagnosis**

A presumptive diagnosis of rabies can be made on clinical signs in live animals, but detection of virus is required to confirm infection. Histological examination may reveal nonsuppurative encephalitis and Negri bodies, but immunochemical identification or, less commonly, animal or tissue culture inoculation to detect replication of rabies virus is required for definitive diagnosis (OIE 2011a; Rupprecht et al. 2001).

The OIE recommends use of the indirect fluorescent antibody test (IFAT), an immunochemical test and an *in vitro* cell culture test for rabies diagnosis. The mouse inoculation test (MIT) is no longer recommended (OIE 2011a). IFAT on slide impressions or smears of brainstem, hippocampus and cerebellum is rapid, sensitive and specific, delivering results in two to four hours. The IFAT has a specificity and sensitivity close to 100% when testing tissues that are not decomposed and have not been fixed with formalin, and agreement of more than 99% with the MIT (Bourhy et al. 1989). The IFAT is the most widely used diagnostic test to confirm rabies virus infection and is recommended by both the World Health Organization (WHO) and OIE (OIE 2011a; Rupprecht et al. 2001).

Measurement of rabies virus antibody titre has limited diagnostic value because, in both human and nonhuman hosts, seroconversion may not occur before death (OIE 2011a; Trimarchi et al. 1986).

Polymerase chain reaction (PCR) methods are highly sensitive and specific, and can detect low titres of rabies virus and other lyssaviruses (Wakeley et al. 2005). However, using PCR-based techniques for routine postmortem diagnosis of rabies is not recommended by WHO or the OIE (OIE 2011a). Despite this, some laboratories such as the CSIRO–Australian Animal Health Laboratory (Animal Health Australia 2011b) may use PCR techniques to provide epidemiological information on the virus, if required.

### **Immunology**

#### *Immune response to infection*

Rabies virus is well adapted to avoiding and suppressing the host immune system. How it does this is not fully understood. Mechanisms include intracellular sequestration of virus within nerve tissue, which is an immunoprivileged site; causing minimal neuronal cell damage, which reduces release of antigens; inducing peripheral immunosuppression; and attracting nonrabies virus specific lymphocytes into the central nervous system, which reduces lymphocytes in the periphery (Lafon 2002; Wiktor et al. 1985).

Fatal infections produce little or no virus neutralising antibody titre in serum (Gerber et al. 1985; Manickam et al. 2008; Swanepoel 1994; WHO 2006b).

Reports of dogs surviving experimental infection are rare. In addition to their rarity, reports of survival due to natural immunity are questionable (Fekadu and Baer 1980; Manickam et al. 2008).

Variable fatality rates ranging from 30% to 85% were reported in experimental infections in cats (Soulebot et al. 1981; Vaughn et al. 1963).

#### *Measurement of antibody titres*

Virus neutralising antibody levels can be measured after vaccination for rabies virus to evaluate the animal's antibody response to the vaccine (Bahloul et al. 2005). There is close correlation between the fluorescent antibody virus neutralisation (FAVN) and the rapid fluorescent focus inhibition tests (RFFIT) in the measurement of post-vaccinal antibody titres (Shimazaki et al. 2003). Results are expressed in international units (IUs) or equivalent units relative to an international standard antiserum. Both tests are prescribed by the OIE for international movement of dogs and cats (OIE 2011a).

The indirect enzyme-linked immunosorbent assay (ELISA) is also prescribed to assess post-vaccinal immunity—provided the kit used was validated and adopted on the 'Register of diagnostic tests certified by the OIE as validated as fit for purpose'. Only one kit, Platelia Rabies II, is listed (OIE 2010). An evaluation trial of this ELISA in 2010 indicated it is best suited as a screening test, such as might be used in an eradication campaign to assess vaccine delivery to target populations (e.g. dogs or foxes) (Wasniewski and Cliquet 2010). Until further information becomes available THE DEPARTMENT OF AGRICULTURE considers that an ELISA technique should not replace the FAVN and RFFIT for assessing neutralising antibody in individual companion animals vaccinated against rabies.

### **Vaccination**

#### *Types of vaccines*

**Attenuated live virus vaccines.** These vaccines use viruses that were processed to reduce their pathogenicity in target and nontarget host species. In general, due to safety reasons live attenuated rabies virus vaccines were replaced by inactivated cell culture vaccines (Esh et al. 1982). At the present time one attenuated live vaccine is recommended by WHO and it is formulated as an oral bait for use in wild and feral animal populations (Cliquet et al. 2007).

**Inactivated cell culture vaccines.** Virtually all commercially available rabies virus vaccines contain inactivated viruses. Commercial inactivated rabies virus vaccines for use in animals are produced from a number of rabies virus biotypes (Bowen-Davies and Lowings 2000). The virus is inactivated by chemical or physical means so it cannot cause infection, but can still induce immunity (OIE 2011a).

**Recombinant vaccines.** Recombinant vaccines do not contain live rabies virus. They are manufactured by inserting rabies virus nucleic acid into a benign virus vector

such as vaccinia or canary pox virus. The recombinant canary pox vaccine is registered for use in cats in the United States; however, its present formulation does not stimulate immunity in dogs (Day et al. 2010).

Plasmid DNA vectors encoding the rabies virus glycoprotein G were shown to elicit strong, antigen-specific immune responses in dogs and cats (Lodmell et al. 2003; Osorio et al. 1999; Tesoro Cruz et al. 2006). Antibodies raised to plasmid DNA vaccines have been shown to be protective in dogs under experimental conditions (Bahloul et al. 2006).

Currently, only one live recombinant oral vaccine, the vaccinia recombinant glycoprotein vaccine, is recommended by WHO. It is safe and efficacious by the oral route in target (e.g. red fox, raccoon dog, skunk, dog) and non-target species (e.g. other wild and domestic species in contact with baits); therefore, it can be distributed in baits to immunise wild (or domestic) animals (OIE 2011a).

#### *Genotypes covered*

Current rabies virus vaccines protect against all known rabies virus biotypes as well as the Australian bat lyssavirus. They provide variable protection against other known Lyssavirus genotypes (Horton et al. 2010) but not against genotypes 2 and 3 (Lagos bat virus and Mokola viruses) or the West Caucasian bat virus (Hanlon et al. 2005; OIE 2011a).

#### *Manufacturing controls*

WHO and the OIE provide recommendations for licensing newly developed vaccines. Vaccines should confer protective immunity for at least one year in target species. National or regional requirements may apply in addition to WHO and the OIE recommendations (OIE 2011a).

To provide protection, vaccines must be efficacious and stored and administered according to manufacturer's instructions. The efficacy of commercial vaccines varies. Exporting countries are responsible for registration of vaccines in their jurisdiction.

#### *Vaccination guidelines*

A summary of vaccination guidelines is provided in Table 8.

Table 8 Guidelines for the vaccination of dogs and cats

Vaccine	Initial young vaccination ( $\leq 16$ weeks)	Initial adult vaccination ( $> 16$ weeks)	Revaccination recommendation
<b>Dogs</b>			
Rabies (killed parenteral)	Administer one dose as early as 3 months of age. In high-risk areas, and if permitted by law, give a second dose 2–4 weeks after the first dose	Administer a single dose	Canine rabies vaccines with either a 1- or 3-year DOI are available. Timing of boosters is determined by this licensed DOI, but in some areas may be dictated by statute
<b>Cats</b>			
Rabies (canary pox virus-vectored recombinant, non-adjuvanted, parenteral)	Administer a single dose as early as 8 weeks of age, with revaccination 1 year later	Administer 2 doses, 12 months apart	Annual booster is required
Rabies (1, 3 and 4-year killed, adjuvanted products are available, parenteral)	Administer a single dose as early as 12 weeks of age, with revaccination 1 year later	Administer 2 doses, 12 months apart	Booster as for licensed DOI or as required by local regulations

DOI = duration of immunity  
Source: Day et al. (2010)

#### *Protection after vaccination*

Vaccination has been shown to induce an effective and relatively long-lasting humoral immune response in vaccinated dogs and cats (Bahloul et al. 2006; Coyne et al. 2001; Fooks 2001; Lakshmanan et al. 2006). Rabies-specific virus-neutralising antibodies produced as a result of vaccination provide a reliable indicator that an animal can withstand challenge with virulent rabies virus (Moore and Hanlon 2010; Wilsmore et al. 2006).

The minimum duration of immunity induced by vaccination of dogs and cats with some commercially available inactivated canine rabies virus is at least three years measured by challenge and seven years measured by serology (Day et al. 2010; Schultz 2006; Sharpee et al. 1985; Soulebot et al. 1981; Tizard and Ni 1998).

The occurrence of rabies in vaccinated dogs and cats has been documented in the United States, but investigations indicate this is rare (Clark et al. 1981; Clark and Wilson 1996; De Benedictis et al. 2009; Murray et al. 2009).

The presence of maternal antibodies in young animals can interfere with the development of active immunity following vaccination. Rabies vaccines should therefore only be administered to animals at three months of age or older (Lakshmanan et al. 2006).

#### *Correlation between protection after vaccination and virus neutralising titre*

The immunological basis of protection against rabies infection following vaccination is not fully understood. Both humoral and cellular immune responses are induced by

rabies virus vaccines and are important in providing protective immunity (Lafon 2002; Schultz 2006).

Neutralising antibodies are useful for evaluating vaccine efficacy, but absence of these antibodies does not preclude protective immunity (Wilsmore et al. 2006). The virus neutralising titre following rabies virus vaccination generally peaks at about four weeks following vaccination and then declines (Mansfield et al. 2004).

WHO and the OIE recommend two types of virus neutralisation tests—the FAVN test or the RFFIT. WHO concluded that a virus neutralising antibody titre of at least 0.5 IU/mL is a reliable indicator of protective immunity in animals and humans (WHO 2006b).

This minimum antibody titre (0.5 IU/mL) was accepted by the OIE as the international standard for safe movement of dogs and cats (Mansfield et al. 2004; OIE 2010b). The adopted international standard represents a conservative value, as significantly lower titres (0.2 IU/mL for dogs; 0.1 IU/mL for cats) were shown to be protective (Aubert 1993).

Most animals receiving two or more doses of rabies vaccine had titres of at least 0.5 IU/mL for at least one year postvaccination (Briggs et al. 1998).

Data analysis from dogs and cats vaccinated for the first time against rabies has shown that aged animals are more likely to not achieve the minimum antibody titre recommended by the OIE as being protective (Kennedy et al. 2007).

Animals infected before or at the time of vaccination can continue to incubate the disease despite developing an antibody titre (Blancou et al. 1989). For dogs and cats from rabies-affected countries a waiting period of six months (i.e. the incubation period of rabies virus), following the development of postvaccinal immunity is standard practice before importation, to allow expression of clinical signs if rabies virus infection was acquired before vaccination (Fooks et al. 2000).

#### **4.16.3 Current biosecurity measures**

Five biosecurity categories apply to the rabies classification of an exporting country, as determined by the Australian Government. For each category, prescribed pre-export and post-arrival biosecurity measures apply. The criteria for categorisation and approved countries listed in each category under the previous policy are shown in Appendix 1.

#### **4.16.4 Risk review**

This section documents the review of the risk of rabies introduction associated with importing domestic dogs and cats under Australia's current requirements, and considers whether those risks have changed significantly since the introduction of the existing requirements in 1995. Under Australia's biosecurity policy, many thousands of dogs and cats have been imported without the introduction of rabies virus.

## Release

Rabies is widely distributed in the world; few countries are rabies-free.

In countries where rabies is endemic in the domestic and wild dog populations, prevalence varies and depends largely on the efficiency, effectiveness and management of the rabies control programs.

In countries and regions where rabies is endemic only in wildlife maintenance hosts (i.e. Canada, United States, Western Europe), effective rabies management and control programs including vaccination of domestic dogs and cats, minimise the occurrence of urban rabies. However, cases of urban rabies in unvaccinated dogs and cats occur sporadically due to spill over infections from wildlife (Krebs et al. 2004; Rupprecht et al. 2006).

Cats are less susceptible to rabies than dogs, but all unvaccinated dogs and cats are susceptible to rabies virus. Dogs are recognised as the principal vector for human rabies virus infection in many parts of the world (Cliquet and Picard-Meyer 2004).

Data on the international movement of dogs is limited, but an estimated 25% of dogs imported into the United States in 2006 were either too young to be vaccinated or were not current for rabies vaccination (McQuiston et al. 2008).

The incubation period for rabies generally ranges between three to twelve weeks and in rare cases has exceeded six months. Six months is the internationally recognised incubation period (OIE 2011b).

Infected dogs and cats can excrete rabies virus from up to 15 days before clinical signs start to appear, and can continue to excrete virus until they die (OIE 2011b).

Increased availability of effective vaccines against rabies has facilitated an increase in the international movement of companion animals from rabies-affected countries (Goddard et al. 2010).

Reports of vaccine failure are rare, but instances of vaccine failure should be anticipated for a variety of reasons (e.g. inappropriate vaccine storage, such as 'cold chain failure', incorrect administration of the vaccine and/or immunocompromised vaccine recipients).

Rabies virus neutralising antibody titres are a reliable indicator of postvaccinal immunity; postvaccinal neutralising antibody titres of > 0.5 IU/mL are accepted internationally as an indicator of effective vaccination (OIE 2011a; OIE 2011b).

Taking the preceding factors into consideration, the review concluded that:

- rabies virus control and management has improved in many developed countries, especially in Europe and North America
- canine rabies continues to be a serious problem in many countries in Asia, Africa, Central and South America and the Middle East

- increased availability of effective vaccines against rabies has facilitated an increase in the international movement of domestic dogs and cats from rabies-affected countries
- the likelihood of rabies virus being present in a dog or cat imported from an approved country has not changed significantly since the introduction of existing quarantine requirements in 1995.

### **Exposure**

Rabies virus is generally transmitted in saliva via the bite of an infected animal or by licking abrasions (Bingham 2005; Rupprecht et al. 2002).

The incubation period for rabies virus is defined as six months (OIE 2011b), but generally ranges from a few days to several months.

Dogs and cats can excrete rabies virus for up to 15 days before they start to show clinical signs; therefore, infected animals may appear healthy and transmit rabies virus to other animals (OIE 2011b).

All mammals, including humans, are susceptible to infection. Australian exposure groups considered susceptible to rabies virus are mammals including:

- humans
- domestic dogs and cats
- feral canids—foxes, feral dogs
- feral felids—feral cats
- native canids—dingoes
- dasyurids—Tasmanian devils, antechinus, dunnarts and quolls.

The majority of dogs and cats imported into Australia are companion animals kept in relatively secure premises with limited but controlled access to other pets and minimal exposure to feral animals and wildlife. Humans and other companion animals living in the same household as a rabies virus-infected dog or cat are most at risk of exposure.

Inquisitive species, such as cattle or humans, are more likely to be bitten by a rabid animal than timid species (Bowen-Davies and Lowings 2000).

Local governments (councils) in Australia generally have effective animal control programs to minimise the stray dog population. This reduces uncontrolled contacts that a rabies virus-infected dog or cat may have with stray dogs.

Taking the preceding factors into consideration, the review concluded that:

- the likelihood of an Australian exposure group being exposed to an imported dog or cat infected with rabies virus has not changed significantly since the introduction of existing quarantine requirements in 1995.

### **Consequences—outbreak scenario of establishment and/or spread**

In the event that an infected dog or cat incubating rabies virus is imported into Australia, is released from post-arrival quarantine and bites other dogs—especially local stray dogs, wild dogs or dingoes—rabies virus infection may establish and be maintained within the exposure group. Spill over into non-native canid and felid species (foxes, wild dogs and feral cats) may lead to the establishment and spread of rabies virus infection in feral and wild animal populations.

Dogs are the main vector for interspecies rabies virus transmission (Bourhy et al. 2008). With 3.41 million pet dogs in Australia (Australian Companion Animal Council Inc. 2010), the majority of which are not vaccinated against rabies virus, it is plausible that if exposed to a rabies virus-infected dog or cat, rabies virus would establish and spread within Australia's dog population.

Local governments (councils) in Australia generally have animal control programs to control and minimise stray dogs, thus reducing but not eliminating the likelihood of establishment and/or spread via stray dogs.

Dingoes and dasyurids (Tasmanian devil, quoll, antechinus, dunnart) are less abundant than feral canids and felids, but are considered susceptible to spill over infection and may contribute to disease agent establishment and spread. Native wildlife on which canid species occasionally prey (possums, wombats, wallabies, kangaroos) may also be infected but are unlikely to maintain a wildlife rabies virus cycle.

Australia has effective veterinary and human health services capable of detecting a rabies outbreak quickly. Pending confirmation of a diagnosis of rabies virus infection, the emergency management strategy outlined in the AUSVETPLAN rabies disease strategy manual would be implemented to reduce the risk of establishment or spread (Animal Health Australia 2011b).

Taking the preceding factors into consideration, the review concluded that:

- the likelihood of establishment and/or spread of rabies virus in Australia via imported dogs and cats has not changed significantly since the introduction of current biosecurity requirements in 1995.

### **Consequences—effects of establishment and/or spread**

Under Australia's Emergency Animal Disease Response Agreement, variation no. 10/02—26.10.10, rabies virus is listed as a Category 1 disease (Phillips Fox Lawyers 2010). Category 1 diseases are emergency animal diseases that seriously affect human health and/or the environment (such as depletion of native fauna), but may only have minimal direct consequences to livestock industries.

If rabies virus infection was to establish in free-ranging feral and wild animal populations, the disease may be difficult to eradicate and may become endemic. Vaccination via oral baits has proven to be an effective method of rabies virus control in reservoir populations of some wildlife and feral animals. For example, ongoing implementation of oral-bait vaccination programs has enabled the eradication of

rabies virus from the red fox population throughout most of Europe (Cliquet and Aubert 2004; Wilsmore et al. 2006).

Resource intensive, ongoing management and control programs are necessary for managing the threats to public health, animal health (including native fauna) and social amenity associated with endemic rabies virus infection in feral and wild animals.

Taking the preceding information into consideration, the review concluded that:

- the consequences of establishment and/or spread of rabies virus in Australia have not changed significantly since the introduction of biosecurity requirements in 1995.

#### **4.16.5 Conclusion**

Based on the preceding information, it was concluded that the overall risk of rabies virus infection associated with the importation of dogs and cats has not changed significantly since the introduction of biosecurity requirements in 1995.

Due to the significant animal health and public health consequences associated with the introduction and establishment of rabies virus, it was concluded that risk management measures for rabies continue to be warranted for both dogs and cats. It was also concluded that risk management measures for rabies was not warranted for dog or cat semen.

Risk management options for rabies virus in dogs and cats are outlined in Appendix 2.

#### **References**

Animal Health Australia (2011a). *Animal health in Australia 2010*. Animal Health Australia, Canberra.

Animal Health Australia (2011b). *Disease strategy: rabies (version 3.0)*. Primary Industries Ministerial Council, Canberra.

Arai YT, Kuzmin IV, Karneoka Y, Botvinkin AD (2003). New lyssavirus genotype from the lesser mouse-eared bat (*Myotis blythi*), Kyrgyzstan. *Emerging Infectious Diseases* 9:333–337.

Aubert MFA (1993). Can vaccination validated by the titration of rabies antibodies in serum of dogs and cats be an alternative to quarantine measures? *Abstracts on Hygiene and Communicable Diseases* 68:R1–R22.

Australian Companion Animal Council Inc. (2010). *Contribution of the pet care industry to the Australian economy*, 7th edn. Australian Companion Animal Council Inc., New South Wales.

- Baer GM (1975). Pathogenesis to the central nervous system. In *The natural history of rabies*, 1st edn, Baer GM (ed.), Academic Press, New York, pp. 181–198.
- Baer GM, Cleary WF (1972). A model in mice for the pathogenesis and treatment of rabies. *The Journal of Infectious Diseases* 125:520–527.
- Bahloul C, Taieb D, Diouani MF, Ahmed SBH, Chtourou Y, B'Chir BI, Kharmachi H, Dellagi K (2006). Field trials of a very potent rabies DNA vaccine which induced long lasting virus neutralizing antibodies and protection in dogs in experimental conditions. *Vaccine* 24:1063–1072.
- Bahloul C, Taieb D, Kaabi B, Diouani MF, Hadjahmed SB, Chtourou Y, B'Chir BI, Dellagi K (2005). Comparative evaluation of specific ELISA and RFFIT antibody assays in the assessment of dog immunity against rabies. *Epidemiology and Infection* 133:749–757.
- Bingham J (2005). Canine rabies ecology in southern Africa. *Emerging Infectious Diseases* 11:1337–1342.
- Blancou J, Soria Baltazar R, Artois M, Toma B, Rollin P (1989). Rabies despite pre- or post-exposure vaccination. In *Progress in rabies control: proceedings of the second international IMVI ESSEN/WHO symposium on 'New developments in rabies control'*, Essen, 5–7 July 1988 and report of the WHO Consultation on Rabies, Essen, 8 July 1988, Thraenhart O, Koprowski H, Bögel K, Sureau P (eds), Wells Medical, Kent, pp. 441–447.
- Blanton JD, Dyer J, McBrayer J, Rupprecht CE (2012) Rabies surveillance in the United States during 2011. *Journal of American Veterinary Medical Association* 241: 712-722.
- Bourhy H, Reynes J-M, Dunham EJ, Dacheux L, Larrous F, Huong VTQ, Xu G, Yan J, Miranda ME, Holmes EC (2008). The origin and phylogeography of dog rabies virus. *Journal of General Virology* 89:2673–2681.
- Bourhy H, Rollin PE, Vincent J, Sureau P (1989). Comparative field evaluation of the fluorescent-antibody test, virus isolation from tissue culture, and enzyme immunodiagnosis for rapid laboratory diagnosis of rabies. *Journal of Clinical Microbiology* 27:519–523.
- Bowen-Davies J, Lowings P (2000). Current perspectives on rabies 1: the biology of rabies and rabies-related viruses. *In Practice* 22:118–124.
- Briggs DJ, Smith JS, Mueller FL, Schwenke J, Davis RD, Gordon CR, Schweitzer K, Orciari LA, Yager PA, Rupprecht CE (1998). A comparison of two serological methods for detecting the immune response after rabies vaccination in dogs and cats being exported to rabies-free areas. *Biologicals* 26:347–355.
- BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper, BRS, Canberra.

- Clark KA, Kelly VP, Newman EC, Bilderback WR, Nettles WD, Rhodes TS (1981). Rabies vaccination: field observations during epizootics in dogs. *Modern Veterinary Practice* 62:907–911.
- Clark KA, Wilson PJ (1996). Postexposure rabies prophylaxis and preexposure rabies vaccination failure in domestic animals. *Journal of the American Veterinary Medical Association* 208:1827–1830.
- Cliquet F, Aubert M (2004). Elimination of terrestrial rabies in western European countries. *Developments in Biologicals* 119:185–204.
- Cliquet F, Gurbuxani JP, Pradhan HK, Pattnaik B, Patil SS, Regnault A, Begouen H, Guiot AL, Sood R, Mahl P, Singh R, Meslin FX, Picard E, Aubert MFA, Barrat J (2007). The safety and efficacy of the oral rabies vaccine SAG2 in Indian stray dogs. *Vaccine* 25:3409–3418.
- Cliquet F, Picard-Meyer E (2004). Rabies and rabies-related viruses: a modern perspective on an ancient disease. *Revue Scientifique et Technique de l'Office International des Epizooties* 23:625–642.
- Coyne MJ, Burr JH, Yule TD, Harding MJ, Tresnan DB, McGavin D (2001). Duration of immunity in dogs after vaccination or naturally acquired infection. *The Veterinary Record* 149:509–515.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Day MJ, Horzinek MC, Schultz RD (2010). WSAVA guidelines for the vaccination of dogs and cats. *Journal of Small Animal Practice* 51:e1–e32.
- De Benedictis P, Mutinelli F, Veggiato C, Capua I, Squecco G, Coassin R, Ferri G (2009). Rabies in a vaccinated dog in Italy. *The Veterinary Record* 165:216.
- Esh JB, Cunningham JG, Wiktor TJ (1982). Vaccine-induced rabies in four cats. *Journal of the American Veterinary Medical Association* 180:1336–1339.
- Fekadu M (1988). Pathogenesis of rabies virus infection in dogs. *Reviews of Infectious Diseases* 10:678–683.
- Fekadu M, Baer GM (1980). Recovery from clinical rabies of 2 dogs inoculated with a rabies virus strain from Ethiopia. *American Journal of Veterinary Research* 41:1632–1634.
- Fekadu M, Shaddock JH, Baer GM (1981). Intermittent excretion of rabies virus in the saliva of a dog two and six months after it had recovered from experimental rabies. *The American Journal of Tropical Medicine and Hygiene* 30:1113–1115.

- Fekadu M, Shaddock JH, Baer GM (1982). Excretion of rabies virus in the saliva of dogs. *The Journal of Infectious Diseases* 145:715–719.
- Fekadu M, Shaddock JH, Chandler FW, Baer GM (1983). Rabies virus in the tonsils of a carrier dog. *Archives of Virology* 78:37–47.
- Fogelman V, Fischman HR, Horman JT, Grigor JK (1993). Epidemiologic and clinical characteristics of rabies in cats. *Journal of the American Veterinary Medical Association* 202:1829–1833.
- Fooks AR (2001). Keeping rabies out by surveillance strategies, vaccination and serology. In *Proceedings of the Southern and Eastern African rabies group / World Health Organization Meeting, 18–22 June 2001, Lilongwe, Malawi*, World Health Organization, Geneva, pp. 131–135.
- Fooks AR, McElhinney L, Pollitt WJ (2000). Rabies antibody and the pet travel scheme. *The Veterinary Record* 147:427.
- Gerber JD, Sharpee RL, Swieczkowski TC, Beckenhauer WH (1985). Cell-mediated immune response to rabies virus in dogs following vaccination and challenge. *Veterinary Immunology and Immunopathology* 9:13–22.
- Goddard A, Donaldson N, Kosmider R, Kelly L, Adkin A, Horton D, Fooks T, Breed A, Freuling C, Muller T, Shaw S, Hallgren G, Snary E (2010). A quantitative risk assessment on the change in likelihood of rabies introduction into the United Kingdom as a consequence of adopting the existing harmonised community rules for the non-commercial movement of pet animals. <http://archive.defra.gov.uk> (accessed 2 August 2011).
- Gould AR, Hyatt AD, Lunt R, Kattenbelt JA, Hengstberger S, Blacksell SD (1998). Characterisation of a novel lyssavirus isolated from Pteropid bats in Australia. *Virus Research* 54: 165-187.
- Greene CE, Rupprecht CE (2006). Rabies and other lyssavirus infections. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 167–183.
- Hanlon CA, Kuzmin IV, Blanton JD, Weldon WC, Manangan JS, Rupprecht CE (2005). Efficacy of rabies biologics against new lyssaviruses from Eurasia. *Virus Research* 111:44–54.
- Horton DL, McElhinney LM, Marston DA, Wood JLN, Russell CA, Lewis N, Kuzmin IV, Fouchier RAM, Osterhaus ADME, Fooks AR, Smith DJ (2010). Quantifying antigenic relationships among the lyssaviruses. *The Journal of Virology* 84:11841–11848.
- ICTV (2009). Genus: Lyssavirus. ICTV Virus Taxonomy: 2009 Release. <http://ictvonline.org/virusTaxonomy.asp?version=2009> (accessed 24 May 2010)

- Jackson AC (1994). Animal models of rabies virus neurovirulence. *Current Topics in Microbiology and Immunology* 187:85–93.
- Jones RD, Kelly L, Fooks AR, Wooldridge M (2005). Quantitative risk assessment of rabies entering Great Britain from North America via cats and dogs. *Risk Analysis* 25:533–542.
- Kaplan MM (1969). Epidemiology of rabies. *Nature* 221:421–425.
- Kayali U, Mindekem R, Yemadji N, Oussieguere A, Naissengar S, Ndoutamia AG, Zinsstag J (2003). Incidence of canine rabies in N'Djamena, Chad. *Preventive Veterinary Medicine* 61:227–233.
- Kennedy LJ, Lunt M, Barnes A, McElhinney L, Fooks AR, Baxter DN, Ollier WER (2007). Factors influencing the antibody response of dogs vaccinated against rabies. *Vaccine* 25:8500–8507.
- Kihm U, Lazarowicz M, Bommeli W, Zutter R (1982). Potency of two rabies vaccines in cats as determined by antibody assay and virulent virus challenge. *Comparative Immunology, Microbiology and Infectious Diseases* 5:227–232.
- Kitala PM, McDermott JJ, Kyule MN, Gathuma JM (2000). Community-based active surveillance for rabies in Machakos District, Kenya. *Preventive Veterinary Medicine* 44:73–85.
- Knobel DL, Cleaveland S, Coleman PG, Fevre EM, Meltzer MI, Miranda MEG, Shaw A, Zinsstag J, Meslin F-X (2005). Re-evaluating the burden of rabies in Africa and Asia. *Bulletin of the World Health Organization* 83:360–368.
- Krebs JW, Mandel EJ, Swerdlow DL, Rupprecht CE (2004). Rabies surveillance in the United States during 2003. *Journal of the American Veterinary Medical Association* 225:1837–1849.
- Krebs JW, Wheeling JT, Childs JE (2003). Rabies surveillance in the United States during 2002. *Journal of the American Veterinary Medical Association* 223:1736–1748.
- Kuzmin IV, Bozick B, Guagliardo SA, Kunkel R, Shak JR, Tong S, Rupprecht CE (2011). Bats, emerging infectious diseases, and the rabies paradigm revisited. *Emergency Health Threats Journal* 4:7159.
- Kuzmin IV, Mayer AE, Niezgodna M, Markotter W, Agwanda B, Breiman RF, Rupprecht CE (2010). Shimoni bat virus, a new representative of the Lyssavirus genus. *Virus Research* 149:197–210.
- Kuzmin IV, Wu X, Tordo N, Rupprecht CE (2008). Complete genomes of Aravan, Khujand, Irkut and West Caucasian bat viruses, with special attention to the polymerase gene and non-coding regions. *Virus Research* 136:81–90.

- Lafon M (2002). Immunology. In *Rabies*, Jackson AC, Wunner WH (eds), Academic Press, Amsterdam, pp. 351–369.
- Lakshmanan N, Gore TC, Duncan KL, Coyne MJ, Lum MA, Sterner FJ (2006). Three-year rabies duration of immunity in dogs following vaccination with a core combination vaccine against canine distemper virus, canine adenovirus type-1, canine parvovirus, and rabies virus. *Veterinary Therapeutics* 7:223–231.
- Lodmell DL, Parnell MJ, Weyhrich JT, Ewalt LC (2003). Canine rabies DNA vaccination: a single-dose intradermal injection into ear pinnae elicits elevated and persistent levels of neutralizing antibody. *Vaccine* 21:3998–4002.
- Manickam R, Basheer MD, Jayakumar R (2008). Post-exposure prophylaxis (PEP) of rabies-infected Indian street dogs. *Vaccine* 26:6564–6568.
- Mansfield KL, Burr PD, Snodgrass DR, Sayers R, Fooks AR (2004). Factors affecting the serological response of dogs and cats to rabies vaccination. *The Veterinary Record* 154:423–426.
- Matouch O (2008). The rabies situation in Eastern Europe. *Developments in Biologicals* 131:27–35.
- McCull KA, Tordo N, Setien AA (2000). Bat lyssavirus infections. *Revue Scientifique et Technique de l'Office International des Epizooties* 19:177–196.
- McQuiston JH, Wilson T, Harris S, Bacon RM, Shapiro S, Trevino I, Sinclair J, Galland G, Marano N (2008). Importation of dogs into the United States: risks from rabies and other zoonotic diseases. *Zoonoses and Public Health* 55:421–426.
- McQuiston JH, Yager PA, Smith JS, Rupprecht CE (2001). Epidemiologic characteristics of rabies virus variants in dogs and cats in the United States, 1999. *Journal of the American Veterinary Medical Association* 218:1939–1942.
- Moore SM, Hanlon CA (2010). Rabies-specific antibodies: measuring surrogates of protection against a fatal disease. *PLoS Neglected Tropical Diseases* 4:e595.
- Murray KO, Holmes KC, Hanlon CA (2009). Rabies in vaccinated dogs and cats in the United States, 1997–2001. *Journal of the American Veterinary Medical Association* 235:691–695.
- Ngoepe CE, Sabeta C, Nel L (2009). The spread of canine rabies into Free State province of South Africa: a molecular epidemiological characterization. *Virus Research* 142:175–180.
- Niezgoda M, Hanlon CA, Rupprecht CE (2002). Animal rabies. In *Rabies*, Jackson AC, Wunner WH (eds), Academic Press, Amsterdam, pp. 163–218.
- OIE (World Organisation for Animal Health) (2010). Register of diagnostic tests certified by the OIE as validated as fit for purpose. OIE, Paris.

[http://web.oie.int/vcda/eng/en\\_VCDA\\_registre.htm?e1d9](http://web.oie.int/vcda/eng/en_VCDA_registre.htm?e1d9) (accessed 14 December 2010)

OIE (World Organisation for Animal Health) (2011a). *Manual of diagnostic tests and vaccines for terrestrial animals 2011*. OIE, Paris.

[http://www.oie.int/eng/normes/mmanual/A\\_summry.htm](http://www.oie.int/eng/normes/mmanual/A_summry.htm) (accessed 7 July 2011)

OIE (World Organisation for Animal Health) (2011b) Rabies. *Terrestrial animal health code 2011*, OIE, Paris.

[http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre\\_1.8.10.htm](http://www.oie.int/index.php?id=169&L=0&htmfile=chapitre_1.8.10.htm) (accessed 28 November 2011)

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris.

<http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Osorio JE, Tomlinson CC, Frank RS, Haanes EJ, Rushlow K, Haynes JR, Stinchcomb DT (1999). Immunization of dogs and cats with a DNA vaccine against rabies virus. *Vaccine* 17:1109–1116.

Perl DP (1975). The pathology of rabies in the central nervous system. In *The natural history of rabies*, 1st edn, Baer GM (ed.), Academic Press, New York, pp. 236–272.

Phillips Fox Lawyers (2010). *Government and livestock industry cost-sharing deed in respect of the Emergency Animal Disease Response Agreement*. Animal Health Australia, Canberra.

<http://www.animalhealthaustralia.com.au/wp-content/uploads/2011/04/EADRA.pdf> (accessed 28 June 2011)

Promed Mail (2013). Rabies—Indonesia (ML) RFI: ProMED Mail.

<http://www.promedmail.org>; published 28 February (accessed 5 May 2013)

Rupprecht CE (2005). Rabies. In *Merck veterinary manual*, 9th edn, Kahn CM, Line S (eds), Merck and Company, Inc., New Jersey, pp. 1067–1071.

Rupprecht CE, Hanlon CA, Hemachudha T (2002). Rabies re-examined. *Lancet* 2:327–343.

Rupprecht CE, Hanlon CA, Slate D (2006). Control and prevention of rabies in animals: paradigm shifts. *Developments in Biologicals* 125:103–111.

Rupprecht CE, Stohr K, Meredith C (2001). Part I: viral and prion diseases: rabies. In *Infectious diseases of wild mammals*, 3rd edn, Williams ES, Barker IK (eds), Iowa State University Press, Ames, pp. 3–36.

Schultz RD (2006). Duration of immunity for canine and feline vaccines: a review. *Veterinary Microbiology* 117:75–79.

- Sharpee RL, Nelson LD, Beckenhauer WH (1985). Inactivated tissue culture rabies vaccine with three years immunogenicity in dogs and cats. In *Rabies in the tropics: proceedings of an international conference on rabies control in the tropics*, Tunis, October 3–6, 1983, Kuwert E, Merieux C, Koprowski H, Bogel K (eds), Springer-Verlag, Heidelberg, pp. 262–269.
- Shimazaki Y, Inoue S, Takahashi C, Gamoh K, Etoh M, Kamiyama T, Makie H (2003). Immune response to Japanese rabies vaccine in domestic dogs. *Journal of Veterinary Medicine, Series B* 50:95–98.
- Soulebot JP, Brun A, Chappuis G, Guillemain F, Petermann HG, Precausta P, Terre J (1981). Experimental rabies in cats: immune response and persistence of immunity. *The Cornell Veterinarian* 71:311–325.
- Swanepoel R (1994). Rabies. In *Infectious diseases of livestock with special reference to southern Africa*, 1st edn, Coetzer JAW, Thomson GR, Tustin RC (eds), Oxford University Press, Cape Town, pp. 493–552.
- Swanepoel R (2004). Rabies. In *Infectious diseases of livestock*, 2nd edn, Coetzer JAW, Tustin RC (eds), Oxford University Press, Oxford, pp. 1123–1182.
- Tepsumethanon V, Lumlerdacha B, Mitmoonpitak C, Sitprija V, Meslin FX, Wilde H (2004). Survival of naturally infected rabid dogs and cats. *Clinical Infectious Diseases* 39:278–280.
- Tesoro Cruz E, Hernández González R, Alonso Morales R, Aguilar-Setién A (2006). Rabies DNA vaccination by the intranasal route in dogs. *Developments in Biologicals* 125:221–231.
- Tesoro-Cruz E, Calderón-Rodríguez R, Hernández-González R, Blanco-Favéla F, Aguilar-Setién A (2008). Intradermal DNA vaccination in ear pinnae is an efficient route to protect cats against rabies virus. *Veterinary Research* 39:1–11.
- Tizard I, Ni Y (1998). Use of serologic testing to assess immune status of companion animals. *Journal of the American Veterinary Medical Association* 213:54–60.
- Trimarchi CV, Rudd RJ, Abelseth MK (1986). Experimentally induced rabies in four cats inoculated with a rabies virus isolated from a bat. *American Journal of Veterinary Research* 47:777–780.
- Vaughn JB, Gerhardt P, Paterson JC (1963). Excretion of street rabies virus in saliva of cats. *Journal of the American Medical Association* 184:705–708.
- Velasco-Villa A, Reeder SA, Orciari LA, Yager PA, Franka R, Blanton JD, Zuckero L, Hunt P, Oertli EH, Robinson LE, Rupprecht CE (2008). Enzootic rabies elimination from dogs and reemergence in wild terrestrial carnivores, United States. *Emerging Infectious Diseases* 14:1849–1854.

- Wakeley PR, Johnson N, McElhinney LM, Marston D, Sawyer J, Fooks AR (2005). Development of a real-time, TaqMan reverse transcription-PCR assay for detection and differentiation of lyssavirus genotypes 1, 5, and 6. *Journal of Clinical Microbiology* 43:2786–2792.
- Wandeler AI (2008). The rabies situation in western Europe. *Developments in Biologicals* 131:19–25.
- Warner CK, Schurr TG, Fekadu M (1996). Molecular characterization of carrier rabies isolates. *Virus Research* 41:133–140.
- Wasniewski M, Cliquet F (2010). Collaborative study to assess the Platelia Rabies II Kit (Bio-Rad). Agence Française de Sécurité Sanitaire des Aliments, France.
- West P (2008). *Assessing invasive animals in Australia 2008*. PN 20628, National Land & Water Resources Audit, Canberra.
- WHO (2006a). About rabies: classification. Rabies Bulletin Europe (Online). [http://www.who-rabies-bulletin.org/About\\_Rabies/Classification.aspx](http://www.who-rabies-bulletin.org/About_Rabies/Classification.aspx) (accessed 15 January 2010)
- WHO (2006b). About rabies: diagnosis of rabies in animals. Rabies Bulletin Europe (Online). [http://www.who-rabies-bulletin.org/About\\_Rabies/Diagnosis.aspx](http://www.who-rabies-bulletin.org/About_Rabies/Diagnosis.aspx) (accessed 15 January 2010b)
- Wiktor TJ, MacFarlan RO, Koprowski H (1985). Rabies virus pathogenicity. In *Rabies in the tropics*, Kuwert E, Mérieux C, Koprowski H, Bögel K (eds), Springer-Verlag, Berlin, pp. 21–29.
- Wilsmore T, Hamblin C, Taylor N, Taylor W, Watson B (2006). *Qualitative veterinary risk assessment of the introduction of rabies into the United Kingdom: a report prepared for DEFRA*. Veterinary Epidemiology and Economics Research Unit, United Kingdom.
- Wu X, Hu R, Zhang Y, Dong G, Rupprecht CE (2009). Reemerging rabies and lack of systemic surveillance in People's Republic of China. *Emerging Infectious Diseases* 15:1159–1164.
- Zhang YZ, Fu ZF, Wang DM, Zhou JZ, Lv TF, Wang ZX, Zou Y, Yao WR, Li MH, Dong GM, Xu GL, Niezgodá M, Kuzmin IV, Rupprecht CE (2008). Investigations of the role of healthy dogs as potential carriers of rabies virus. *Vector-Borne and Zoonotic Diseases* 8:313–320.

## 4.17 Rift Valley fever

### 4.17.1 Background

Rift Valley fever (RVF) is a zoonotic viral disease characterised by mortality in young domestic ruminants and abortions in pregnant animals. RVF virus is an RNA virus in the genus *Phlebovirus* of the family *Bunyaviridae* (ARMCANZ 1996; Nichol et al. 2005)

RVF is endemic in sub-Saharan Africa, including Madagascar (Clements et al. 2007; Fontenille et al. 1985; Fontenille et al. 1988). The virus has also been detected in Egypt (Hoogstraal et al. 1979), Saudi Arabia and Yemen (Arishi et al. 2000; Gould and Higgs 2009).

RVF epidemics result from a combination of high mosquito numbers following heavy rain with large numbers of susceptible animals (Clements et al. 2006; Davies et al. 1985). Outbreaks in livestock are often accompanied by human disease (Swanepoel and Coetzer 2004).

Dogs and cats have not been demonstrably implicated in natural epidemics, although seroconversion was reported in dogs during the 1977 epidemic in Egypt (Hoogstraal et al. 1979). In a survey of domestic and wild dogs and cats in endemic regions, seroconversion was only detected in free-roaming lions (House et al. 1996).

RVF virus is not present in Australia; however, competent mosquito vectors for virus transmission are present in Australia (Turell and Kay 1998).

RVF is an OIE-listed disease of multiple species (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011).

### 4.17.2 Technical information

#### Epidemiology

RVF virus can infect many species of animals including cattle, goats, sheep, buffalo, camels, monkeys and humans, as well as grey squirrels and other rodents. The primary amplifying hosts are cattle and sheep. Viraemia without disease may occur in some adults of other species and severe disease can occur in newborn animals. Horses, donkeys, cats, dogs and rodents are low on the susceptibility scale and inapparent infections are the most likely outcome (ARMCANZ 1996). The mammalian reservoir for infection may exist in exotic carnivores such as the lion; however, dogs and cats can be experimentally infected and maintain a viraemia sufficient to infect mosquitoes (Walker et al. 1970a).

Humans do not seem to be infected by casual contact with live hosts but can be infected by aerosols or direct contact with tissues during parturition, slaughter,

postmortem examinations, laboratory procedures, meat preparation (i.e. for cooking), as well as via mosquitoes (CFSPH 2007).

RVF virus is transmitted by mosquitoes and amplified in ruminant hosts (Favier et al. 2008; Hoogstraal et al. 1979). Mosquitoes transmit RVF virus by transovarial means and the virus appears to survive in the dried eggs of *Aedes* mosquitoes (Swanepoel and Coetzer 2004). It takes 10–20 days for mosquitoes to become infective after feeding on an infected host, with this interval becoming shorter with increasing environmental temperature. There is concern that climate change could increase the frequency and distribution of this disease. Outbreaks typically occur in savannah grasslands every 5 to 15 years and in semi-arid regions every 25 to 35 years. After amplification in ruminants the RVF virus can be transmitted by other mosquito species and biting insects. RVF virus can be transmitted to the foetus during pregnancy and has been found in semen and milk.

### **Clinical signs**

In dogs inoculated with RVF virus, clinical signs were observed only in puppies seven days of age or less and infection was uniformly fatal (Walker et al. 1970a). Some pups became hyperthermic during infection, while all died in a terminal hypothermic state. Some of the animals showed signs of central nervous system dysfunction near death. In 84 day old pups, 50% developed viraemia and a corresponding increase in serum neutralisation titre. Clinical signs were inapparent; however, horizontal transmission of infection from pup to mother and from pup to pup was demonstrated (Walker et al. 1970a).

High mortality was observed when kittens less than three weeks of age were experimentally inoculated with virus (Walker et al. 1970b). A transient pyrexia was followed by terminal hypothermia. Kittens showed neurological signs of ataxia, followed by recumbency and paddling movements within the 24 hours before death. As with pups, evidence indicated the horizontal spread of RVF virus to kittens and adult cats.

### **Diagnosis**

Clinical diagnosis in adult dogs and cats is unlikely as RVF virus infection is usually inapparent. Several serological tests are available including virus and plaque reduction neutralisation, complement fixation, haemagglutination inhibition immunofluorescence test and enzyme-linked immunosorbent assay (ELISA).

Infected animals develop specific antibodies that may become demonstrable by ELISA as early as 6–7 days following infection.

#### **4.17.3 Current biosecurity measures**

There are no specific biosecurity measures for RVF.

#### 4.17.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by RVF:

- RVF is an OIE-listed disease and is a nationally notifiable disease in Australia
- infection in dogs and cats appears to be rare
- dogs and cats are not recognised as amplifying hosts of RVF virus
- dogs and cats have not been implicated in the spread of RVF virus.

#### 4.17.5 Conclusion

Based on the preceding key points, it was concluded risk management measures for RVF are not warranted for dogs, cats or their semen.

#### References

Arishi H, Ageel A, Rahman MA, Hazmi AA, Arishi AR, Ayoola B, Menon C, Ashraf J, Frogusin O, Sawwan F, Hazmi M, As-Sharif A, Al-Sayed M, Ageel AR, Alrajhi ARA, Al-Hedaithy MA, Fatani A, Sahaly A, Ghelani A, Al-Basam T, Turkistani A, Al-Hamadan N, Mishkas A, Al-Jeffri MH, Al-Mazroa YY, Alamri MMA (2000).

Outbreak of Rift Valley fever – Saudi Arabia, August–October, 2000. *Morbidity and Mortality Weekly Report* 49:905–908.

ARMCANZ (Agriculture and Resource Management Council of Australia and New Zealand) (1996). Disease strategy: Rift Valley fever. ARMCANZ, Canberra.

CFSPH (Center for Food Security and Public Health) (2007). Rift Valley fever. CFSPH, Iowa State University, Ames.

[http://www.cfsph.iastate.edu/Factsheets/pdfs/rift\\_valley\\_fever.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/rift_valley_fever.pdf) (accessed 13 October 2010)

Clements ACA, Pfeiffer DU, Martin V (2006). Application of knowledge-driven spatial modelling approaches and uncertainty management to a study of Rift Valley fever in Africa. *International Journal of Health Geographics* 5:57.

Clements ACA, Pfeiffer DU, Martin V, Otte MJ (2007). A Rift Valley fever atlas for Africa. *Preventive Veterinary Medicine* 82:72–82.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.

<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

Davies FG, Linthicum KJ, James AD (1985). Rainfall and epizootic Rift Valley fever. *Bulletin of the World Health Organization* 63:941–943.

- Favier C, Chalvet-Monfray K, Sabatier P, Lancelot R, Fontenille D (2008). Rift Valley fever in West Africa: the role of space in endemicity. *Tropical Medicine and International Health* 11:1878–1888.
- Fontenille D, Mathiot C, Coulanges P (1985). Les cycles arbovirus-vecteurs-vertèbres dans les forêts malgaches (The arbovirus-vectors-vertebrates cycles in the Malagasy forests). *Archives de l'Institut Pasteur de Madagascar* 52:171–180.
- Fontenille D, Mathiot C, Rodhain F, Coulanges P (1988). Les arboviroses dans la région de Nosy-Be, Madagascar: données sérologiques et entomologiques (Arboviral infections in the Nosy-Be area, Madagascar: serological and entomological data). *Bulletin de la Société de Pathologie Exotique et de ses Filiales* 81:57–70.
- Gould EA, Higgs S (2009). Impact of climate change and other factors on emerging arbovirus diseases. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 103:109–121.
- Hoogstraal H, Meegan JM, Khalil GM, Adham FK (1979). The Rift Valley fever epizootic in Egypt 1977–1978 2. Ecological and entomological studies. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 73:624–629.
- House C, Alexander KA, Kat PW, O'Brien SJ, Mangiafico J (1996). Serum antibody to Rift Valley fever virus in African carnivores. *Annals of the New York Academy of Sciences* 791:345–349.
- Nichol ST, Beaty BJ, Elliott RM, Goldbach R, Plyusnin A, Schmaljohn CS, Tesh RB (2005). Bunyaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses*, Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (eds), Elsevier, San Diego, pp. 695–716.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Swanepoel R, Coetzer JAW (2004). Rift Valley fever. In *Infectious diseases of livestock*, 2nd edn, Coetzer JAW, Tustin RC (eds), Oxford University Press, Oxford, pp. 1037–1070.
- Turell MJ, Kay BH (1998). Susceptibility of selected strains of Australian mosquitoes (Diptera: Culicidae) to Rift Valley fever virus. *Journal of Medical Entomology* 35:132–135.
- Walker JS, Remmele NS, Carter RC, Mitten JQ, Schuh LG, Stephen EL, Klein F (1970a). The clinical aspects of Rift Valley fever virus in household pets: I. Susceptibility of the dog. *Journal of Infectious Diseases* 121:9–18.
- Walker JS, Stephen EL, Remmele NS, Carter RC, Mitten JQ, Schuh LG, Klein F (1970b). The clinical aspects of Rift Valley fever virus in household pets: II. Susceptibility of the cat. *Journal of Infectious Diseases* 121:19–24.

## 4.18 Screw-worm fly myiasis

### 4.18.1 Background

The larval stages of the screw-worm fly (SWF) are obligate parasites of warm-blooded animals (including humans) and rarely birds. The fly larvae (maggots) feed on skin and underlying tissues of the live host producing a condition known as wound or traumatic myiasis which can be fatal. Three stages (instars) of larval development follow in the host tissue. Third-stage larvae have heavy bands of backwardly directed thorn-like spines resembling a wooden screw, hence the name 'screw-worm' (CFSPH 2007).

There are two species of SWF, *Chrysomya bezziana* (Old World screw-worm fly [OWSWF], Bezzi's blowfly) and *Cochliomyia hominivorax* (New World screw-worm fly [NWSWF]).<sup>13</sup> Both species are members of the family Calliphoridae, subfamily Chrysomyinae. The geographical ranges of these flies do not overlap; however, they have similar life cycles and biological characteristics.

SWF myiasis causes serious production losses to livestock industries. Due to its presence in Papua New Guinea, OWSWF constitutes the most direct exotic animal disease threat to Australia.

Both OWSWF and NWSWF are OIE-listed diseases (infestations) of multiple species (OIE 2012) and are nationally notifiable in Australia (DAFF 2011).

### 4.18.2 Technical information

#### Epidemiology

*Ch. bezziana* is a tropical to subtropical fly found mainly in sub-Saharan Africa, the Arabian peninsula, India, South-East Asia (including throughout much of Indonesia), Malaysia and the Philippines. High densities of OWSWF have been found in coastal swamps of Papua New Guinea adjacent to the Torres Strait (Animal Health Australia 2009). *Ch. bezziana* is also present in New Ireland and New Britain.

*Co. hominivorax* is confined to the Western Hemisphere and occurs in tropical and subtropical areas of Central and South America as far south as Argentina. Previously, *Co. hominivorax* occurred in the southern United States, but it has been eliminated from that country and most of Mexico by a large-scale sterile insect-release method (SIRM) campaign.

Both *Ch. bezziana* and *Co. hominivorax* are obligate parasites of warm-blooded animals. SWFs tend to be attracted to parts of the animal where the skin has been perforated and exudes blood. Adult flies may be attracted to lesions as small as tick bites. Adult females lay eggs in masses at wound margins or body orifices of living animals. The larvae emerge within 12–24 hours and immediately begin to feed, burrowing head-downwards into the wound. After developing through three larval stages, involving two moults in 5–7 days, the larvae leave the wound and drop to the

---

<sup>13</sup> Old World refers to Africa, Asia and Europe; New World refers to the Americas.

ground into which they burrow and pupate. The duration of the life cycle off the host is temperature-dependent, being shorter at higher temperatures and may be completed in less than three weeks in the tropics (OIE 2008).

In Hong Kong, *Ch. bezziana* myiasis has been reported in dogs, cattle and pigs (FEHD 2011). A retrospective study of OWSWF myiasis identified 59 affected dogs in Hong Kong during a one-year period (McNae and Lewis 2004).

No evidence for venereal transmission in dogs or cats was found in the scientific literature.

### **Clinical signs**

SWF myiasis produces a characteristic odour. Initially, a small wound may be difficult to see due to fur or hair covering its location (e.g. prepuce, vulva, ear canal). Later, wounds become larger and a secondary strike may result in hundreds of larvae at different stages of development in the wound. Secondary infection and tissue necrosis follow in untreated cases, resulting in weight loss, debility and death. In dogs, the larvae often tunnel under the skin (CFSPH 2007).

In Hong Kong, larvae have been recovered from dog-fight wounds (Chemonges-Nielsen 2003). Infestations in dogs commonly affect the face/head, neck/torso, legs/feet and tail/perineal areas (McNae and Lewis 2004).

### **Diagnosis**

Laboratory identification of the parasites is performed using a dissecting microscope.

SWF myiasis should be suspected in animals that have draining or enlarged wounds with signs of infestation.

Third instar larvae of both *Ch. bezziana* and *Co. hominivorax* have a robust, typical maggot shape, with a cylindrical body from 6–17 mm long and from 1.1–3.6 mm in diameter with one pointed and one blunt end. Younger larvae are creamy white and fully mature third-stage larvae may have a reddish-pink tinge. Both species have prominent rings of spines around the body and these appear conspicuous under a microscope when compared with most non-SWF species (Spradbery 2002).

#### **4.18.3 Current biosecurity measures**

There are no specific biosecurity measures for SWF myiasis. Current biosecurity measures for the management of external parasites are as follows:

- Within four days immediately before export, dogs and cats must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.

- Within four days immediately before export, dogs and cats must be subject to a thorough physical examination by a government-approved veterinarian and found to be visibly free from external parasites.

#### 4.18.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by SWF myiasis:

- Infestations with OWSWF and NWSWF are OIE-listed diseases of multiple-species and are nationally notifiable in Australia.
- SWF myiasis in dogs is not common, but has been reported in association with bite wounds and lacerations.

#### 4.18.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for SWF myiasis continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for SWF myiasis are not warranted for dog or cat semen.

The following biosecurity measures would provide appropriate risk management for dogs and cats.

##### Pre-export measures

- Within five days immediately before export, dogs and cats must be subject to a thorough physical examination by a government-approved veterinarian and found to be visibly free from external parasites.

##### Post-arrival measures

- As for post-arrival measures proposed for external parasite control.

#### References

AHA (Animal Health Australia) (2009). About screw worm fly. AHA, Canberra. <http://www.animalhealthaustralia.com.au/programs/biosecurity/screw-worm-fly-program/about-screw-worm-fly/> (accessed 7 June 2011)

CFSPH (Center for Food Security and Public Health) (2007). Screwworm myiasis. CFSPH, Iowa State University, Ames. [http://www.cfsph.iastate.edu/Factsheets/pdfs/screwworm\\_myiasis.pdf](http://www.cfsph.iastate.edu/Factsheets/pdfs/screwworm_myiasis.pdf) (accessed 10 February 2009)

Chemonges-Nielsen S (2003). *Chrysomya bezziana* in pet dogs in Hong Kong: a potential threat to Australia. *Australian Veterinary Journal* 81:202–205.

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra. <http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)

FEHD (Hong Kong Government Food and Environmental Hygiene Department) (2011). *Chrysomya bezziana*. FEHD, Hong Kong. <http://www.fehd.gov.hk/english/safefood/pest-post-chrysomya.html> (accessed 15 March 2012)

McNae JC, Lewis SJ (2004). Retrospective study of Old World screwworm fly (*Chrysomya bezziana*) myiasis in 59 dogs in Hong Kong over a one-year period. *Australian Veterinary Journal* 82:211–214.

OIE (World Organisation of Animal Health) (2008). New World screwworm (*Cochliomyia hominivorax*) and Old World screwworm (*Chrysomya bezziana*). *Manual of diagnostic tests and vaccines for terrestrial animals 2011*, OIE, Paris. [http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.01.10\\_SCREWW.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.10_SCREWW.pdf) (accessed 1 September 2009)

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Spradbery JP (2002). *A manual for the diagnosis of screw-worm fly*. Commonwealth Scientific and Industrial Research Organisation, Division of Entomology, Canberra.

## 4.19 Surra

### 4.19.1 Background

Surra is a disease caused by the flagellate protozoan *Trypanosoma evansi*, which can affect most domesticated mammals and some wild species. Infection may be subclinical or result in signs ranging from chronic weight loss to acute death. The disease is most severe in donkeys, horses, mules, deer, camels, llamas, dogs and cats (Geering et al. 1995). Wallabies are susceptible to experimental infection and develop acute fatal disease (Reid et al. 2001).

Surra is present in Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East (OIE 2010; Radostits et al. 2007).

Surra is an OIE-listed disease of multiple species (OIE 2012) and is a nationally notifiable disease in Australia (DAFF 2011). The *Manual of diagnostic tests and vaccines for terrestrial animals* (OIE 2010) includes a chapter on surra, but there are no OIE *Terrestrial animal health code* recommendations for surra relevant to the importation of live animals.

### 4.19.2 Technical information

#### Epidemiology

*T. evansi* is transmitted mechanically by biting flies, particularly of the genera *Tabanus*, *Stomoxys*, *Atylous* and *Lyperosia*. Vampire bats have been reported to transmit the parasite in South and Central America (Geering et al. 1995). Mechanical

vectors, including horseflies or march flies (family Tabanidae) and stable flies (family Muscidae), are widespread throughout Australia (Seddon and Albiston 1967a; Seddon and Albiston 1967b). Other modes of infection include feeding on infected tissues (Singh et al. 1993), transmission in milk and possibly via the venereal route (CFSPH 2009).

Surra occurs widely throughout Asia although companion animals are uncommonly affected (Irwin and Jefferies 2004). The reason for companion animals being less frequently affected is unclear. Dogs may be less susceptible to bites from tabanids due to their thick fur coat (Hoare 1972), although this is likely to be breed-dependent. Little information is available regarding the importance and epidemiology of many arthropod vectors of companion animals in Asia. Published surveys tend to record resident ectoparasites but tabanid flies are not described; their importance as disease vectors in dogs and cats is poorly understood (Irwin and Jefferies 2004). Non-vector routes of transmission (e.g. ingestion of infected tissues) may be a more important source of infection in dogs (Hoare 1972; Singh et al. 1993) and possibly cats.

Several studies have looked at the introduction of foreign disease agents to countries as a result of international travel of pets (Duscher et al. 2010; Hendrix et al. 1998b). Although the international movement of pets has resulted in the introduction of a number of exotic diseases to previously free countries, there is no evidence that surra has been introduced via companion animals (Hendrix et al. 1998a; Hendrix et al. 1998b). A single case of surra was diagnosed in the Netherlands from a young dog imported directly from Nepal. Despite successful treatment of the infection, the dog subsequently died of complications (Hellebrekers and Slappendel 1982). No additional cases have been reported since.

### **Clinical signs**

During the acute phase of the disease, there is intermittent pyrexia, subcutaneous oedema, progressive anaemia, blindness, lethargy and haemostatic abnormalities. During the chronic phase, there is a worsening of clinical signs, and other signs such as cachexia, widespread oedema, corneal opacity, incoordination and posterior paralysis are observed (Da Silva et al. 2010; Singh et al. 1993). Dogs may show severe neurological signs and the disease is often fatal in dogs and cats (Da Silva et al. 2010; Geering et al. 1995; Singh et al. 1993). Dogs may also appear clinically normal despite parasitaemia (Irwin and Jefferies 2004).

### **Diagnosis**

The parasite may be directly identified in stained thick or thin blood films, or wet mounts in the acute phase when the animal is parasitaemic. The parasite may also be identified in lymph node biopsy smears from fine-needle aspirates (OIE 2009). Serological tests include enzyme-linked immunosorbent assay and indirect fluorescent antibody testing (Geering et al. 1995). A real-time polymerase chain reaction assay using TaqMan and ribosomal DNA has been developed to detect *T. evansi*, allowing it to be easily distinguished from other *Trypanosoma* spp. (Taylor et al. 2008).

## Treatment

A number of drugs have been used for treatment and/or prevention of surra with conflicting reports on efficacy and curative doses. Dogs have been treated with a combination of quinpyramine sulphate and chloride, with some success (Singh et al. 1993). One study showed that treatment with diminazene aceturate resulted in a cure for six out of seven cats (Da Silva et al. 2009). Treatment tends to be more successful for clearing infection in acute rather than chronic cases for many affected species (Gillingwater et al. 2007).

### 4.19.3 Current biosecurity measures

There are no specific biosecurity measures for surra.

### 4.19.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by surra:

- Surra is widespread in Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East.
- Subclinical infection with *T. evansi* is known to occur in dogs and cats.
- Treatment cannot be relied upon to eliminate infection.
- Although surra is widespread, it affects dogs and cats uncommonly.
- The role that vectors play in the spread of *T. evansi* in association with dogs and cats is poorly defined and other modes of transmission (e.g. feeding on infected tissues) may be more important.
- There is no evidence that implicates dogs and cats in the epidemiology of *T. evansi*.

### 4.19.5 Conclusion

Based on the preceding factors, it was concluded that risk management measures for surra are not warranted for dogs, cats or their semen.

## References

CFSPH (The Center for Food Security and Public Health) (2009). Surra. CFSPH, Iowa State University, Ames.  
<http://www.cfsph.iastate.edu/Factsheets/pdfs/surra.pdf> (accessed 17 November 2010)

Da Silva AS, Wolkmer P, Costa MM, Zanette RA, Oliveira CB, Soares CDM, Otto MA, Santurio JM, Lopes STA, Monteiro SG (2010). Clinical aspects of cats experimentally infected with *Trypanosoma evansi*. *Comparative Clinical Pathology* 19:85–89.

- Da Silva AS, Zanette RA, Wolkmer P, Costa MM, Garcia HA, Lopes STA, Santurio JM, Teixeira MMG, Monteiro SG (2009). Diminazene aceturate in the control of *Trypanosoma evansi* infection in cats. *Veterinary Parasitology* 165:47–50.
- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National list of notifiable animal diseases. DAFF, Canberra.  
<http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable> (accessed 24 March 2011)
- Duscher G, Edelhofer R, Leschnik M, Joachim A (2010). Reisekrankheiten bei hund und katze (Travel-related diseases of dogs and cats). *Kleintierpraxis* 55:204 (abstract only).
- Geering WA, Forman AJ, Nunn MJ (1995). Surra. In *Exotic diseases of animals: a field guide for Australian veterinarians*. Australian Government Publishing Service, Canberra, pp. 380–384.
- Gillingwater K, Buscher P, Brun R (2007). Establishment of a panel of reference *Trypanosoma evansi* and *Trypanosoma equiperdum* strains for drug screening. *Veterinary Parasitology* 148:114–121.
- Hellebrekers LJ, Slappendel RJ (1982). Trypanosomiasis in a dog imported in The Netherlands. *Veterinary Quarterly* 4:182–186.
- Hendrix CM, Wohl JS, Bloom BC, Ostrowski SR, Benefield LT (1998a). International travel with pets. Part II. The threat of foreign pathogens. *Compendium on Continuing Education for the Practicing Veterinarian* 20:1239–1251.
- Hendrix CM, Wohl JS, Bloom BC, Ostrowski SR, Benefield LT (1998b). International travel with pets. Part III. Recognizing imported pathogens. *Compendium on Continuing Education for the Practicing Veterinarian* 20:1342–1348.
- Hoare CA (1972). Subgenus Trypanozoon. In *The trypanosomes of mammals: a zoological monograph*, Blackwell Scientific Publications, Oxford, pp. 554–604.
- Irwin PJ, Jefferies R (2004). Arthropod-transmitted diseases of companion animals in Southeast Asia. *Trends in Parasitology* 20:27–34.
- OIE (World Organisation for Animal Health) (2009). *Trypanosoma evansi* infections (including surra). OIE technical disease cards. OIE, Paris.  
[http://www.oie.int/fileadmin/Home/eng/Animal\\_Health\\_in\\_the\\_World/docs/pdf/TRYPANO\\_EVANSI\\_FINAL.pdf](http://www.oie.int/fileadmin/Home/eng/Animal_Health_in_the_World/docs/pdf/TRYPANO_EVANSI_FINAL.pdf) (accessed 18 January 2011)
- OIE (World Organisation for Animal Health) (2010). *Trypanosoma evansi* infections (surra). *Manual of diagnostic tests and vaccines for terrestrial animals* 2011.  
[http://www.oie.int/fileadmin/Home/eng/Health\\_standards/tahm/2.01.17\\_TRYPANO.pdf](http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.01.17_TRYPANO.pdf) (accessed 7 December 2010)

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007). Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats*, 10th edn, Radostits OM, Gay CC, Hinchcliff KW, Constable PD (eds), Saunders Elsevier, Edinburgh, pp. 1483–1540.

Reid SA, Husein A, Partoutomo S, Copeman DB (2001). The susceptibility of two species of wallaby to infection with *Trypanosoma evansi*. *Australian Veterinary Journal* 79:285–288.

Seddon HR, Albiston HE (1967a). Stable fly. In *Diseases of domestic animals in Australia: part 2. arthropod infestations (flies, lice and fleas)*, 2nd edn, Department of Health, Canberra, pp. 10–11.

Seddon HR, Albiston HE (1967b). Tabanid (March) flies. In *Diseases of domestic animals in Australia: part 2. arthropod infestations (flies, lice and fleas)*, 2nd edn, Department of Health, Canberra, p. 33.

Singh B, Kalra IS, Gupta MP, Nauriyal DC (1993). *Trypanosoma evansi* infection in dogs: seasonal prevalence and chemotherapy. *Veterinary Parasitology* 50:137–141.

Taylor TK, Boyle DB, Bingham J (2008). Development of a TaqMan PCR assay for the detection of *Trypanosoma evansi*, the agent of surra. *Veterinary Parasitology* 153:255–264.

## 4.20 Tularaemia

### 4.20.1 Background

Tularaemia is a zoonosis caused by *Francisella tularensis*, a Gram-negative coccobacillus of the family Francisellaceae. Tularaemia occurs endemically in temperate regions of the Northern Hemisphere, predominantly between 20° and 70° latitude, including North America, continental Europe, the former Soviet Union, China, Japan and Korea (Greene and DeBey 2006). The highly virulent *F. tularensis* (type A) is found in North America, whereas the less virulent *F. tularensis holarctica* (type B) is found throughout the Northern Hemisphere. The natural reservoir hosts are certain rodents and lagomorphs, and their associated parasites, which includes ticks, mosquitoes, fleas and horseflies. In 2002, *F. tularensis novicida*, a less virulent subspecies, was isolated from a human in Australia (Whipp et al. 2003). Two human cases due to *F. tularensis holarctica* were identified in 2011 in wildlife carers who had been in close contact with possums (DoHA: Australian NFP 2011).

*F. tularensis* (type A) is not present in Australia or New Zealand. Tularaemia is an OIE-listed disease of multiple species (OIE 2012) and is nationally notifiable in Australia (DAFF 2011).

## 4.20.2 Technical information

### Epidemiology

The epidemiology is complex with dogs and cats most commonly infected through the bite of an infected tick, or by ingestion of, or direct contact with, tissues of an infected wildlife host (e.g. rabbits, hares, rodents). Adult ticks and, less commonly, nymphal stages are the most important in transmitting the bacterium to dogs, cats and humans. The incubation period is approximately two days and disease, if it occurs, is self-limiting. Reports of naturally acquired infections in dogs are rare; cats appear to be more susceptible to clinical disease following infection (Feldman 2003; Greene and DeBey 2006; Meinkoth et al. 2004). There is no evidence that dogs or cats are carriers of *F. tularensis* (Greene and DeBey 2006; Magnarelli et al. 2007).

Species of ticks implicated in tularaemia include those from four genera: *Amblyomma*, *Dermacentor*, *Haemaphysalis* and *Ixodes*. Ticks remain infected throughout their lifetime and transmit *F. tularensis* transtadially. Biting flies from the family Tabaniae can also act as mechanical vectors. The role of fleas in tularaemia is uncertain. Fleas can remain infected for weeks but reportedly do not readily transmit the organism between animals (CFSPH 2009; WHO 2007).

Transmission of *F. tularensis* occurs through a variety of modes, including bites of infected vectors, direct contact with infected animals or tissues, ingestion of the organism in contaminated food or drink, and through inhalation (Feldman 2003; Greene and DeBey 2006).

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

### Clinical signs

Infected dogs typically show only minor clinical signs of mild pyrexia, anorexia and listlessness that may resolve within five days (Greene and DeBey 2006; Meinkoth et al. 2004). In cats, a more severe range of clinical signs has been observed, including pyrexia, marked depression, localised or generalised lymphadenopathy, palpable splenomegaly and hepatomegaly, icterus, acute oral ulcerations, draining abscesses and occasionally death (Feldman 2003; Greene and DeBey 2006).

### Diagnosis

A microscopic agglutination (MA) antibody test is the most commonly used diagnostic serology procedure for detecting exposure to *F. tularensis* (Greene and DeBey 2006). Titres from 1:140 to 1:160 are typical of recent infection in dogs (Johnson 1944; Schmid et al. 1983). After an MA test, serum antibody titres above 1:20 have been reported in felines (Woods et al. 1998). Enzyme-linked immunosorbent assay techniques have been developed to detect antibodies to specific *F. tularensis* antigens in infected people, but their usefulness for testing canine and feline sera is uncertain (Bevanger et al. 1989).

## Treatment

No substantial reports have been made on antimicrobial therapy of canine or feline tularaemia. Treatment for feline tularaemia is largely adapted from human medicine, with the administration of aminoglycoside, chloramphenicol, fluoroquinolone or tetracycline antibiotics advocated. These should reportedly be administered for 2–4 weeks (Shaw 2005). In one cat, surgical removal of a subcutaneous mass followed by treatment with amoxicillin-clavulanate was curative (Valentine et al. 2004)

### 4.20.3 Current biosecurity measures

There are no specific biosecurity measures for tularaemia. Current biosecurity measures for the management of external parasites that may act as disease vectors are as follows:

#### Vector management

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be subject to a thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks and fleas.

### 4.20.4 Risk review

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by Tularaemia:

- *F. tularensis* (type A) is known to be present in North America
- *F. tularensis* (type B) appears to be present in wildlife in Australia
- Tularaemia is an OIE-listed and nationally notifiable disease in Australia.
- Both dogs and cats can be infected and can play a role in the transmission of disease.
- Infected ticks are the most common source of transmission of *F. tularensis* to dogs and cats.

### 4.20.5 Conclusion

Based on the preceding key points, it was concluded that risk management measures for tularaemia caused by *F. tularensis* (type A) continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for tularaemia caused by *F. tularensis* are not warranted for dog or cat semen.

The following biosecurity measures would provide appropriate risk management for dogs and cats.

#### **Pre-export measures**

##### *Vector management*

- As for pre-export measures for external parasite control (see 4.13.5).

#### **Post-arrival measures**

##### *Vector management*

- As for post-arrival measures for external parasite control (see 4.13.5).
- To manage the biosecurity risk of tularaemia, dogs and cats may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

#### **References**

Bevanger L, Maeland JA, Naess AI (1989). Competitive enzyme immunoassay for antibodies to a 43,000-molecular-weight *Francisella tularensis* outer membrane protein for the diagnosis of tularemia. *Journal of Clinical Microbiology* 27:922–926.

CFSPH (2009) Tularemia.

<http://www.cfsph.iastate.edu/Factsheets/pdfs/tularemia.pdf> (Accessed 25 January 2013).

DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National notifiable diseases list of terrestrial animals December 2010, DAFF, Canberra.

[http://www.daff.gov.au/\\_\\_data/assets/pdf\\_file/0019/1015075/notifiable-diseases.pdf](http://www.daff.gov.au/__data/assets/pdf_file/0019/1015075/notifiable-diseases.pdf) (accessed 1 July 2011)

DoHA (Australian Government Department of Health and Ageing): Australian NFP (Australian National Focal Point) (2011). *Australian IHR Focal Point notification to World Health Organization: Tularaemia – Tasmania, November 2011*. DOHA: Australian NFP, Canberra.

Feldman KA (2003). Tularemia. *Journal of the American Veterinary Medical Association* 222:725–730.

Greene CE, DeBey BM (2006). Tularemia. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 446–451.

Johnson HN (1944). Natural occurrence of tularemia in dogs used as a source of canine distemper virus. *Journal of Laboratory and Clinical Medicine* 29:906–915.

Magnarelli L, Levy S, Koski R (2007). Detection of antibodies to *Francisella tularensis* in cats. *Research in Veterinary Science* 82:22–26.

Meinkoth KR, Morton RJ, Meinkoth JH (2004). Naturally occurring tularemia in a dog. *Journal of the American Veterinary Medical Association* 225:545–547.

OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)

Schmid GP, Kornblatt AN, Connors CA, Patton C, Camey J, Hobbs J, Kaufmann AF (1983). Clinically mild tularemia associated with tick-borne *Francisella tularensis*. *Journal of Infectious Diseases* 148:63–67.

Shaw S (2005) Other arthropod-borne infections of dogs and cats. In *Arthropod-borne infectious diseases of the dog and cat* (eds. Shaw SE, Day MJ) pp. 138–142. Lippincott, Williams & Wilkins, Baltimore.

Valentine BA, DeBey BM, Sonn RJ, Stauffer LR, Pielstick LG (2004). Localized cutaneous infection with *Francisella tularensis* resembling ulceroglandular tularemia in a cat. *Journal of Veterinary Diagnostic Investigation* 16:83–85.

Whipp MJ, Davis JM, Lum G, de Boer J, Zhou Y, Bearden SW, Petersen JM, Chu MC, Hogg G (2003). Characterization of a novicida-like subspecies of *Francisella tularensis* isolated in Australia. *Journal of Medical Microbiology* 52:839–842.

WHO (2007) *WHO guidelines on Tularaemia*. WHO/CDS/EPR/2007.7, World Health Organization, Geneva.

Woods JP, Crystal MA, Morton RJ, Panciera RJ (1998). Tularaemia in two cats. *Journal of the American Veterinary Medical Association* 212:81–83.

## 4.21 Yersiniosis

### 4.21.1 Background

Yersiniosis (plague) is caused by *Yersinia pestis*, a coccobacillus of the family Enterobacteriaceae. *Y. pestis* occurs on every continent except Australia, and exists in endemic foci in North and South America, Africa, Asia and the Middle East (Macy 2006).

*Y. pestis*, a zoonotic disease agent, infects more than 200 species of rodents and small mammals and about 1500 species of fleas. However, many mammalian and flea species are unlikely to play an epidemiologically significant role in transmission of *Y. pestis*—with only 31 species of fleas being proven vectors of yersiniosis and only 30 to 40 rodent species considered permanent natural reservoirs (Eisen and Gage 2009; Macy 2006; Perry and Fetherston 1997). Dogs are generally infected by the oral route or by flea bites, but are considered relatively resistant to infection. They can carry populations of infected fleas while showing few clinical signs. Cats are quite susceptible to infection and are a significant source of infection for people either by direct contact or by carrying infected fleas into contact with people (Macy 2006).

Yersiniosis is not an OIE-listed disease (OIE 2012) and is not a nationally notifiable disease in Australia (DAFF 2011).

#### 4.21.2 Technical information

##### Epidemiology

Transmission of *Y. pestis* is primarily by fleas that feed on rodents, but may also occur via direct contact with infected animals, inhalation of droplets produced by animals with plague pneumonia or ingestion of infected animals.

No evidence for venereal routes of transmission in dogs or cats was found in the scientific literature.

##### Clinical signs

Dogs can develop mild to moderate pyrexia shortly after infection and recover clinically without treatment within about a week (Macy 2006). Cats develop pyrexia, lethargy, anorexia, lymphadenopathy, abscesses, bacteraemia, septicaemia or pneumonia. The mortality rate in affected cats can be 50% (Gasper et al. 1993).

##### Diagnosis

*Y. pestis* is usually found in large numbers in infected tissues. Aseptically collected specimens of fluids, tissues, lymph node aspirates or blood should be submitted for culture. Examination via direct fluorescent antibody technique of air-dried impression smears of affected tissues provides a rapid, presumptive diagnosis with good reliability. Serology is often unreliable in detecting the presence of infection early in the course of disease. A passive haemagglutination assay is used to detect antibody specific to the F1 antigen of *Y. pestis* (Li et al. 2008). A four-fold rise in serial titres from specimens collected 10–14 days apart should be used to enable active infection to be differentiated from previous exposure because dogs and cats in endemic areas frequently have high titres that persist for more than 12 months (Macy 2006).

##### Treatment

Although there is an increase in the detection of multiple drug-resistant strains, *Y. pestis* is generally susceptible to a wide variety of antimicrobial agents (Dennis and Hughes 1997). This includes aminoglycosides, doxycycline, chloramphenicol and fluoroquinolones. It is recommended that antimicrobial therapy be administered for a minimum of 21 days. Oral administration of doxycycline (5-10mg/kg body weight twice daily decreasing to once daily) has been used to treat the earlier stages of disease and can reportedly be used for prophylaxis in exposed cats (Shaw 2005).

#### 4.21.3 Current biosecurity measures

There are no specific biosecurity measures for yersiniosis. Current biosecurity measures for the management of external parasites that may act as disease vectors are as follows:

##### Vector management

- At the time of blood sampling for ehrlichiosis, dogs must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be

applied according to manufacturer's instructions by a government-approved veterinarian.

- Within four days immediately before export, dogs and cats must be treated with a parasiticide effective against ticks and fleas on contact; the parasiticide must be applied according to manufacturer's instructions by a government-approved veterinarian.
- Within four days immediately before export, dogs and cats must be subject to thorough physical examination by a government-approved veterinarian and found to be visibly free from ticks and fleas.

#### **4.21.4 Risk review**

The following key points were drawn from the preceding information to inform the review of biosecurity risk presented by yersiniosis:

- Dogs are relatively resistant to infection by *Y. pestis*. Clinically healthy dogs are considered to pose a negligible risk of harbouring and transmitting *Y. pestis*.
- Cats are susceptible and there is a short incubation period between infection and expression of clinical disease (1–3 days).
- Cats are more likely than dogs to be severely affected and infection may be fatal in cats.
- Cats that are clinically healthy at pre-export inspection and remain clinically healthy during post-arrival quarantine are considered to pose a negligible risk of harbouring and transmitting *Y. pestis*.

#### **4.21.5 Conclusion**

Based on the preceding key points, it was concluded that risk management measures for yersiniosis continue to be warranted for dogs and cats. In addition, it was concluded that risk management measures for yersiniosis are not warranted for dog or cat semen.

The following biosecurity measures would provide appropriate risk management for dogs and cats.

##### **Pre-export measures**

###### *Vector management*

- As for pre-export measures for external parasite control (see 4.13.5).

##### **Post-arrival measures**

###### *Vector management*

- As for post-arrival measures for external parasite control (see 4.13.5).
- To manage the biosecurity risk of vector-borne disease, dogs and cats may be detained for an extended period of PAQ as required. Inspection and treatment of in-contact animals and/or facilities must be carried out to manage the risk of tick infestation.

## References

- DAFF (Australian Government Department of Agriculture, Fisheries and Forestry) (2011). National notifiable diseases list of terrestrial animals, December 2010. [http://www.daff.gov.au/\\_\\_data/assets/pdf\\_file/0019/1015075/notifiable-diseases.pdf](http://www.daff.gov.au/__data/assets/pdf_file/0019/1015075/notifiable-diseases.pdf) (accessed 1 July 2011)
- Dennis DT, Hughes JM (1997). Multidrug resistance in plague. *New England Journal of Medicine* 337:702–704.
- Eisen R, Gage K (2009) Adaptive strategies of *Yersinia pestis* to persist during inter-epizootic and epizootic periods. *Vet.Res.* 40: 1-14.
- Gasper PW, Barnes AM, Quan TJ, Benziger JP, Carter LG, Beard ML, Maupin GO (1993). Plague (*Yersinia pestis*) in cats: description of experimentally induced disease. *Journal of Medical Entomology* 30:20–26.
- Li B, Guo Y, Guo Z, Liang Y, Zhu Z, Zhou Q, Yan Y, Song Z, Yang R (2008). Serologic survey of the sentinel animals for plague surveillance and screening for complementary diagnostic markers to F1 antigen by protein microarray. *The American Journal of Tropical Medicine and Hygiene* 79:799–802.
- Macy D (2006). Plague. In *Infectious diseases of the dog and cat*, 3rd edn, Greene CE (ed.), Saunders Elsevier, St. Louis, pp. 439–446.
- OIE (World Organisation for Animal Health) (2012). OIE-listed diseases. OIE, Paris. <http://www.oie.int/en/animal-health-in-the-world/oie-listed-diseases-2012/> (accessed 16 January 2012)
- Perry RD, Fetherston JD (1997) *Yersinia pestis* - Etiologic agent of plague. *Clinical Microbiology Reviews* 10: 35-66.
- Shaw S (2005) Other arthropod-borne infections of dogs and cats. In *Arthropod-borne infectious diseases of the dog and cat* (eds. Shaw SE, Day MJ) pp. 138-142. Lippincott, Williams & Wilkins, Baltimore.

## 5 Risk management

---

Risk management aims to reduce the likelihood that importation of a commodity (animal product or live animal) would lead to the entry, establishment and/or spread of a disease agent of biosecurity concern.

The *Terrestrial animal health code* (the Code) states in Article 2.1.5. (OIE 2011b) that:

Risk management is the process of deciding upon and implementing measures to achieve the Member's appropriate level of protection (ALOP).

In the risk review of diseases, technical information on risk factors relevant to the biosecurity risk (encompassing entry, exposure, establishment or spread, and consequences) associated with the importation of dogs and cats was reviewed. Where a significant change in a risk factor was identified since the previous review of a disease agent, the change was evaluated in the context of Australia's current biosecurity measures. If current biosecurity measures were considered to be inadequate to achieve Australia's ALOP then alternative and/or complementary risk management options were evaluated.

Thus, re-evaluation of the risk factors relevant to each disease enabled conclusions to be drawn regarding whether the associated biosecurity risk was being managed sufficiently to achieve Australia's ALOP.

For the diseases listed below, this review concluded that risk management is not warranted to achieve Australia's ALOP:

- canine pulmonary angiostrongylosis
- Chagas' disease
- nagana
- Nipah virus encephalitis
- Rift Valley fever
- surra.

For certain disease agents, the review concluded that risk management is warranted and that current biosecurity measures are appropriate to achieve Australia's ALOP. This conclusion was drawn for the following diseases or disease agents:

- canine piroplasmosis
- canine influenza virus
- hepatozoonosis
- leishmaniasis
- Lyme disease
- screw-worm fly myiasis

- tularaemia
- yersiniosis.

For some disease agents, the review concluded that risk management is warranted, and that alternative and/or complementary risk management options are available that would achieve Australia's ALOP. This conclusion was drawn for the following disease agents:

- canine brucellosis
- canine monocytic ehrlichiosis
- leptospirosis
- rabies.

For some vector-borne disease agents, the review concluded that risk management is warranted for external parasites known to act as disease vectors (e.g. ticks, fleas) and that generic biosecurity measures that include post-arrival quarantine, are appropriate for such diseases. This conclusion was drawn for the following diseases:

- Lyme disease
- hepatozoonosis
- tularaemia
- yersiniosis.

## 5.1 Risk management options

The Code includes the following definition in Article 2.1.6. (OIE 2011b) regarding evaluation of risk management options:

Option evaluation—the process of identifying, evaluating the efficacy and feasibility of, and selecting measures in order to reduce the risk associated with an importation in line with the Member's ALOP. The efficacy is the degree to which an option reduces the likelihood and/or magnitude of adverse health and economic consequences.

Biosecurity measures that are appropriate to manage the biosecurity risks associated with the importation of dogs and cats and their semen necessarily have the following limitations:

- Dogs and cats are live animals and semen is viable genetic material. Therefore, biosecurity measures appropriate for nonviable products (e.g. heat treatment) are not appropriate.
- Dogs and cats may be subclinically infected with disease agents of concern and therefore detection of disease by physical examination is unreliable.
- Disease agents of concern in live animals differ in epidemiological characteristics (e.g. incubation period) and therefore the duration of quarantine detention appropriate for one disease agent may not be appropriate for another disease agent.

- Diagnostic test sensitivity may be inadequate to detect some disease agents of concern and therefore would not provide an appropriate risk management measure.
- Vaccinations and/or preventive treatments may be effective in preventing clinical disease, but may not be effective in preventing infection and shedding of a disease agent.
- Vectors and/or iatrogenic factors can transmit disease agents to other animals and therefore measures to manage the biosecurity risk of disease vectors may be appropriate for some disease agents.

The following risk management options (seven pre-export measures and five post-arrival measures) were included in the evaluation of options to manage the biosecurity risk associated with the importation of dogs and cats.

### **Pre-export measures**

#### **Identification**

Identification of animals with an International Organization for Standardization (ISO)-compatible microchip (radio frequency identification device) is an effective and practical technology that provides unique identification of individual animals. This technology provides a highly reliable identification system that enables all pre-export documentation to be verified as relating to each individual dog or cat presented for importation.

In this review, identification of dogs and cats intended for export to Australia with an ISO-compatible microchip device is recommended as a generic risk management measure to apply to the importation of all dogs and cats into Australia. Therefore, microchip identification is not reviewed in evaluating disease-specific risk management measures.

#### **Approved country**

Countries, administrative regions and territories from which Australia permits the importation of dogs and cats are referred to in this policy review as approved countries. Countries approved under the previous policy are listed in Appendix 1.

For countries to be approved to export dogs and cats to Australia a number of criteria are assessed. These include the animal health status of the country, animal health legislation, the effectiveness of systems for control over certification of animals and products, the effectiveness of veterinary and laboratory services and the standard of reporting disease outbreaks to the World Organisation for Animal Health (OIE). Australia has issued *Guidelines for the approval of countries to export animals (including fish) and their products to Australia* (Animal Quarantine Policy Memorandum 1999/62).

The importation of dogs and cats into Australia from countries approved by the Australian Government Department of Agriculture provides a high level of confidence that each animal imported is prepared in accordance with Australia's

biosecurity measures. This policy review recommends that the approved country system be retained as a generic risk management measure for the importation of dogs and cats.

#### **Country or area freedom**

For certain disease agents, requiring that importation is only allowed from countries (or zones) that are free from the disease agent of biosecurity concern may be an appropriate biosecurity measure.

Where such a pre-export measure was deemed necessary, a continuous period of residency in a disease-free country(ies) or zone(s) at least equivalent to the incubation period of the disease agent was specified.

Determination of disease freedom must be to a standard consistent with that recommended in Article 1.4.6. in the Code (OIE 2011b) or to an equivalent standard for those diseases not listed in the Code. To accept that a country or area is free from a given disease the Department of Agriculture must have a knowledge of the country's Competent Authority (e.g. the government veterinary service or equivalent) and be assured that the Competent Authority has the surveillance, monitoring, control and reporting capacity that is appropriate for the disease agent.

#### **Vaccination**

For certain disease agents, vaccination before export may be an appropriate measure to manage the biosecurity risk associated with importation of dogs and/or cats. Where such a pre-export measure was deemed necessary, the standards of vaccine production should be consistent with methods and quality management standards specified in the *Manual of diagnostic tests and vaccines for terrestrial animals* (OIE Manual) (OIE 2011a) for OIE-listed diseases, or to an equivalent standard for those diseases not listed in the Code.

In addition, although not relevant to disease agents of biosecurity concern, dogs and cats being prepared for export to Australia should be vaccinated against significant infectious diseases that do occur in Australia (e.g. canine distemper, canine parvovirus, kennel cough, infectious canine hepatitis, feline enteritis) to minimise the risk of an animal becoming infected while undergoing post-arrival quarantine.

#### **Diagnostic testing**

For certain disease agents, diagnostic testing before export may be an appropriate measure to manage the biosecurity risk associated with the importation of dogs and/or cats. Where such a pre-export measure was deemed necessary, testing would need to be conducted using methods specified in the OIE Manual (OIE 2011a) where such descriptions exist, and in an appropriately accredited laboratory that meets the standards specified by the OIE and is recognised by the Competent Authority in the country of export.

### **Preventive treatment(s)**

For certain disease agents, preventive treatments, such as anthelmintics, parasiticides and/or insect repellents, before export may be an appropriate measure to manage the biosecurity risk associated with the importation of dogs and cats. Where such a pre-export measure was deemed necessary, the standard of treatment must be with a commercial product of appropriate efficacy and registered for use in the country of export. Parasite resistance to chemical treatments was not considered in this risk review.

### **Documentation**

Each dog and cat must travel with an original international veterinary certificate consistent with the Code, signed by an official veterinarian from the exporting country.

### **Noncompliance at the border**

In the event that an imported dog or cat does not meet Australia's biosecurity requirements upon arrival in Australia, with due regard to animal welfare, that dog or cat may be:

- subjected to additional testing and/or treatment prescribed by Australian government authorities at the importer's expense, and detained in quarantine until approved for release by the Australian government authorities
- or
- exported at the importer's expense
- or
- euthanased without recompense.

### **Post-arrival measures**

#### **Post-arrival quarantine**

For certain disease agents, quarantine of dogs and cats on arrival in Australia may be an appropriate biosecurity measure. Post-arrival quarantine (PAQ) allows isolation and separation of imported dogs and cats from the Australian animal population. Dogs and cats can be monitored during PAQ for clinical signs of disease, the presence of exotic parasites, and tested and/or treated for disease agents of biosecurity concern.

In the event of detection of a pest or disease agent of biosecurity concern in the PAQ facility, contingency measures and standard operating procedures must be available. It must be possible to readily implement measures and procedures to contain and minimise the spread of the agent within the PAQ facility.

More stringent biosecurity measures should be implemented in PAQ facilities to prevent the transmission of infection within the facility and release of disease agents from the facility. Contingency measures may include, but not be restricted to:

- physical separation of affected animals from unaffected animals

- treatment of affected and in-contact animals
- increased movement restrictions on animals, fomites and personnel in and out of the facility
- hygienic operating practices, such as disinfection and decontamination procedures.

All equipment used in feeding, handling and treating dogs and cats must remain in PAQ, or be cleaned and disinfected on entry and before removal from PAQ.

Provided all biosecurity requirements are met, only dogs and cats that are deemed to be healthy and free from disease agents of biosecurity concern will be released from PAQ.

### **Diagnostic testing**

For certain disease agents, diagnostic testing of dogs and cats during PAQ may be an appropriate measure to manage the biosecurity risk associated with the importation of dogs and cats. The tests would need to be conducted using methods specified in the OIE Manual (OIE 2011a). Where such a post-arrival measure was deemed necessary, testing would need to be conducted using methods specified in the OIE Manual (OIE 2011a) or equivalent diagnostic techniques, where such descriptions exist. Testing should be conducted in an appropriately accredited laboratory that meets the standards specified by the OIE.

### **Preventive treatment(s)**

For certain disease agents, preventive treatments—such as anthelmintics, parasiticides and insect repellents—administered in PAQ may be an appropriate measure to manage the biosecurity risk associated with the importation of dogs and cats. Where such a post-arrival measure was deemed necessary, the standard of treatment must be with a commercial product of appropriate efficacy and registered for use in Australia. Some therapeutic treatments can adversely affect the sensitivity of diagnostic tests for disease agents of biosecurity concern. If a preventive treatment is required during PAQ, it must not be administered before diagnostic specimens that are required to enable an animal to be confirmed as eligible for release from PAQ, have been collected for laboratory submission. Therapeutic treatments for diseases not of biosecurity concern may be administered to dogs or cats during PAQ only after consultation with Australian Government authorities.

### **Contingency measures**

In the event that an imported dog or cat arrives in Australia and does not meet Australia's biosecurity requirements—either before entry into PAQ or during PAQ—and is suspected of and/or confirmed as being infected with a disease agent of biosecurity concern, then that dog or cat, and any or all dogs and cats in the PAQ facility may be:

- detained in quarantine for observation and subjected to additional testing and/or treatment prescribed by Australian government authorities at the importer's expense

- or
- exported at the importer’s expense
- or
- euthanased without recompense.

## 5.2 Biosecurity policy perspective

Under Australia’s biosecurity policy for the importation of dogs and cats (implemented in December 1995), importation is only permitted from approved countries. Country approval is based on a desk-based assessment of a country’s rabies status (with particular regard to dog-mediated rabies), its biosecurity requirements for the importation of dogs and cats, and its animal health services capacity.

The policy is rabies-centric but hazard-specific biosecurity measures also apply for canine brucellosis, canine ehrlichiosis, canine leishmaniasis, canine leptospirosis, canine piroplasmiasis and Nipah virus encephalitis. The country categorisation system has five categories of approved countries as outlined in Table 9. Pre-export requirements and PAQ requirements are category specific and vary in line with the differing level of risk. Countries approved under each of the five categories are listed in Appendix 1.

With respect to rabies virus, the five category system of approved countries has enabled the safe importation of dogs and cats into Australia and has met Australia’s ALOP.

Category 1 is restricted to New Zealand, Cocos (Keeling) Islands and Norfolk Island. Australia and New Zealand have bilaterally harmonised trade arrangements, with both countries being free from rabies. The Australia–New Zealand Closer Economic Relations Free Trade Agreement reflects the unique relationship between Australia and New Zealand; both countries cooperate closely to harmonise biosecurity policies.

Table 9 Categories of countries approved to export dogs and cats to Australia

Category	Assessment	Quarantine (days)
1	Rabies-free, dog and cat health status at least equivalent to Australia, adequate Competent Authority	Not required
2	Rabies-free, adequate Competent Authority	30
3	Rabies-free, Pacific island, Competent Authority poorly resourced or lacking	60
4	Rabies-affected, urban rabies well controlled, adequate Competent Authority	30–120 <sup>a</sup>
5	Rabies-affected, urban rabies endemic, adequate Competent Authority	210 <sup>b</sup>

<sup>a</sup> The quarantine period varies with the timing of pre-export serological testing.

<sup>b</sup> The quarantine period is shared between exporting country and Australia, with a minimum requirement of 30 days’ quarantine in Australia.

Category 2 countries are typically those countries recognised by the OIE as rabies virus-free. Category 3 of the classification system was established to recognise the rabies virus-free status of developing countries in the Pacific island region. Many of

these countries have a limited animal health services capacity and therefore do not fulfil the OIE definition for a rabies virus-free country. Although relatively few animals have been imported into Australia from Category 3 countries, rates of operational noncompliance are high and have resulted in significant operational difficulties, including the re-export of animals intended for importation.

Categories 4 and 5 of the classification system were established as a key outcome of the Bureau of Resource Sciences (BRS) review (BRS 1993) to enable the safe importation of dogs and cats from rabies-affected countries. Category 4 countries include Canada, the United States and the majority of countries in Western Europe. The Republic of South Africa is the only approved Category 5 country.

In any approved country classification system linked to Australia's biosecurity requirements, monitoring of noncompliance is a critical control point required to maintain system integrity and minimise the risk of an incursion. Where significant noncompliance issues substantially decrease the Department of Agriculture's confidence in the ability to safely import dogs and cats from an approved country, that country's approval may be suspended until the Department of Agriculture is satisfied that all issues of concern have been adequately addressed.

While the classification system implemented in 1995 for approved countries (and its underlying requirements) is effective from a biosecurity perspective, its complexity is a disadvantage. The extended PAQ periods ranging from 30 to 120 days, that are associated with Categories 2, 3, 4 and 5 countries, are expensive for animal importers and resource intensive to administer. In addition, a prolonged period of separation can be a great source of stress for both owners and their animals.

This policy review recommends that a simpler classification system for country approval, combined with appropriate biosecurity measures, would enable streamlined biosecurity requirements and a reduction in the duration of PAQ.

Taking into consideration factors including (i) the increased reliability of rabies vaccines, (ii) effective rabies management and control in approved countries (iii) the absence of any clinical cases of rabies in PAQ and (iv) an increasing demand by Australian pet owners to travel internationally with their companion animals, this policy review recommends that it is appropriate for Australia to adopt an approach to risk management for rabies with an increased reliance on vaccination as a principal biosecurity measure. An increased emphasis on 'offshore' management via rabies vaccination and serological testing to confirm vaccination efficacy would effectively enable dogs and cats to be imported from approved rabies-affected countries without the need for detention in PAQ.

This policy review recommends that the five category classification system implemented in 1995 be replaced by a three category classification system to simplify requirements for importation. Rabies status should continue to be used as a key determinant of country classification (see Table 10).

Table 10 Revised categories of countries approved to export dogs and cats to Australia

Category	Animal health status
1	Rabies-free <sup>a</sup> , with dog and cat health status at least equivalent to Australia
2	Other rabies-free <sup>a</sup> countries
3	All other approved countries

<sup>a</sup> recognised by the Department of Agriculture as rabies-free

This policy review makes the following recommendations for a revised three category classification system for approved countries:

Category 1: *Rabies-free, with dog and cat health status at least equivalent to Australia*

This category to comprise countries listed in Category 1 under the previous policy – Cocos (Keeling) Islands, New Zealand and Norfolk Island.

Category 2: *Other rabies-free countries*

This category to comprise countries listed in Categories 2 and 3 under the previous policy.

Category 3: *All other approved countries*

This category to comprise countries listed in Categories 4 and 5 under the previous policy.

Implementation of the recommended three category classification system would simplify requirements for the assessment of import permit applications as well as the post-arrival management of imported dogs and cats. Adoption of such an approach would effectively harmonise Australia’s risk management for rabies with that of New Zealand, simplifying arrangements for the importation of dogs from New Zealand.

This policy review recommends that the Department of Agriculture continues to recognise the rabies-free status of Pacific Island countries based on their historical freedom from rabies and the presence of adequate biosecurity systems to minimise the likelihood of entry of dogs and cats infected with rabies virus. It is recommended that the Department of Agriculture continue to monitor Pacific Island country animal health status and Veterinary Services capacity in collaboration with the Animal Health and Production Group of the Secretariat of the Pacific Community, located in Suva, Fiji.

## References

BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper. BRS, Canberra.

Fooks AR (2001). Keeping rabies out by surveillance strategies, vaccination and serology. In *Proceedings of the Southern and Eastern African rabies group / World Health*

*Organization Meeting*, 18–22 June 2001, Lilongwe, Malawi, World Health Organization, Geneva, pp. 131–135.

OIE (World Organisation for Animal Health) (2011a). *Manual of diagnostic tests and vaccines for terrestrial animals 2011*. OIE, Paris.  
[http://www.oie.int/eng/normes/mmanual/A\\_summry.htm](http://www.oie.int/eng/normes/mmanual/A_summry.htm) (accessed 7 July 2011).

OIE (World Organisation for Animal Health) (2011b). *Terrestrial animal health code 2011*. OIE, Paris.  
<http://www.oie.int/en/international-standard-setting/terrestrial-code/access-online/> (accessed 3 November 2011)

## 6 Biosecurity measures for dogs, cats and their semen

---

### Objective and scope

The biosecurity measures in this chapter apply to the importation of domestic dogs (*Canis lupus familiaris*) and domestic cats (*Felis catus*) and/or their semen from countries approved by the Department of Agriculture as eligible to export dogs and cats to Australia (Section 6.4).

The biosecurity measures described provide the hazard-specific biosecurity policy for the importation of dogs and cats and/or their semen from approved countries. The biosecurity measures are consistent with the recommendations of the relevant risk review chapter and provide the basis for operational conditions for the importation of dogs and cats and/or their semen into Australia.

Conditions for the importation of dogs and cats and/or their semen into Australia may include details of specific operational requirements for animal identification, transport, welfare, standards of treatment and certification, prohibited breeds and other specifications that are not included in the biosecurity policy.

### Approved countries

The Department of Agriculture assesses and determines which countries<sup>14</sup> are approved to prepare dogs and cats for export to Australia.

Dogs and cats that are either visiting or resident in non-approved countries may only become eligible for export to Australia by being transported to an approved country and meeting all Australian biosecurity requirements, including any test verification procedures that apply, from the approved country of export.

The protocol for country assessment is for the Chief Veterinary Officer (or equivalent) of a non-approved country to prepare and forward a submission that includes details of the country's animal health status, legislation, disease notification, biosecurity requirements, disease management and control programs and Veterinary Services capacity. For further information enquiries may be directed to:

Animal Biosecurity  
Department of Agriculture  
GPO Box 858  
CANBERRA ACT 2601  
Telephone: +61 2 6272 4465  
Facsimile: +61 2 6272 3399  
Email: [animal@daff.gov.au](mailto:animal@daff.gov.au)

---

<sup>14</sup> Countries, administrative regions and territories from which Australia permits the importation of dogs and cats and their semen are referred to as approved countries.

## General biosecurity measures for dogs and cats

The following general biosecurity measures apply to the importation of all dogs and cats:

1. Dogs and cats must be individually identified with a microchip.
2. Dogs and cats must only be directly exported to Australia from a country approved by the Department of Agriculture as eligible to export dogs and cats to Australia (hereinafter referred to as an approved country).
3. Dogs and cats must satisfy all hazard-specific requirements for importation from the relevant approved country.
4. Dogs and cats that are resident in a country not approved by the Department of Agriculture as eligible to export dogs and cats to Australia must only be exported to Australia after:
  - a. being transported to an approved country, and
  - b. satisfying all hazard-specific requirements for importation from the relevant country of export, including any test verification requirements that may apply.
5. Dogs and cats imported from countries other than New Zealand, Cocos (Keeling) Islands and Norfolk Island must complete a minimum post-arrival quarantine (PAQ) of ten days at a Department of Agriculture quarantine facility.
6. Dogs and cats must not be released from PAQ until all conditions for importation have been fulfilled.
7. If PAQ is required, dogs that fulfil the requirements of a disability assistance dog can complete a minimum PAQ period of ten days under quarantine surveillance at the handler's residence, provided the dog has met all other biosecurity requirements for importation.
8. All treatments, collection of specimens and examinations must be conducted by an approved representative<sup>15</sup> of the country of export.
9. All testing must be conducted in an approved country and in a laboratory recognised by the Competant Authority of the country of export.

Hazard-specific biosecurity risk management objectives and measures for the importation of dogs are detailed in Section 6.1, those for the importation of cats in Section 6.2, and those for the importation of dog and cat semen in Section 6.3.

---

<sup>15</sup> An approved representative of the country of export will generally be a government-approved veterinarian. In approved Pacific Island countries that do not have a government-approved veterinarian the Department of Agriculture may consider service delivery by a government-approved paraveterinarian.

## Equivalence

In accordance with Australia's international obligations under the Application of Sanitary and Phytosanitary Measures Agreement<sup>16</sup>, 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 would provide an equivalent level of sanitary protection, the Department of Agriculture will consider relevant submissions.

### 6.1 Hazard-specific biosecurity measures for dogs

#### 6.1.1 Pre-export requirements for dogs

The following hazard-specific risk management objectives and measures apply to the importation of dogs from approved countries.

#### Canine brucellosis

##### For breeding dogs<sup>17, 18</sup>

##### Objective(s)

- The dog is seronegative for *Brucella canis* by an appropriate test.
- The dog is free from clinical signs of canine brucellosis.

##### Specified measure(s)

- a. **Serology:** Within the 45 days immediately before export, a blood sample must be collected from the dog and tested using a rapid slide agglutination test (RSAT), a tube agglutination test (TAT), or an indirect fluorescent antibody test (IFAT) for *Brucella canis* with a negative result.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine brucellosis.

---

<sup>16</sup> The World Trade Organization Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement). [http://www.wto.org/english/tratop\\_e/sps\\_e/spsagr\\_e.htm](http://www.wto.org/english/tratop_e/sps_e/spsagr_e.htm) (Accessed 7 February 2012).

<sup>17</sup> Modified objectives and measures apply to dogs imported from New Zealand, Cocos (Keeling) Islands and Norfolk Island.

<sup>18</sup> Dogs older than 16 weeks of age that are not desexed are considered to be breeding dogs.

**Note 1:** An RSAT using 2-mercaptoethanol and a less mucoid (M-) variant of *Brucella canis* as antigen is recommended to reduce non-specific reactions.

**Note 2:** If RSAT, TAT or IFAT results are positive or inconclusive, a blood sample collected from the dog in the 45 days immediately before export must be tested using a cytoplasmic antigen agar gel immunodiffusion (CPAg-AGID) with a negative result.

**Note 3:** If the dog is mated or inseminated within the 30 days immediately before export, blood sample collection must be conducted at least 21 days after the date of last mating or insemination. Dogs that are more than 30 days pregnant are not eligible for export to Australia.

OR

**For breeding dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island**

**Objective(s)**

- The dog is imported from a country that is free from canine brucellosis due to *Brucella canis*.
- The dog is free from clinical signs of canine brucellosis.

**Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that canine brucellosis due to *Brucella canis* has not been confirmed in that country within the 12 months immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine brucellosis.

OR

**For desexed dogs**

**Objective(s)**

- The dog is desexed.

**Specified measure(s)**

- a. **Documentation:** Appropriate documentary evidence that the dog is desexed.

**Note:** If appropriate documentary evidence of desexing is not available then serological testing must be conducted as for breeding dog requirements.

### **Canine influenza (only applies to dogs imported from the United States)**

#### **Objective(s)**

- The dog has protective immunity against canine influenza.
- The dog is free from clinical signs of canine influenza.

#### **Specified measure(s)**

- a. **Vaccination:** Within 12 months and at least 14 days immediately before export, the dog must have a current vaccination status against canine influenza, in accordance with the vaccine manufacturer's recommendations.

AND

- b. **Examination:** Within five days immediately before export, the dog must be subjected to a thorough physical examination by a registered veterinarian and found to be free from clinical signs of canine influenza.

### **Canine monocytic ehrlichiosis**

#### **Objective(s)**<sup>19</sup>

- The dog is seronegative for *Ehrlichia canis* by an appropriate test.
- The dog is free from clinical signs of canine monocytic ehrlichiosis.

#### **Specified measure(s)**

- a. **Serology:** Within the 21 days immediately before export, a blood sample must be collected from the dog and tested using an indirect fluorescent antibody test (IFAT) for *Ehrlichia canis* with a negative result at a serum dilution of 1:40 (unless an alternative cut-off value for a positive result is specified by the testing laboratory and is approved by the Department of Agriculture).

AND

- b. **Treatment:** The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before IFAT blood sampling. The treatment must be repeated in accordance with the

---

<sup>19</sup> Modified objectives and measures apply to dogs that have been continuously resident in New Zealand, Cocos (Keeling) Islands, and/or Norfolk Island since birth or direct importation from Australia (whichever applies).

manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- c. **Examination:** At the time of blood collection and any subsequent treatment with an acaricide, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation with ticks<sup>20</sup>.

AND

- d. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine monocytic ehrlichiosis.

**Note:** If a tick is detected at any examination the tick must be removed and the following measures apply:

**Treatment and serology:**

- i. the dog must be re-treated with an acaricide effective against ticks on contact; and
- ii. a blood sample must be collected from the dog at least 21 days following parasite detection and tested using an IFAT for *Ehrlichia canis* with a negative result at a serum dilution of 1:40.

OR

**For dogs continuously resident since birth, or direct importation from Australia (whichever applies) in New Zealand, Cocos (Keeling) Islands or Norfolk Island**

**Objective(s)**

- Since birth, or direct importation from Australia (whichever applies), the dog has been continuously resident in a country free from vectors of canine ehrlichiosis.
- No cases of indigenously acquired canine monocytic ehrlichiosis have been reported in the country of export
- The dog is free from clinical signs of canine monocytic ehrlichiosis.

**Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:

---

<sup>20</sup> This means tick species that are vectors of *Ehrlichia canis*.

- i. the dog has been continuously resident since birth, or direct importation from Australia (whichever applies) in the relevant country; and
- ii. canine monocytic ehrlichiosis due to *Ehrlichia canis* has not been confirmed in the country of export within the 12 months immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority and found to be free from clinical signs of canine monocytic ehrlichiosis.

## Hepatozoonosis

### Objective(s)

- The dog is free from infestation with tick vectors of hepatozoonosis (*Hepatozoon* spp.).
- The dog is free from clinical signs of hepatozoonosis.

### Specified measure(s)

- a. **Treatment**<sup>21</sup>: The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation with ticks<sup>22</sup>.

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of hepatozoonosis.

---

<sup>21</sup> A modified single treatment measure applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>22</sup> Tick species that are vectors of *Hepatozoon* spp.

**NOTE:** If a tick is detected at examination it must be removed and the dog must be re-treated with an acaricide effective against ticks on contact.

## **Leishmaniasis**

### **Objective(s)**<sup>23</sup>

- The dog is seronegative for *Leishmania infantum* by an appropriate test.
- The dog is free from clinical signs of canine leishmaniasis.

### **Specified measure(s)**

- a. **Serology:** Within the 45 days immediately before export, a blood sample must be collected from the dog and tested using an enzyme linked immunosorbent assay (ELISA) or indirect fluorescent antibody test (IFAT) for *Leishmania infantum* with a negative result.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine leishmaniasis.

**OR**

**For dogs continuously resident since birth, or direct importation from Australia (whichever applies) in New Zealand, Cocos (Keeling) Islands or Norfolk Island**

### **Objective(s)**

- Since birth, or direct importation from Australia (whichever applies), the dog has been continuously resident in a country free from vectors of canine leishmaniasis.
- No cases of indigenously acquired canine leishmaniasis have been reported in the relevant country.
- The dog is free from clinical signs of canine leishmaniasis.

### **Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:
  - i. the dog has been continuously resident since birth, or direct importation from Australia (whichever applies) in the relevant country; and

---

<sup>23</sup> Modified objectives and measures apply to dogs that have been continuously resident in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island since birth or direct importation from Australia (whichever applies).

- ii. canine leishmaniasis due to *Leishmania infantum* has not been confirmed in the country of export within the 12 months immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine leishmaniasis.

## Leptospirosis

### Objective(s)<sup>24</sup>

- The dog is seronegative for *Leptospira interrogans* serovar Canicola by an appropriate test.  
OR  
The dog is treated with an approved course of antibiotics to eliminate inapparent infection with *Leptospira interrogans* serovar Canicola.  
OR  
The dog has a current vaccination status<sup>25</sup> against canine leptospirosis due to *Leptospira interrogans* serovar Canicola.
- The dog is free from clinical signs of canine leptospirosis.

### Specified measure(s)

- a. **Serology:** Within 45 days immediately before export, a blood sample must be collected from the dog and tested using a microscopic agglutination test (MAT) for *Leptospira interrogans* serovar Canicola with a negative result (less than 50% agglutination at a serum dilution of 1:100).

OR

- b. **Treatment:** The dog must be treated with doxycycline at a therapeutic dose rate of at least 5 mg/kg twice daily for 14 consecutive days in the 45 days immediately before export.

OR

- c. **Vaccination:** At least 14 days immediately before export, the dog must have a current vaccination status with an approved vaccine against *Leptospira interrogans* serovar Canicola in accordance with the manufacturer's recommendations.

---

<sup>24</sup> Modified objectives and measures apply to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>25</sup> Vaccination is not recommended in toy breeds due to an increased risk of adverse reaction.

AND

- e. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine leptospirosis.

OR

**For dogs imported from New Zealand, Cocos (Keeling) Islands and Norfolk Island**

**Objective(s)**

- The dog is imported from a country that is free from canine leptospirosis due to *Leptospira interrogans* serovar Canicola.
- The dog is free from clinical signs of canine leptospirosis.

**Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that canine leptospirosis due to *Leptospira interrogans* serovar Canicola has not been confirmed in the country of export within the 12 months immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the country of export and found to be free from clinical signs of canine leptospirosis.

## Lyme disease

### Objective(s)

- The dog is free from infestation by tick vectors of Lyme disease (*Borrelia burgderfori* senso latu).
- The dog is free from clinical signs of Lyme disease.

### Specified measure(s)

- a. **Treatment<sup>26</sup>:** The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from ticks<sup>27</sup>.

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of Lyme disease.

**NOTE:** If a tick is detected at examination it must be removed and the dog must be re-treated with an acaricide effective against ticks on contact.

---

<sup>26</sup> A modified single treatment measure applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>27</sup> Tick species that are vectors of *Borrelia burgderfori* senso latu.

## Parasites—external

### Objective(s)

- The dog is free from infestation with external parasites of biosecurity concern, including exotic parasite species and vectors of canine monocytic ehrlichiosis, hepatozoonosis, Lyme disease, piroplasmosis, tularaemia and yersiniosis.

### Specified measure(s)

- a. **Treatment**<sup>28</sup>: The dog must be treated with a parasiticide (or combination of parasiticides) effective against ticks and fleas on contact, with treatment to commence within the 45 days<sup>29</sup> immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination**: Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation with ticks and/or fleas of biosecurity concern.

**NOTE:** If a tick and/or flea is detected at examination it must be removed and the dog must be re-treated with a parasiticide effective against ticks and fleas on contact.

OR

### For dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island

### Objective(s)

- The dog is free from infestation with external parasites of biosecurity concern, including exotic species and vectors of canine monocytic ehrlichiosis, hepatozoonosis, Lyme disease, piroplasmosis, tularaemia and yersiniosis.

### Specified measure(s)

- a. **Treatment**: Within the five days immediately before export, the dog must be treated with a parasiticide (or combination of parasiticides) effective against ticks and fleas on contact.

---

<sup>28</sup> A modified single treatment measure applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>29</sup> The purpose of treatment is to minimise the likelihood of exposure to a vector of *Ehrlichia canis* before serological testing. The treatment period may be less than 45 days duration but should commence not less than 21 days before blood sample collection for *E. canis* serology.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the country of export and found to be visibly free from infestation with ticks and fleas of biosecurity concern.

**NOTE:** If a tick and/or flea is detected at examination it must be removed and the dog must be re-treated with a parasiticide effective against ticks and fleas on contact.

### Parasites—internal

#### Objective(s)

- The dog is free from infestation with exotic, intestinal cestodes (e.g. *Echinococcus multilocularis*) and nematodes.
- The dog is free from clinical signs of infestation by internal parasites.

#### Specified measure(s)

- a. **Treatment:** Within the 45 days immediately before export, the dog must be treated twice, with a product (or combination of products) registered for the control of intestinal cestodes and nematodes, in accordance with the manufacturer's recommendations (the product/s used must include praziquantel). The interval between treatments must be at least 14 days and the second treatment must be administered within the five days immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of infestation by internal parasites.

OR

### For dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island

#### Objective(s)

- The dog is free from infestation with exotic, intestinal cestodes (e.g. *Echinococcus multilocularis*) and nematodes.
- The dog is free from clinical signs of infestation with internal parasites.

### Specified measure(s)

- a. **Treatment:** Within the five days immediately before export, the dog must be treated once with a product (or combination of products) registered for the control of intestinal cestodes and nematodes, in accordance with the manufacturer's recommendations.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of infestation with internal parasites.

### Piroplasmosis

#### Objective(s)

- The dog is free from infestation by tick vectors of canine piroplasms. (*Babesia canis canis*, *Babesia canis rossi*, *Babesia conradae*, *Theileria annae*).
- The dog is free from clinical signs of canine piroplasmosis.

#### For dogs that have ever visited or resided in Africa<sup>30</sup>

- The dog is treated with an anti-babesial agent to eliminate inapparent infection with *Babesia canis rossi*.

### Specified measure(s)

- a. **Treatment<sup>31</sup>:** The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved

---

<sup>30</sup> For this policy, Africa is defined as any country or territory on the African mainland. Visited or resided refers to any part of a dog's life spent on the African mainland outside of quarantine control.

<sup>31</sup> A modified single treatment measure applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

representative of the Competent Authority of the country of export and found to be visibly free from ticks<sup>32</sup>.

**NOTE:** If a tick is detected at examination it must be removed and the dog must be re-treated with an acaricide effective against ticks on contact.

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine piroplasmiasis.

AND

**For dogs that have ever visited or resided in Africa:**

- d. **Treatment:** Within the 28 days immediately before export, the dog must be treated with imidocarb dipropionate in accordance with one of the following treatment options:
- i. a single treatment at a rate of 7.5 mg/kg body weight by subcutaneous injection; or
  - ii. two treatments at a rate of at least 6.0 mg/kg body weight by subcutaneous injection, administered at least 14 days apart.

### **Rabies—for importation from an approved country not recognised as rabies-free**

#### **Objective(s)**

- For at least 180 days immediately before export to Australia, the dog demonstrates protective immunity against rabies virus by an appropriate serological test
- The dog maintains a current vaccination status against rabies virus for at least 180 days immediately before export.
- The dog is free from clinical signs of rabies.

#### **Specified measure(s)**

- a. **Serology:** A blood sample must be collected from the dog at least 180 days immediately before export to Australia and tested using a rabies neutralising antibody titre test (RNATT)—either fluorescent antibody virus neutralisation (FAVN) test or rapid fluorescent focus inhibition test (RFFIT)—with a positive result of at least 0.5 IU/mL.

AND

---

<sup>32</sup> Tick species that are vectors of *Babesia canis rossi*.

- b. **Serology:** The date of sample collection for the RNATT serology result must be between 180 days and 24 months before the date of export to Australia.

**NOTE:** If the valid duration<sup>33</sup> of a previous RNATT serology result expires within 180 days before export to Australia, to maintain eligibility for importation, a blood sample must be collected before the date of RNATT expiry and tested using a RNATT with a positive result of at least 0.5 IU/mL.

AND

- c. **Vaccination:** For at least 180 days immediately before export to Australia the dog must maintain a current vaccination status against rabies, with an inactivated virus vaccine, in accordance with the manufacturer's recommendations.

AND

- d. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

### **Rabies—for importation from an approved country recognised as rabies-free**

#### **Objective(s)** <sup>34</sup>

- For at least 180 days immediately before export, or since birth, the dog avoids exposure to rabies virus by only visiting or residing in a country (or countries) recognised by the Department of Agriculture as rabies-free.
- The dog is free from clinical signs of rabies.

#### **Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:
- i. the country of export is officially free from rabies; and
  - ii. for at least 180 days immediately before export to Australia, or since birth, the dog has been continuously resident in an approved country (or countries) recognised by the Department of Agriculture as rabies-free.

---

<sup>33</sup> The Department of Agriculture recognises an RNATT to have a valid duration of 24 months from the date of sampling.

<sup>34</sup> Modified objectives and measures apply to dogs imported from New Zealand, Cocos (Keeling) Islands, or Norfolk Island.

**NOTE:** If an animal is not able to fulfil the specified measures for importation from an approved country recognised as rabies-free, the measures specified for importation from an approved country that is not recognised as rabies-free can be applied.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

OR

**For dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island**

**Objective(s)**

- Immediately before export to Australia, the dog avoids exposure to rabies virus by only visiting or residing in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island.
- The dog is free from clinical signs of rabies.

**Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:
- i. the country of export is officially free from rabies; and
  - ii. that immediately before export to Australia the dog has been resident in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island and has not been under quarantine restriction.

AND

- b. **Examination:** Within the five days immediately before export, the dog must be thoroughly examined by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**Rabies—short stay returning dog policy**

**For importation of Australian origin dogs returning within 180 days of export to an approved country not recognised as rabies-free**

**Objective(s)**

- Before departure from Australia, the dog demonstrates protective immunity against rabies virus by an appropriate serological test.
- Throughout the period of absence from Australia the dog maintains a current vaccination status against rabies virus.
- The dog is free from clinical signs of rabies.

#### Specified measure(s)

- a. **Serology:** Within the 18 months before departure from Australia a blood sample must be collected from the dog and tested using a rabies neutralising antibody titre test (RNATT)—either fluorescent antibody virus neutralisation (FAVN) test or rapid fluorescent focus inhibition test (RFFIT)—with a result of at least 0.5 IU/mL.

AND

- b. **Vaccination:** Throughout the period of absence from Australia the dog must maintain a current vaccination status against rabies, with an approved inactivated virus vaccine in accordance with the manufacturer's recommendations.

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**NOTE:** For an absence of 180 days or longer, measures for the importation of dogs from an approved country not recognised as rabies-free apply.

OR

**For importation of Australian origin dogs returning within 180 days of export to an approved country recognised as rabies-free<sup>35</sup>**

#### Objective(s)

- Since departure from Australia, the dog avoids exposure to rabies by only visiting or residing in a country (or countries) recognised as rabies-free.
- The dog is free from clinical signs of rabies.

#### Specified measure(s)

---

<sup>35</sup> These measures do not apply to dogs returning from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

- a. **Documentation:** An original (or certified copy) export certificate completed by an official veterinarian in Australia when the dog was exported to an approved rabies-free country.

AND

- b. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that the dog has only resided in that country since direct importation from Australia

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**NOTE:** For an absence of 180 days or longer, the standard measures for the importation of dogs from an approved country recognised as rabies-free apply.

## Screw-worm fly myiasis

### Objective

- The dog is free from cutaneous myiasis due to screw-worm fly larvae (*Chrysomya bezziana*; *Cochliomyia hominivorax*).

### Specified measure(s)

- a. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from cutaneous myiasis.

## Tularaemia

### Objective(s)

- The dog is free from infestation by tick vectors of tularaemia (*Francisella tularensis* type A).
- The dog is free from clinical signs of tularaemia.

### Specified measure(s)

- a. **Treatment**<sup>36</sup>: The dog must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination**: Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from ticks<sup>37</sup>.

AND

- c. **Examination**: Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of tularaemia.

**NOTE:** If a tick is detected at examination it must be removed and the dog must be re-treated with an acaricide effective against ticks on contact.

## Yersiniosis

### Objective(s)

- The dog is free from infestation by flea vectors of yersiniosis (*Yersinia pestis*).
- The dog is free from clinical signs of yersiniosis.

### Specified measure(s)

- a. **Treatment**<sup>38</sup>: The dog must be treated with a parasiticide effective against fleas on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination**: Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved

---

<sup>36</sup> A modified single treatment applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>37</sup> Tick species that are vectors of *Francisella tularensis* type A.

<sup>38</sup> A modified single treatment applies to dogs imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

representative of the Competent Authority of the country of export and found to be visibly free from infestation with fleas<sup>39</sup>.

AND

- c. **Examination:** Within the five days immediately before export, the dog must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of yersiniosis.

**NOTE:** If a live flea is detected at examination it must be removed and the dog must be re-treated with a parasiticide effective against fleas on contact.

### 6.1.2 Post-arrival quarantine requirements for dogs

#### Post-arrival quarantine

Dogs imported from approved countries other than New Zealand, Cocos (Keeling) Islands and Norfolk Island, must undergo a minimum post-arrival quarantine (PAQ) period of 10 days at a Department of Agriculture quarantine facility.<sup>40</sup>

Within two working days of entry into the PAQ facility, the dog must be examined by a Department of Agriculture veterinary officer and have its identity confirmed by microchip scan.

Within two working days before release from PAQ control, the dog must again be subject to veterinary examination.

In addition, each of the following biosecurity measures apply:

#### *Parasites—external*

- a. Within two working days of arrival at the PAQ facility, the dog must be thoroughly examined by a Department of Agriculture veterinary officer and found to be free from infestation with external parasites of biosecurity concern, including exotic species and vectors of canine monocytic ehrlichiosis, hepatozoonosis, Lyme disease, piroplasmosis, tularaemia and yersiniosis.

AND

- b. If an external parasite is detected at examination, it must be removed and identified.

AND

---

<sup>39</sup> Flea species that are vectors of *Yersinia pestis*.

<sup>40</sup> Dogs that qualify as an assistance dog may complete post-arrival quarantine under quarantine surveillance at the handler's residence.

- c. If identified as an external parasite of biosecurity concern the following general measures apply:
  - i. The dog must be treated with a parasiticide effective on contact against ticks and fleas in accordance with the manufacturer's recommendations.
  - ii. Dogs that have been in close proximity to a dog carrying an external parasite of biosecurity concern in the quarantine facility or during transport, must also be examined for external parasites and the pens treated using a registered pyrethroid product in accordance with the manufacturer's recommendations.

*Canine monocytic ehrlichiosis*

- a. As for PAQ examination and treatment measures for external parasites.

AND

- b. If a tick is detected at examination that is a vector of *Ehrlichia canis*, the tick must be removed and the dog treated with an acaricide effective against ticks on contact. The dog must be detained in PAQ and a blood sample collected at least 21 days after the date of export to Australia and tested using an indirect fluorescent antibody test (IFAT) for *E. canis*. The dog must have a negative result at a serum dilution of 1:40 before it is released from PAQ.

*Hepatozoonosis*

- a. As for PAQ examination and treatment measures for external parasites.

*Lyme disease*

- a. As for PAQ examination and treatment measures for external parasites.

*Piroplasmosis*

- a. As for PAQ examination and treatment measures for external parasites.

*Screw-worm fly myiasis*

- a. As for PAQ examination and treatment measures for external parasites.

*Tularaemia*

- a. As for PAQ examination and treatment measures for external parasites.

*Yersiniosis*

- a. As for PAQ examination and treatment measures for external parasites.

## 6.2 Hazard-specific biosecurity measures for cats

### 6.2.1 Pre-export requirements for cats

The following hazard-specific risk management objectives and measures apply to the importation of cats from approved countries.

#### Lyme disease

##### Objective(s)

- The cat is free from infestation with vectors of Lyme disease (*Borrelia burgderfori* sensu lato).
- The cat is free from clinical signs of Lyme disease.

##### Specified measure(s)

- a. **Treatment**<sup>41</sup>: The cat must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination**: Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from ticks<sup>42</sup>.

AND

- c. **Examination**: Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of Lyme disease.

**NOTE:** If a tick is detected at examination it must be removed and the cat must be re-treated with an acaricide effective against ticks on contact.

---

<sup>41</sup> A modified single treatment applies to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>42</sup> Tick species that are vectors of *Borrelia burgderfori* sensu lato.

## Parasites—external

### Objective(s)<sup>43</sup>

- The cat is free from infestation with external parasites of biosecurity concern, including exotic parasite species and vectors of Lyme disease, tularaemia and yersiniosis.

### Specified measure(s)

- a. **Treatment:** The cat must be treated with a parasiticide (or combination of parasiticides) effective against ticks and fleas on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation by ticks and fleas of biosecurity concern.

**NOTE:** If a tick or flea is detected at examination it must be removed and the cat must be re-treated with a parasiticide effective against ticks and fleas on contact.

OR

## For cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island

### Objective(s)

- The cat is free from infestation with external parasites of biosecurity concern, including exotic parasite species and vectors of Lyme disease, tularaemia and yersiniosis.

### Specified measure(s)

- a. **Treatment:** Within the five days immediately before export, the cat must be treated with a parasiticide (or combination of parasiticides) effective against ticks and fleas on contact.

AND

---

<sup>43</sup> Modified objectives and measures apply to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation with ticks and fleas of biosecurity concern.

**NOTE:** If a tick or flea is detected at examination it must be removed and the cat must be re-treated with a parasiticide effective against ticks and fleas on contact.

## Parasites—internal

### Objective(s)

- The cat is free from infestation by exotic, intestinal cestodes (e.g. *Echinococcus multilocularis*) and nematodes.
- The cat is free from clinical signs of infestation by internal parasites.

### Specified measure(s)<sup>44</sup>

- a. **Treatment:** Within the 45 days immediately before export, the cat must be treated twice with a product (or combination of products) registered for the control of intestinal cestodes and nematodes in accordance with the manufacturer's recommendations (the product/s used must include praziquantel). The interval between treatments must be at least 14 days and the second treatment must be administered within the five days immediately before export.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of infestation by internal parasites.

OR

## For cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island

### Objective(s)

- The cat is free from infestation by exotic, intestinal cestodes (e.g. *Echinococcus multilocularis*) and nematodes.
- The cat is free from clinical signs of infestation by internal parasites.

---

<sup>44</sup> A modified single treatment measure applies to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

### Specified measure(s)

- a. **Treatment:** Within the five days immediately before export, the cat must be treated once with a product (or combination of products) registered for the control of intestinal cestodes and nematodes, in accordance with the manufacturer's recommendations.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of infestation by internal parasites.

### Rabies—for importation from an approved country not recognised as rabies-free

#### Objective(s)

- For at least 180 days immediately before export to Australia, the cat demonstrates protective immunity against rabies virus by an appropriate serological test
- The cat maintains a current vaccination status against rabies virus for at least 180 days immediately before export.
- The cat is free from clinical signs of rabies.

### Specified measure(s)

- a. **Serology:** A blood sample must be collected from the cat at least 180 days before export to Australia and tested using a rabies neutralising antibody titre test (RNATT)—either fluorescent antibody virus neutralisation (FAVN) test or rapid fluorescent focus inhibition test (RFFIT)—with a positive result of at least 0.5 IU/mL.

AND

- b. **Serology:** The date of sample collection for the RNATT serology result must be between 180 days and 24 months before the date of export to Australia.

**NOTE:** If the valid duration<sup>45</sup> of a previous RNATT serology result expires within 180 days before export to Australia, to maintain eligibility for importation, a blood sample must be collected before the date of RNATT expiry and tested using an RNATT with a positive result of at least 0.5 IU/mL.

AND

---

<sup>45</sup> The Department of Agriculture recognises an RNATT to have a valid duration of 24 months from the date of sampling.

- c. **Vaccination:** For at least 180 days immediately before export to Australia the cat must maintain a current vaccination status against rabies virus, with an approved vaccine (either a recombinant or inactivated virus vaccine), in accordance with the manufacturer's recommendations.

AND

- d. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

### **Rabies—for importation from an approved country recognised as rabies-free**

#### **Objective(s)**

- For at least 180 days<sup>46</sup> immediately before export, or since birth, the cat avoids exposure to rabies virus by only visiting or residing in a country (or countries) recognised by the Department of Agriculture as rabies-free.
- The cat is free from clinical signs of rabies.

#### **Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:
- i. the country of export is officially free from rabies; and
  - ii. for at least 180 days immediately before export to Australia, or since birth, the cat has been continuously resident in an approved country (or countries) recognised by the Department of Agriculture as rabies-free.

**NOTE:** If an animal is not able to fulfil the specified measures for importation from an approved country recognised as rabies-free, the measures specified for importation from an approved country that is not recognised as rabies-free can be applied.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

---

<sup>46</sup> Modified objectives and measures apply to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

OR

**For cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island**

**Objective(s)**

- Immediately before export, or since birth, the cat avoids exposure to rabies virus by only visiting or residing in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island.
- The cat is free from clinical signs of rabies.

**Specified measure(s)**

- a. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that:
  - i. the country of export is officially free from rabies; and
  - ii. that immediately before export to Australia, the cat has been resident in New Zealand, Cocos (Keeling) Islands and/or Norfolk Island and has not been under quarantine restriction.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be thoroughly examined by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**Rabies—short stay returning cat policy**

**For importation of Australian origin cats returning within 180 days of export to an approved country that is not recognised as rabies-free**

**Objective(s)**

- Before departure from Australia, the cat demonstrates protective immunity against rabies virus by an appropriate serological test.
- Throughout the period of absence from Australia the cat maintains a current vaccination status against rabies virus.
- The cat is free from clinical signs of rabies.

**Specified measure(s)**

- a. **Serology:** Within the 18 months before departure from Australia a blood sample must be collected from the cat and tested using a rabies neutralising antibody titre test (RNATT)—either fluorescent antibody virus neutralisation (FAVN) test or rapid fluorescent focus inhibition test (RFFIT)—with a result of at least 0.5 IU/mL].

AND

- b. **Vaccination:** Throughout the period of absence from Australia the cat must maintain a current vaccination status against rabies, with an approved vaccine (either a recombinant or inactivated virus vaccine) in accordance with the manufacturer's recommendations.

AND

- c. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**NOTE:** For an absence of 180 days or longer, the standard measures for the importation of cats from an approved country not recognised as rabies-free apply.

OR

**For importation of Australian origin cats returning within 180 days of export to a country recognised as rabies-free<sup>47</sup>**

Objective(s)

- Since departure from Australia, the cat avoids exposure to rabies virus by only visiting or residing in a country (or countries) recognised as rabies-free.
- The cat is free from clinical signs of rabies.

**Specified measure(s)**

- a. **Documentation:** An original (or certified copy) export certificate completed by an official veterinarian in Australia when the cat was exported to an approved rabies-free country.

AND

- b. **Documentation:** Certification by an approved representative of the Competent Authority of the country of export that the cat has only resided in that country since direct importation from Australia.

AND

- c. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative

---

<sup>47</sup> This does not apply to cats returning from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

of the Competent Authority of the country of export and found to be free from clinical signs of rabies.

**NOTE:** For an absence of 180 days or longer, the standard measures for the importation of cats from an approved country recognised as rabies-free apply.

### **Screw-worm fly myiasis**

#### **Objective (s)**

- The cat is free from cutaneous myiasis due to screw-worm fly larvae (*Chrysomya bezziana*; *Cochliomyia hominivorax*).

#### **Specified measure(s)**

- a. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from cutaneous myiasis.

### **Tularaemia**

#### **Objective(s)**

- The cat is free from infestation by tick vectors of tularaemia (*Francisella tularensis* type A).
- The cat is free from clinical signs of tularaemia.

#### **Specified measure(s)**

- a. **Treatment<sup>48</sup>:** The cat must be treated with an acaricide effective against ticks on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from ticks<sup>49</sup>.

AND

---

<sup>48</sup> A modified single treatment measure applies to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>49</sup> This means tick species that are vectors of *Francisella tularensis* type A.

- c. **Examination:** Within the five days immediately before export, the cat must be subjected to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of tularaemia.

**NOTE:** If a tick is detected at examination it must be removed and the cat must be re-treated with an acaricide effective against ticks on contact.

## Yersiniosis

### Objective(s)

- The cat is free from infestation by flea vectors of yersiniosis (*Yersinia pestis*).
- The cat is free from clinical signs of tularaemia.

### Specified measure(s)

- a. **Treatment**<sup>50</sup>: The cat must be treated with a parasiticide effective against fleas on contact, with treatment to commence at least 21 days immediately before export. The treatment must be repeated in accordance with the manufacturer's recommendations to maintain continued effectiveness until at least the day of export.

AND

- b. **Examination:** Within the five days immediately before export, the cat must be subject to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be visibly free from infestation by fleas<sup>51</sup>.

AND

- c. **Examination:** Within the five days immediately before export, the cat must be subject to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of yersiniosis.

**NOTE:** If a flea is detected at examination it must be removed and the cat must be re-treated with a parasiticide effective against ticks on contact.

---

<sup>50</sup> A modified single treatment measure applies to cats imported from New Zealand, Cocos (Keeling) Islands or Norfolk Island.

<sup>51</sup> This means flea species that are vectors of *Yersinia pestis*.

## 6.2.2 Post-arrival quarantine requirements for cats

### Post-arrival quarantine

Cats imported from an approved country other than New Zealand, Cocos (Keeling) Islands and Norfolk Island will be subject to a minimum post-arrival quarantine (PAQ) period of 10 days at a Department of Agriculture quarantine facility.

Within two working days of entry into the PAQ facility, the cat must be examined by a Department of Agriculture veterinary officer and have its identity confirmed by microchip scan.

Within two working days before release from PAQ control, the cat must again be subject to veterinary examination.

In addition, each of the following biosecurity measures apply.

#### *External parasites*

- a. Within two working days of arrival at the PAQ facility, the cat must be thoroughly examined by a Department of Agriculture veterinary officer and found to be visibly free from infestation by external parasites of biosecurity concern, including exotic parasite species and vectors of Lyme disease, tularaemia and yersiniosis.

AND

- b. If an external parasite is detected at examination, it must be removed and identified.

AND

- c. If identified as an external parasite of biosecurity concern the following general measures apply:
  - i. The cat must be treated with a parasiticide effective on contact against ticks and fleas in accordance with the manufacturer's recommendations.
  - ii. Cats that have been in close proximity to a cat carrying an external parasite of biosecurity concern in the quarantine facility or during transport, must also be examined for external parasites and the pens treated using a registered pyrethroid product in accordance with the manufacturer's recommendations.

#### *Lyme disease*

- a. As for PAQ examination and treatment measures for external parasites.

#### *Screw-worm fly myiasis*

- a. As for PAQ examination and treatment measures for external parasites.

#### *Tularaemia*

- a. As for PAQ examination and treatment measures for external parasites.

*Yersiniosis*

- a. As for PAQ examination and treatment measures for external parasites.

### 6.3 Hazard-specific biosecurity measures for dog and cat semen <sup>52</sup>

Frozen dog or cat semen, processed in either pellets or straws, may only be imported into Australia from countries approved by the Department of Agriculture for the direct importation of dogs and cats.

The following hazard-specific biosecurity risk management objectives and measures apply to the importation into Australia of dog and cat semen from approved countries.

#### Canine brucellosis (donor dogs only)

##### Objective(s)

- The donor dog is seronegative for *Brucella canis* by an appropriate test.
- The donor dog is free from clinical signs of canine brucellosis.

##### Specified measure(s)

- a. **Serology:** Between 30 and 45 days following the last collection of semen in the consignment for export to Australia, a blood sample must be collected from the donor dog and subjected to a rapid slide agglutination test (RSAT), a tube agglutination test (TAT) or an indirect fluorescent antibody test (IFAT) with a negative result.

AND

- b. **Examination:** Between 30 and 45 days after the last collection of semen in the consignment for export, the donor must be subject to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine brucellosis.

**Note 1:** An RSAT using 2-mercaptoethanol and a less mucoid (M-) variant of *B. canis* as antigen is recommended to reduce non-specific reactions.

**Note 2:** If a positive or inconclusive RSAT, TAT or IFAT result occurs, a blood sample collected from the dog between 30 and 45 days following the last collection of semen in the consignment for export to Australia must be subjected to a cytoplasmic antigen agar gel immunodiffusion (CPAg-AGID) with a negative result.

---

<sup>52</sup> These biosecurity measures are generic. The Department of Agriculture may consider the development of country-specific measures for the importation of dog semen on request from the Competent Authority of an exporting country.

## Leptospirosis (donor dogs only)

### Objective(s)

- The donor dog is seronegative for *Leptospira interrogans* serovar Canicola by an appropriate test.  
OR  
The dog has protective immunity (a current vaccination status)<sup>53</sup> against canine leptospirosis due to *Leptospira interrogans* serovar Canicola.
- The donor dog is free from clinical signs of canine leptospirosis.

### Specified measure(s)

- a. **Serology:** Between 30 and 45 days following the last collection of semen in the consignment for export to Australia, a blood sample must be collected from the donor dog and tested using a microscopic agglutination test (MAT) for *Leptospira interrogans* serovar Canicola with a negative result (less than 50% agglutination at a serum dilution of 1:100).

OR

- b. **Vaccination:** Not less than 14 days before the first collection of semen in the consignment for export to Australia, the dog must have a current vaccination status against *Leptospira interrogans* serovar Canicola with an approved, inactivated vaccine, in accordance with the manufacturer's recommendations.

**NOTE:** The vaccination status against *Leptospira interrogans* serovar Canicola must be current until at least the date of the last collection of semen in the consignment for export to Australia.

AND

- d. **Examination:** Between 30 and 45 days after the last collection of semen in the consignment for export, the donor must be subject to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of canine leptospirosis.

## Leishmaniasis (donor dogs only)

### Objective(s)

- The dog is seronegative for *Leishmania infantum* by an appropriate test.

---

<sup>53</sup> Vaccination is not recommended in toy breeds due to an increased risk of adverse reaction.

- The dog is free from clinical signs of canine leishmaniasis.

**Specified measure(s)**

- a. **Serology:** Between 30 and 45 days following the last collection of semen in the consignment for export to Australia, a blood sample must be collected from the donor dog and tested using either an indirect fluorescent antibody test (IFAT) or enzyme-linked immunosorbent assay (ELISA) for *Leishmania infantum* with a negative result.

AND

- b. **Examination:** Between 30 and 45 days following the last collection of semen in the consignment for export, the donor dog must be subject to a thorough physical examination by an approved representative of the Competent Authority of the country of export and found to be free from clinical signs of leishmaniasis.

## 6.4 Revised category listings of approved countries<sup>54</sup>

**Category 1:** *Rabies-free with dog and cat health status at least equivalent to Australia*

Cocos (Keeling) Islands, New Zealand, Norfolk Island.

- No post-arrival quarantine required

**Category 2:** *Other rabies-free countries*

American Samoa, Bahrain, Barbados, Christmas Island, Cook Islands, Falkland Islands, Federated States of Micronesia, Fiji, French Polynesia, Guam, Hawaii, Iceland, Japan, Kiribati, Mauritius, Nauru, New Caledonia, Niue, Palau, Papua New Guinea, Samoa, Singapore, Solomon Islands, Kingdom of Tonga, Tuvalu, Vanuatu, Wallis and Futuna

- Minimum of 10 days post-arrival quarantine

**Category 3:** *All other approved countries*

Antigua & Barbuda, Argentina, Austria, Bahamas, Belgium, Bermuda, British Virgin Islands, Brunei, Bulgaria, Canada, Canary & Balearic Islands, Cayman Islands, Chile, the Republic of Croatia, the Republic of Cyprus, Czech Republic, Denmark, Finland, France, Germany, Gibraltar, Greece, Greenland, Guernsey, Hong Kong, Hungary, Ireland, Isle of Man, Israel, Italy, Jamaica, Jersey, Kuwait, Latvia, Luxembourg, Macau, Malta, Malaysia (Peninsular, Sabah & Sarawak only), Monaco, Montenegro, the Netherlands, Netherlands Antilles & Aruba, Norway, Poland, Portugal, Puerto Rico, Qatar, the Republic of South Africa, Reunion, Saipan, Serbia, Seychelles, Slovakia, Slovenia, South Korea, Spain, St Kitts & Nevis, St Lucia, St Vincent & the Grenadines, Sweden, Switzerland (including Liechtenstein), Taiwan, Trinidad and Tobago, the United Arab Emirates, the United Kingdom, the United States (including the District of Columbia, Northern Mariana Islands, Puerto Rico and the Virgin Islands of the United States)<sup>55</sup>, the United States Virgin Islands, Uruguay.

- Rabies vaccination and rabies neutralising antibody titre test required for importation
- Minimum of 10 days post-arrival quarantine

END BIOSECURITY MEASURES

---

<sup>54</sup> Countries, administrative regions and territories from which Australia permits the importation of dogs and cats and their semen are referred to as approved countries.

<sup>55</sup> Guam and Hawaii are rabies-free and classified as Category 2 countries



# Appendix 1—Current category listings

---

## Current category listings of approved countries (at 19 July 2013)

### Category 1

Approved rabies-free countries requiring no quarantine:

- Cocos (Keeling) Islands, New Zealand, Norfolk Island.

### Category 2

Approved rabies-free countries —minimum of 30 days quarantine:

- Bahrain, Barbados, Falkland Islands, Fiji, French Polynesia, Guam, Hawaii, Iceland, Japan, Mauritius, New Caledonia, Norway (excluding Spitsbergen/Svalbard), Singapore, Vanuatu.

### Category 3

Approved rabies-free island countries —minimum of 60 days quarantine:

- American Samoa, Cook Islands, Federated States of Micronesia, Papua New Guinea, Solomon Islands, Wallis and Futuna, Western Samoa.

Approved rabies-free island countries and territories that may not have an official veterinary service—minimum of 60 days quarantine:

- Christmas Island, Kiribati, Nauru, Niue, Palau, Kingdom of Tonga, Tuvalu.

### Category 4

Approved countries and territories recognised by the Australian Government as countries and territories in which dog-mediated rabies is absent or well controlled—minimum of 30 days quarantine and rabies vaccination:

- Antigua and Barbuda, Argentina, Austria, Bahamas, Belgium, Bermuda, British Virgin Islands, Brunei, Bulgaria, Canada, Canary and Balearic Islands, Cayman Islands, Chile, the Republic of Croatia, the Republic of Cyprus, Czech Republic, Denmark, Finland, France, Germany, Gibraltar, Greece, Greenland, Guernsey, Hong Kong, Hungary, Ireland, Isle of Man, Israel, Italy, Jamaica, Jersey, Kuwait, Latvia, Lichtenstein, Luxembourg, Macau, Malta, Monaco, Montenegro, the Netherlands, Netherlands Antilles and Aruba, Peninsular Malaysia, Poland, Portugal, Puerto Rico, Qatar, Reunion, Sabah, Saipan, Sarawak, Serbia, Seychelles, Slovak Republic, Slovenia, South Korea, Spain, St Kitts and Nevis, St Lucia, St Vincent and the Grenadines, Sweden, Switzerland (including Liechtenstein), Taiwan, Trinidad and Tobago, United Arab Emirates, the United Kingdom, the United States, the United States Virgin Islands, Uruguay.

## Category 5

Approved countries and territories recognised by the Australian Government as countries and territories in which dog-mediated rabies is endemic—minimum 210 days quarantine (180 days in the Republic of South Africa + 30 days in Australia) and rabies vaccination:

- Republic of South Africa.

**Note:** The time spent in quarantine in the Republic of South Africa determines the amount of time the animal must stay in quarantine in Australia. Cats and dogs must spend a minimum of 30 days in quarantine in Australia. To be eligible for the minimum 30 days in Australian quarantine, the animal must spend 180 days in quarantine in the Republic of South Africa.

## Appendix 2—Risk management for rabies

---

### Background

This section reviews biosecurity risk management options for rabies virus for imported dogs and cats. It identifies appropriate risk management options and examines which measure, or combination of measures, is required to achieve Australia's appropriate level of protection (ALOP) and meet international obligations.

In 1995, Australia's import conditions for dogs and cats were amended to allow importation from rabies-affected countries. No case of rabies has occurred either in post-arrival quarantine (PAQ) or following release from PAQ of animals imported under the amended import conditions.

To be eligible for export to Australia, dogs and cats from rabies-affected countries are required to satisfy the following pre-export requirements:

- individual identification by microchip
- residency for a continuous period of at least six months in an approved country
- vaccination against rabies virus with an approved inactivated vaccine
- a post-vaccination rabies virus neutralising antibody titre of at least 0.5 IU/mL between 60 days and 12 months before export.

For semen to be eligible for export to Australia, donor dogs and cats must have a current vaccination status against rabies, with an approved inactivated vaccine.

Most rabies-affected countries that are approved to export dogs and cats to Australia have active rabies virus management programs that minimise the occurrence of dog-mediated urban rabies. The Republic of South Africa is the only country approved to export dogs and cats to Australia in which dog-mediated rabies is endemic.

PAQ is a key biosecurity measure of Australia's import conditions for dogs and cats from approved, rabies-affected countries. A minimum PAQ duration of 30 days applies. Internationally, other rabies-free countries (e.g. New Zealand, the United Kingdom) have adopted measures for the importation of dogs and cats from rabies-affected countries to manage the rabies virus risk offshore, without the need for PAQ.

Vaccination with an approved rabies vaccine, combined with post-vaccination serology to confirm protective immunity against rabies virus, form the basis of these biosecurity measures. These measures are already required under Australia's conditions for the importation of dogs and cats from rabies-affected countries.

In 2000, the United Kingdom introduced its Pet Travel Scheme (PETS) to facilitate an increasing demand for the frequent movement of dogs and cats to and from Europe. The scheme had the following pre-entry eligibility requirements (Fooks 2001):

- individual animal identification by microchip
- vaccination with an approved rabies vaccine
- post-vaccination serological testing
- minimum six-month interval between confirmatory serological testing and importation
- importation from approved countries only
- border control at point of entry.

The United Kingdom did not require PAQ for dogs and cats that met the PETS eligibility requirements for importation. The PETS operated for 12 years (2000 to 2012) and no case of rabies occurred in either dogs or cats imported into the United Kingdom under the scheme.

In January 2012, the United Kingdom harmonised its import conditions for dogs and cats with that of the European Pet Movement Policy (EUPMP). A major difference between the EUPMP and PETS is that the latter required a minimum six month interval between confirmatory post-vaccination serology and importation, while under the EUPMP, importation of dogs and cats from EU-member states and other approved countries is allowed 21 days after vaccination, placing reliance on regular vaccination against rabies across the EU. In addition, the EUPMP allows for importation of dogs and cats from non-approved countries following vaccination against rabies virus and an interval of not less than three months after recording a post-vaccination rabies neutralising antibody titre test (RNATT) result of at least 0.5 IU/mL.

Despite the reduced interval between rabies vaccination and importation allowed under the EUPMP, it was estimated that the likelihood of rabies entry into the United Kingdom would remain very low in absolute terms (Goddard et al. 2010).

Empirical evidence indicates that the likelihood of introducing a rabies-infected animal increases with importation from rabies-affected countries in which veterinary resources are limited, animal movement controls are poor, and vaccination of dogs and cats against rabies is not widely practised.

### **Pre-export measures**

After reviewing the available technical literature on rabies in Section 4.16, and consideration of stakeholder submissions, the following pre-export measures are recommended to manage the risk of rabies virus presented by imported dogs and cats.

### **Identification**

Animals must be identifiable by a radio frequency identification device microchip that provides unique identification of individual animals. This is a generic risk management measure for all dogs and cats exported to Australia to enable export examinations, tests and treatments to be linked to each individual dog or cat.

### **Approved countries**

Animals may only be imported directly from countries approved by the Australian Government Department of Agriculture as eligible to export dogs and cats to Australia. This is a generic risk management measure for all dogs and cats exported to Australia. Dogs and cats in non-approved countries must be transported to an approved country to become eligible for importation into Australia.

### **Country or zone freedom**

The OIE *Terrestrial animal health code* (the Code) provides a definition for a rabies-free country, but not for a rabies-free zone (OIE 2011b).

Rabies has a broad global distribution with relatively few countries remaining free from rabies virus infection. Some countries (e.g. France, the United States) are infected with rabies virus but have states (e.g. Hawaii) or territories (e.g. French Polynesia, New Caledonia) that are free from rabies virus infection.

As rabies is endemic in the wildlife of many approved countries, the exporting authority in those countries (or zones) would not be able to certify country freedom. This includes European and North American countries from which the majority of dogs and cats are imported into Australia.

For the international movement of mammals, the World Organisation for Animal Health (OIE) defines the incubation period for rabies as six months (OIE 2011b). Accordingly, as per Article 8.10.3 of the Code, a continuous pre-export residency period of at least six months, or since birth, in a country (or countries) that is rabies-free, would enable expression of clinical signs of rabies in an infected dog or cat.

Provided that a country has adequate animal biosecurity systems to minimise the likelihood of entry of dogs and cats infected with rabies virus and, in the event of an incursion, adequate disease diagnostic and notification systems to report the occurrence of rabies, the measures recommended in the Code for importation of dogs or cats from countries that are recognised by Australia as rabies-free, would provide adequate assurance that an animal is not infected with rabies virus.

Countries' animal biosecurity systems, animal health services capacities and vulnerabilities to disease incursions vary significantly. These factors are considered in the Department of Agriculture's 'approved country' assessment system. Restricting the importation of dogs and cats to approved countries is intended to provide a high level of assurance that imported dogs and cats are prepared in accordance with Australia's biosecurity requirements.

## **Vaccination**

Single-dose vaccination against rabies virus infection is a practical and effective technique that provides protective immunity in both dogs and cats for up to three years.

To provide a high level of confidence of vaccine efficacy, the rabies vaccine used must meet OIE standards (OIE 2011a) and be approved by the animal health service in the country of export. Instances of vaccination failure should be anticipated due to a variety of causes (e.g. cold-chain failure, incorrect administration, immunocompromised vaccine recipients).

The occurrence of vaccination failure due to incorrect administration is likely to be reduced by requiring that, for export to Australia, a government-approved veterinarian vaccinates in accordance with the vaccine manufacturer's recommendations. An animal infected before or at the time of vaccination can continue to incubate rabies virus despite developing an antibody titre (Blancou et al. 1989). A waiting period of 180 days (equivalent to the incubation period of rabies virus) following the development of post-vaccinal immunity, is therefore required before importation.

Based on the preceding information, vaccination against rabies virus with a vaccine that meets the requirements specified by OIE is considered an appropriate risk management option. Most developed countries have established capacity to vaccinate dogs and cats against rabies virus as well as arrange post-vaccination serology to confirm vaccination efficacy.

As there is a potential for dogs and cats that are infected at the time of vaccination to incubate and develop clinical rabies, a waiting period of six months would be required following the development of post-vaccination immunity, before importation. There is also the potential for vaccination failure.

Due to the potential for dogs and cats to be infected at the time of vaccination, as well as the potential for vaccination failure, vaccination alone would not be a sufficient measure to achieve Australia's appropriate level of protection (ALOP).

## **Confirmatory testing**

The presence of rabies virus-specific virus-neutralising antibodies is a reliable indicator of effective vaccination (Moore and Hanlon 2010; Wilsmore et al. 2006).

A virus neutralising titre of at least 0.5 IU/mL, using either the fluorescent antibody virus neutralisation (FAVN) test or the rapid fluorescent focus inhibition test (RFFIT), is indicative of protective immunity in dogs and cats (Mansfield et al. 2004; OIE 2011a).

For quality assurance purposes, as prescribed in the OIE *Manual of diagnostic tests and vaccines for terrestrial animals* (OIE 2011a), serological testing for rabies virus

neutralising antibodies should be conducted at an appropriately accredited laboratory<sup>56</sup>, recognised by the Competent Authority of the exporting country.

Based on the preceding information, serological testing in an appropriately accredited laboratory in an approved country, using an OIE-recognised virus neutralisation test for rabies virus, is considered an appropriate risk management option to protect against vaccination failure.

An antibody titre of at least 0.5 IU/mL is considered to be a positive result. Blood should be collected for serological testing at least 25 days after vaccination to coincide with peak antibody titres.

Since there is a potential for an animal that responds to vaccination to be incubating rabies virus infection, vaccination combined with confirmatory serology of vaccination efficacy, would not be sufficient to achieve Australia's ALOP.

#### **Preventive treatment**

If an unvaccinated or recently vaccinated dog or cat that has not yet established protective immunity against rabies virus is bitten by a rabid animal, there are no post-exposure treatments for rabies virus infection that could be relied upon as a measure that would be sufficient to achieve Australia's ALOP.

For animals in a rabies-affected country that do not have a current history of protective immunity against rabies<sup>57</sup>, it is not possible to reliably determine that the animal has not been bitten by a rabid animal and may therefore be in the incubation phase of rabies virus infection.

#### **Australian origin animals**

For dogs and cats exported directly from Australia to a rabies-affected country, it was determined that an interval of at least 180 days between the date of blood sampling and the date of export to Australia, would not be required provided that the animal demonstrates protective immunity against rabies before departure from Australia.

For Australian origin animals it was considered that the following combination of measures, completed before departure from Australia, would be an appropriate risk management option sufficient to achieve Australia's ALOP:

- for the duration of the animal's absence from Australia, it must maintain a current vaccination status with an approved rabies vaccine, in accordance with the manufacturer's recommendations; and

---

<sup>56</sup> Accredited to ISO/IEC 17025 to conduct a rabies neutralising antibody test procedure prescribed by the World Organisation for Animal Health

<sup>57</sup> For Australian biosecurity requirements, a current history of protective immunity is defined as documentary evidence of a rabies virus neutralising antibody titre of at least 0.5 IU/mL between 180 days and 24 months before export, combined with a current vaccination status against rabies at least 180 days immediately before the date of export.

- a post-vaccination rabies neutralising antibody titre of at least 0.5 IU/mL not more than 24 months before the date of export to Australia.

### **Conclusion—pre-export measures**

Based on the preceding considerations, it was concluded that, for importation of dogs and cats from approved countries not recognised by Australia as rabies-free, the following combination of pre-export measures is an appropriate risk management option that would be sufficient to achieve Australia's ALOP:

- vaccination with an approved rabies vaccine (produced in accordance with the methods prescribed in the OIE Manual), in accordance with the manufacturer's recommendations; and
- a post-vaccination rabies neutralising antibody titre of at least 0.5 IU/mL within 24 months immediately before export; and
- an interval of at least 180 days and not more than 24 months between the date of blood sampling at which a rabies neutralising antibody titre of > 0.5 IU/mL is confirmed and the date of export to Australia; and
- the animal shows no clinical signs of rabies on the day of export.

### **Post-arrival measures**

The *Review of quarantine policy for dogs and cats, with particular reference to rabies* (BRS Review) (BRS 1993) recommended for:

- Category 1 countries—no PAQ
- Category 2 countries—a minimum of 30 days in PAQ to allow for diagnosis and reporting of a change of rabies status of the exporting country, which has effective quarantine policy and animal health programs
- Category 3 countries—a minimum of 90 days in PAQ to provide adequate safeguard for the exporting country, which has limited capacity for animal disease surveillance, diagnosis and reporting
- Category 4 countries, in which urban rabies is well controlled—a minimum of 30 days, but up to 120 days, in PAQ (depending on the date of successful rabies neutralising antibody titre testing) to address the incubation period
- Category 5 countries, in which urban rabies is endemic—a total of 210 days, with at least 30 days in PAQ.

Details of the previous policy are summarised in Table 9.

Since conditions for the import of dogs and cats from rabies-affected countries into Australia were introduced in 1995, there have been no cases of rabies either in PAQ or following release from PAQ. This Australian experience, combined with the United Kingdom's experience under the PETS program and New Zealand experience since implementation of modified biosecurity requirements for dogs and cats in 2011, provides strong empirical evidence that, subject to dogs and cats being prepared in

accordance with the pre-export measures specified, animals can be safely imported from rabies-affected countries without the need for PAQ.

### **Conclusion—post-arrival measures**

Based on the preceding considerations it is concluded that, as the specified combination of pre-export measures is an appropriate risk management option sufficient to achieve Australia's ALOP, PAQ measures for rabies virus would not be required.

PAQ for rabies would only be required for animals that have not been prepared in compliance with the specified pre-export measures.

### **Biosecurity measures for rabies**

Dogs and cats can only be imported into Australia directly from an approved country.

#### **Importation of dogs or cats from approved countries not recognised as rabies-free**

To achieve Australia's ALOP it is recommended that the following biosecurity measures should apply to dogs and cats imported from an approved country not recognised as rabies-free:

- a. A blood sample must be collected from the animal at least 180 days before export to Australia and tested using a rabies neutralising antibody titre test (RNATT)—either fluorescent antibody virus neutralisation (FAVN) test or rapid fluorescent focus inhibition test (RFFIT)—with a positive result of at least 0.5 IU/mL.

AND

- b. The date of sample collection for the RNATT serology result must be between 180 days and 24 months before the date of export to Australia.

AND

- c. Before export to Australia the animal must have a current vaccination status against rabies of at least 180 days duration, with an approved vaccine (inactivated virus or recombinant), in accordance with the manufacturer's recommendations.

AND

- d. Before export to Australia the animal must be free from clinical signs of rabies.

### Importation of dogs or cats from approved countries recognised as rabies-free

To achieve Australia's ALOP it is recommended that the following biosecurity measures should apply to dogs and cats imported from a country recognised as rabies-free:

- a. The dog or cat must be continuously resident for at least 180 days<sup>58</sup> immediately before export, or since birth, in a country (or countries) recognised by the Department of Agriculture as rabies-free.

AND

- b. Before export to Australia the animal must be free from clinical signs of rabies.

## References

- Blancou J, Soria Baltazar R, Artois M, Toma B, Rollin P (1989). Rabies despite pre- or post-exposure vaccination. In *Progress in rabies control: proceedings of the second international IMVI ESSEN/WHO symposium on 'New developments in rabies control'*, Essen, 5–7 July 1988 and report of the WHO Consultation on Rabies, Essen, 8 July 1988, Thraenhart O, Koprowski H, Bögel K, Sureau P (eds), Wells Medical, Kent, pp. 441–447.
- BRS (Bureau of Resource Sciences) (1993). *Review of quarantine policy for dogs and cats, with particular reference to rabies*, working paper. BRS, Canberra.
- Fooks AR (2001). Keeping rabies out by surveillance strategies, vaccination and serology. In *Proceedings of the Southern and Eastern African rabies group / World Health Organization Meeting*, 18–22 June 2001, Lilongwe, Malawi, World Health Organization, Geneva, pp. 131–135.
- Goddard A, Donaldson N, Kosmider R, Kelly L, Adkin A, Horton D, Fooks T, Breed A, Freuling C, Muller T, Shaw S, Hallgren G, Snary E (2010). A quantitative risk assessment on the change in likelihood of rabies introduction into the United Kingdom as a consequence of adopting the existing harmonised community rules for the non-commercial movement of pet animals.  
<http://archive.defra.gov.uk> (accessed 2 August 2011)
- Mansfield KL, Burr PD, Snodgrass DR, Sayers R, Fooks AR (2004). Factors affecting the serological response of dogs and cats to rabies vaccination. *The Veterinary Record* 154:423–426.

---

<sup>58</sup> Does not apply to importation from Cocos Islands, New Zealand and Norfolk Island.

Moore SM, Hanlon CA (2010). Rabies-specific antibodies: measuring surrogates of protection against a fatal disease. *PLoS Neglected Tropical Diseases* 4:e595.

OIE (World Organisation for Animal Health) (2011a). *Manual of diagnostic tests and vaccines for terrestrial animals*. OIE, Paris.  
[http://www.oie.int/eng/normes/mmanual/A\\_summry.htm](http://www.oie.int/eng/normes/mmanual/A_summry.htm) (accessed 7 July 2011)

OIE (World Organisation for Animal Health) (2011b). Rabies. *Terrestrial animal health code*. OIE, Paris.  
[http://www.oie.int/index.php?id=169&L=0&htmlfile=chapitre\\_1.8.10.htm](http://www.oie.int/index.php?id=169&L=0&htmlfile=chapitre_1.8.10.htm) (accessed 28 November 2011)

Wilsmore T, Hamblin C, Taylor N, Taylor W, Watson B (2006). *Qualitative veterinary risk assessment of the introduction of rabies into the United Kingdom: a report prepared for DEFRA*. Veterinary Epidemiology and Economics Research Unit, United Kingdom