Import risk analysis: Cats, dogs and canine semen

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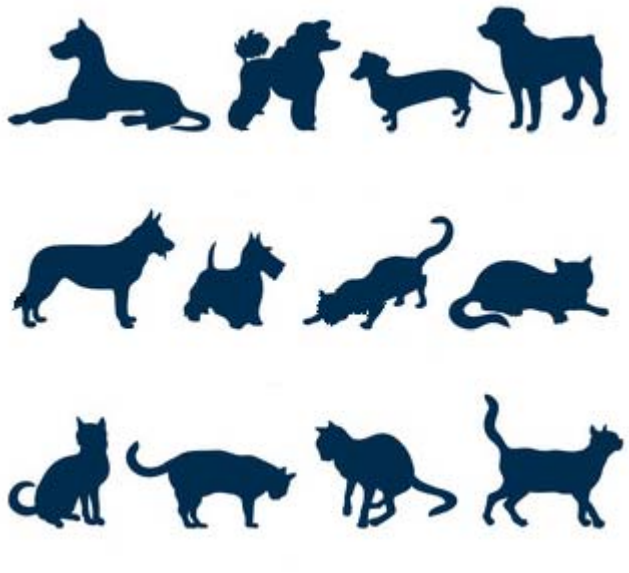

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Policy and Risk
MAF Biosecurity New Zealand

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Approved for general release

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Contents

ACRONYMS

CDC	United States Centers for Disease Control and Prevention
CF (T)	complement fixation (test)
CSIRO	The Commonwealth Scientific and Industrial Research Organisation
DEFRA	United Kingdom Department for Environment, Food and Rural Affairs
DNA	deoxyribonucleic acid
DOC	New Zealand Department of Conservation
ELISA	enzyme-linked immunosorbent assay
IFA(T)	indirect fluorescent antibody (test)
IHS	Import Health Standard
IM	intramuscular
IU	international unit
MAF	New Zealand Ministry of Agriculture and Forestry
MAFBNZ	Ministry of Agriculture and Forestry Biosecurity New Zealand
mcg	microgram
mg	milligram
mm	millimetre
OIE	World Organisation for Animal Health
PCR	polymerase chain reaction
RNA	ribonucleic acid
RT-PCR	reverse transcriptase polymerase chain reaction
WHO	World Health Organization

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Executive Summary

This risk analysis examines the risks involved with the importation of domestic cats (*Felis catus*), dogs (*Canis familiaris*) and canine semen from all countries.

A project team was established, with representatives from the Department of Conservation, Ministry of Health, Ministry of Fisheries, the New Zealand Food Safety Authority and Ministry of Agriculture and Forestry. In 2005 the project team finalised a preliminary hazard list on which this analysis was to be based. That list is included as Appendix 1. By eliminating organisms that could clearly be considered not to be hazards in the commodities, the list was refined, leading to a list of agents that require further consideration.

Risk management is not warranted for many agents because they are arthropod-borne, mainly through specific ticks, flies and mosquitoes that are not present in New Zealand. However, if new tick or mosquito species were to establish here, measures would be justified for many organisms which would otherwise be eliminated from the preliminary hazard list.

Further, many agents do not warrant specific safeguards beyond veterinary certification that the animals are clinically healthy. This is because many diseases have relatively short incubation periods and cause obvious and acute clinical signs. Excluding sick animals further reduces the likelihood of any disease introduction.

This document examines each of the agents of concern by applying MAF's standard risk analysis process. This begins with the hazard identification step, where the epidemiology of the disease including distribution, clinical signs, transmission, diagnosis and any available treatment is considered. As a result, each organism is classified as a potential hazard or not in the commodities.

Organisms identified as potential hazards are subjected to detailed individual risk assessments, by considering the likelihood of entry, exposure and the likely resulting adverse consequences. For organisms that are assessed to be hazards in the commodities, the risk management step considers options that could be used to effectively manage the risk.

The risk analysis concludes that the following agents pose non-negligible risks in imported cats, dogs and canine semen, and that sanitary measures can be justified for them:

- Canine brucellosis
- Leptospirosis
- Plague
- Salmonellosis
- Babesiosis
- Q Fever
- Filariosis
- Leishmaniosis
- Surra
- Canine transmissible venereal tumour
- Ectoparasitic infestations (fleas, leeches, lice, mites, ticks and fly larvae infestation)
- Endoparasites (cestodes, nematodes, acanthocephalans and trematodes)

- Rabies

Ticks are important vectors of exotic blood parasites and any tick on an imported animal could harbour debilitating human and animal pathogens. Many exotic ticks are likely to be able to establish in this country if introduced. Since many exotic blood parasites of dogs and cats are transmitted by ticks, it is strongly recommended that animals are subjected to effective measures to control the risk of importing ticks with the commodity.

Because of the developments in technology and the advancement in scientific knowledge of *Dirofilaria immitis*, one of the the risk management options presented for this organism is to replace the currently required microfilarial concentration test with an antigen ELISA as a screening test for importing dogs.

For similar reasons, one of the risk management options presented for *Babesia* spp. is to use a PCR test as well as the currently required serological test. The currently required examination of an ear margin blood smear is no longer considered to be a justifiable option.

In the case of *Ehrlichia canis* and Nipah virus, the risk analysis concludes that the risk posed by these organisms in cats and dogs is negligible. As a result, it is considered that the sanitary measures for these organisms in the current Import Health Standard are not warranted.

The risk posed by semen is assessed to be negligible for all diseases except rabies, leptospirosis and brucellosis. Therefore options for risk management are presented only for these three agents.

The risk management options presented in this draft risk analysis, and stakeholder views on them, will be taken into consideration in producing a final risk analysis and in the development of any import health standards for these commodities.

1. Introduction

Current Import Health Standards for domestic cats and dogs are restricted to certain countries. The importation of cats and dogs is increasing each year. In the period from May 2006 to April 2007, about 2000 cats and 3100 dogs were imported from approximately 40 countries. The majority of these (60%) came from Australia, followed by the United Kingdom (23 %) and then the USA (8 %) (Waite 2007). Requests to import from countries that are currently ineligible are becoming more frequent.

The preliminary hazard list comprising a comprehensive list of disease agents known to infect domestic cats and dogs was completed in 2005 and is attached as Appendix 1. Because of the complexity of the preliminary hazard list, it was considered appropriate to group organisms together for analysis, for example, groups of taxonomically related organisms are presented together in single chapters.

2. Scope and Commodity Definition

This risk analysis examines the biosecurity risks involved with the importation of domestic cats (*Felis catus*), dogs (*Canis familiaris*) and canine semen. Cats and dogs will have been certified on the day of travel to be showing no clinical signs of infectious or parasitic disease, and they will have been thoroughly groomed prior to travel to manage risks posed by weed seeds and ectoparasites.

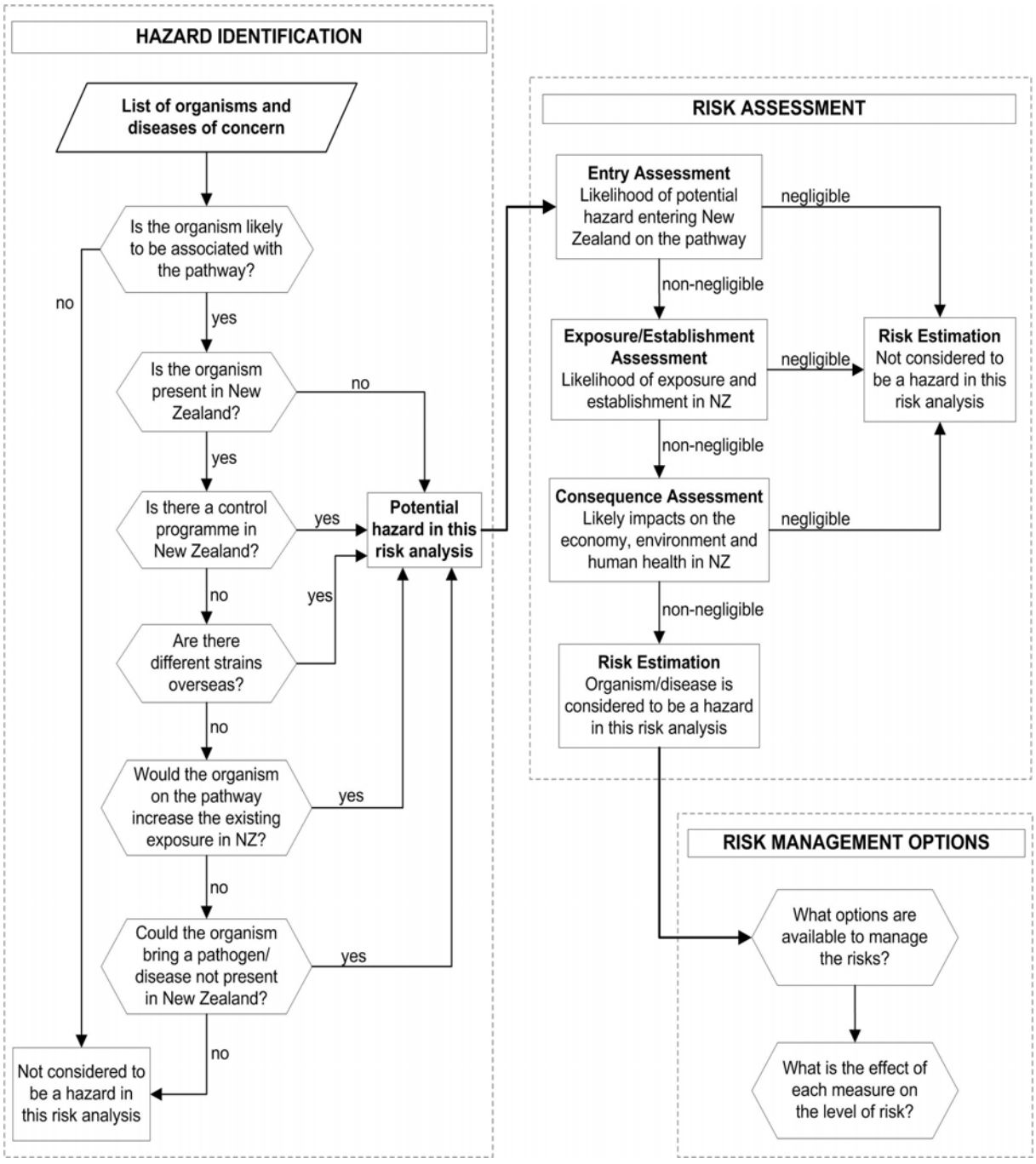
Therefore the biosecurity risks assessed in this document are viruses, prions, canine transmissible venereal tumour, ectoparasites, endoparasites, fungi, bacteria and blood parasites.

3. Risk Analysis Methodology

The methodology used in this risk analysis follows the MAF Biosecurity New Zealand risk analysis procedures (Biosecurity New Zealand 2006). These procedures combine the guidelines in the *Terrestrial Animal Health Code* (hereafter referred to as the *Code*) of the World Organisation for Animal Health and International Plant Protection Convention guidelines. The procedures provide a framework which adheres to the requirements set out under the World Trade Organisation Agreement on the application of Sanitary and Phytosanitary measures, 1995 and of the Biosecurity Act, 1993.

The process followed is shown in Figure 1.

Figure 1. The risk analysis process.



3.1. RISK ASSESSMENT

Risk assessment consists of:

a) *Entry assessment*: The likelihood of a pathogenic organism being imported with the animal.
b) *Exposure assessment*: The likelihood of animals or humans in New Zealand being exposed to the potential hazard.

c) *Consequence assessment*: The consequences of entry, establishment or spread of an imported organism.
d) *Risk estimation*: An estimation of the risk posed by the biological products based on the entry, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is a potential threat and risk management measures are justified to reduce the level of risk to an acceptable level.

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

3.2. RISK MANAGEMENT

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

As obliged under Article 3.1 of the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (the SPS Agreement), the measures adopted in IHSs will be based on international standards, guidelines and recommendations where they exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards can be applied if there is scientific justification, or if there is a level of protection that the member country considers is more appropriate following a risk assessment).

3.3. RISK COMMUNICATION

MAF releases draft import risk analyses for a six-week period of public consultation to verify the scientific basis of the risk assessment and to seek stakeholder comment on the risk management options presented. Stakeholders are also invited to present alternative risk management options that they consider necessary or preferable.

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

Following this process of consultation and review, the Imports Standards team of MAF Biosecurity New Zealand decides on the appropriate combination of sanitary measures to ensure the effective management of identified risks. These are then presented in a draft IHS which is released for a six-week period of stakeholder consultation. Stakeholder submissions in relation to the draft IHS are reviewed before a final IHS is issued.

4. Preliminary Hazard List

The first step in the risk analysis is hazard identification, the collation of a list of organisms that might be associated with cats and dogs.

A preliminary hazard list was consulted on in September 1999 and after further additions, consulted on in 2004. Final additions were made during 2005. The complete preliminary hazard list of all organisms that might be associated with cats and dogs is included in Appendix 1.

This risk analysis is based on the groups of organisms in the consulted preliminary hazard list and those described in the following sources:

- **Greene CE (ed) (2006).** *Infectious Diseases of the Dog and Cat*. Elsevier; St. Louis; USA; 1387 pp (3rd edition).
-
- **Shaw SE, Day MJ (eds) (2005).** *Arthropod-borne Infectious Diseases of the Dog and Cat*. Lippincott Williams and Wilkins; Baltimore; USA; 152 pp.

Because of the large number of organisms involved, they are generally considered in groups rather than individually. Thus, where convenient there are chapters on *Brucella* spp, fleas, flaviviridae etc. rather than a chapter for each organism. The groups of organisms identified from these sources are listed in Table 1.

Table 1. Initial hazard list of organism groups

BACTERIA
Anthrax
Borrelia spp.
Brucella spp.
Leptospira spp.
Melioidosis
Mycobacterium spp.
Plague
Salmonella spp.
Tularemia
Q fever

BLOOD PARASITE GROUPS
Babesia spp.
Bartonella spp.
Cytauxzoon felis
Ehrlichia spp.
Family *Anaplasmataceae* (including *Anaplasma* and *Ehrlichia* spp.)
Filarial spp.
Hepatozoon spp.
Leishmania spp.
Rickettsia spp.
Trypanosoma spp.

ECTOPARASITES
Fleas
Myiasis (fly larvae infestation)
Leeches
Lice
Mites
Ticks

ENDOPARASITES
Nematodes and acanthocephalans
Trematodes
Cestodes

FUNGAL AND ALGAL GROUPS
Aspergillus spp.
Blastomyces (Ajellomyces) dermatitidis
Coccidioides immitis
Histoplasma capsulatum
Pythium insidiosum
Rhinosporidium seeberi
Trichosporon spp.

VIRUS FAMILIES AND PRIONS
Bornaviridae
Bunyaviridae
Coronaviridae
Feline spongiform encephalopathy prion
Flaviridae
Herpesviridae
Orthomyxoviridae
Paramyxoviridae
Poxviridae
Reoviridae
Rhabdoviridae
Togaviridae

MISCELLANEOUS
Acanthamoeba spp.
Canine transmissible venereal tumour [A]
Exotic strain variations of the major endemic diseases (canine and feline parvovirus, feline herpesvirus and calicivirus, canine distemper and infectious canine hepatitis)

[A] As a result of a request in 2006 to add canine transmissible veneral tumour to the preliminary hazard list, this disease was assessed in the miscellaneous section.

Common agents that are universal in distribution and endemic throughout New Zealand have been excluded from further consideration. Because more pathogenic exotic strain variations of the major endemic diseases are known to exist overseas, these infectious agents are retained as potential agents of concern. Apart from *Mycobacterium bovis*, no other endemic organism that is subject to official control has been identified for retention.

Some organisms that appear in the preliminary hazard list are clearly not hazards. Brief information indicating why these can be removed is given below.

Feline spongiform encephalopathy (FSE) belongs to the transmissible spongiform encephalopathies (TSEs) and their aetiological agents are generally considered to be prions. These are infectious protein agents that affect the central nervous system causing neurodegenerative disease in humans and animals (European Commission 2000). FSE was first recognised during the bovine spongiform encephalopathy (BSE) epidemic in Britain. The first case in a felid was diagnosed in 1990 (Leggett et al 1990). There is no evidence that FSE occurs in any manner other than through ingestion of contaminated food containing the BSE agent. TSEs have long incubation periods and development of clinical signs in the cat takes about five years. There is no evidence of vertical transmission of TSEs in the cat.

Approximately 90 cases of FSE were reported worldwide to 2004, predominantly from the UK (Vandevelde & Greene 2006). FSE has probably now disappeared, since world-wide strict measures are in place to exclude BSE infected cattle from entering the food chain. There have been no reports from the UK since 2001. TSE has not been reported in the dog (Vandevelde & Greene 2006). The likelihood of importing an infected cat is remote. In addition FSE is not contagious and cats are extremely unlikely to end up in the food chain. Therefore the likelihood that the agent could be imported and transmitted to other animals is negligible.

Coronaviridae can be removed from the initial hazard list. Feline coronaviruses associated with feline infectious peritonitis are present in New Zealand. However the family Coronaviridae contains the contagious exotic disease of pigs, transmissible gastroenteritis that is caused by the transmissible gastroenteritis virus (TGEV). Pigs are the only animals for which TGEV is pathogenic (Paton 2004). The cat and dog have been described as being able to be infected experimentally but without clinical signs (Larson et al 1979). The possibility that any other species than the pig could be a natural source of infection is considered negligible.

Acanthamoeba species are ubiquitous, omnipresent and abundant free-living amoebas found in water, soil, and the atmosphere. Several *Acanthamoeba* spp. are rarely pathogenic to animals and humans who are immunocompromised. No transmission of infection between hosts is known and infections are thought to originate solely from environmental sources (Greene & Howarth 2006). *Acanthamoeba* are therefore not considered to be potential hazards.

The protozoal tick-borne organism *Cytauxzoon felis* was discarded from the preliminary hazard list as it has no zoonotic potential and primarily affects American wild cats such as the panther and bobcat (Rotstein et al 1999). Inadvertent infection in the domestic cat is thought to be through the tick *Dermacentor variabilis* or from fighting with wild cats. The domestic cat is regarded as a dead-end host, with rapid death resulting from infection (Greene et al 2006). The likelihood of importing an infected cat is low and establishment in New Zealand would not be possible without the tick vector and wild cat reservoir hosts.

Bartonella vinsonii spp.var *berkhoffii* is a haemotropic bacterium which generally infects dogs without causing clinical signs. Dogs may be chronically infected. However, it is not known whether infected dogs are able to transmit infection (Birtles 2005). There is a high frequency of co-infection between *B. vinsonii* spp.var *berkhoffii* and other tick-borne pathogens (Birtles 2005; Breitschwerdt & Chomel 2006) and it is likely that transmission is via a tick vector, probably *Rhipicephalus sanguineus* which is not present in New Zealand. For these reasons, this organism is not considered to be a potential hazard.

Taxonomic changes in 2001 resulted in some species of *Ehrlichia* being reclassified into the genera *Anaplasma* or *Neorickettsia* and all were placed in the Family *Anaplasmataceae*, containing four genera: *Ehrlichia*, *Neorickettsia*, *Anaplasma* and *Wolbachia* (CFSPH 2005). The initial hazard list is taxonomically out of date in regards *Anaplasmataceae* and has been reviewed. The risk analysis covers only organisms known to naturally infect cats and/or dogs.

The *Anaplasmataceae* known to naturally infect dogs and/or cats are:

Ehrlichia chaffeensis
Ehrlichia ewingii
Ehrlichia canis
Ehrlichia equi
Anaplasma phagocytophilum
Anaplasma platys
Neorickettsia helminthoeca

Ehrlichia chaffeensis is found only in the United States of America (USA), and is transmitted by the ticks *Amblyomma americanum* and *Dermacentor variabilis*. It causes monocytic ehrlichiosis in humans which has similar symptoms to Rocky Mountain spotted fever. Although it has been shown by serology that dogs are able to be infected naturally (Murphy et al 1998; Neer & Harrus 2006), it is considered an asymptomatic disease of no significance in dogs and cannot establish without the tick vectors. It is therefore not considered to pose a risk in imported dogs.

Ehrlichia ewingii is found only in the southern and southeastern parts of the USA. This regional distribution depends on the geographic range of its tick vector, *Amblyomma americanum*. Both dogs and humans are likely to be incidental hosts, with the major reservoir host being the white-tailed deer (*Odocoileus virginianus*). Clinical disease in dogs is not severe and there have been no reported canine or human deaths attributed to this organism (Greig et al 2006). This organism will be excluded from consideration as it is not found outside *Amblyomma americanum's* geographic range.

Anaplasma phagocytophilum affects humans and a variety of domestic and wild mammals. The organism is transmitted by *Ixodes ricinus* in Europe and *Ixodes scapularis* and *Ixodes pacificus* in the USA. The dog is an incidental host for this organism and it is not known if a carrier status occurs. Subclinical or mild infections are common (Greig & Armstrong 2006; Harrus et al 2005) but disease has been reported on rare occasions from Slovenia and Austria (Tozon et al 2003). This organism is excluded from further consideration as New Zealand does not have the required tick vectors.

E. equi naturally infects humans, dogs and horses. Its geographic distribution includes the USA and the Canadian West Coast. The vector, *Ixodes pacificus* does not occur in New Zealand. Therefore *E. equi* is not considered a potential risk (Greene 2006).

Neorickettsia helminthoeca has a complex life cycle involving a trematode vector, snails, fish and dogs. The snail intermediate host *Oxytrema silicula* inhabits coastal areas of Washington, Oregon and northern California. Areas of trematode infestation are dependent on the distribution of the snail intermediate host. Dogs which eat an infected salmonid fish develop a severe fever and mortality is high if untreated (Gorham & Foreyt 2006). This organism is not considered a potential risk as its complicated life cycle is not sustainable in New Zealand, in the absence of the trematode vector and the snail intermediate host.

The remaining members of Family *Anaplasmataceae* that naturally infect dogs and or cats and may cause chronic infection or significant disease are *Anaplasma platys* and *Ehrlichia canis*. These organisms have been retained on the hazard list.

No other organism in the family *Anaplasmataceae* is considered a potential hazard.

Hepatozoon canis and *Hepatozoon americanum* infect dogs that ingest an infected tick during grooming, not by tick bite (Baneth 2006; MacIntire et al 2006). The tick vectors, *Rhipicephalus sanguineus* and *Amblyomma maculatum* are not present in New Zealand. Therefore these organisms were excluded from further consideration.

As a result of eliminating the above organisms from Table 1, the groups of agents of concern that are considered in this risk analysis are listed below.

5. Agents of Concern

Bacteria
Anthrax (*Bacillus anthracis*)
Borreliosis (*Borrelia* spp.)
Canine brucellosis (*Brucella* spp.)
Leptospirosis (*Leptospira* spp.)
Melioidosis (*Burkholderia pseudomallei*)
Mollicutes (*Mycoplasma, Ureaplasma* and *Acholeplasma* spp.)
Tuberculosis (*Mycobacterium* spp.)
Plague (*Yersinia pestis*)
Salmonellosis (*Salmonella* spp.)
Tularemia (*Franciscella tularensis*)
Q fever (*Coxiella burnetii*)

Blood parasites
Anaplasmosis (*Anaplasma platys*)
Babesiosis (*Babesia* spp.)
Ehrlichiosis (*Ehrlichia canis*)
Filariosis (*Filarial* and *Brugia* nematodes)
Leishmaniosis (*Leishmania* spp.)
Rickettsiosis (*Rickettsial* spp.)
Canine Chagas disease (*Trypanosoma cruzi* and *rangeli*)
Surra (*Trypanosoma evansi*)
Nagana (*Trypanosoma brucei*)

Ectoparasites
Fleas
Leeches
Lice
Mites
Myiasis (fly larvae infestation)
Ticks

Endoparasites
Nematodes and Acanthocephalans
Trematodes
Cestodes

Virus families
Bornaviridae
Bunyaviridae
Flaviviridae
Herpesviridae
Orthomyxoviridae
Paramyxoviridae
Poxviridae
Reoviridae
Rhabdoviridae
Togaviridae

Miscellaneous
Canine transmissible venereal tumour
Exotic strain variations of the major endemic diseases (canine and feline parvovirus, feline herpesvirus and calicivirus, canine distemper and infectious canine hepatitis)
Fungal and Algal infections

A full risk assessment was carried out for each agent of concern listed.

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BACTERIAL DISEASES SECTION

6. Anthrax (*Bacillus anthracis*)

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Anthrax is caused by *Bacillus anthracis*, a gram-positive, aerobic, spore-forming bacillus (NCBI 2007).

6.1.2. OIE List

Listed under 'multiple species diseases'.

6.1.3. New Zealand's status

B. anthracis is listed as an unwanted, notifiable organism (Ministry of Agriculture & Forestry 2008). The last case of anthrax occurred in 1954 (Barry 1954).

6.1.4. Epidemiology

Anthrax is a natural disease of herbivores which are most susceptible, followed by humans and pigs. Carnivores such as the dog and cat are quite resistant to infection (Coker 2004; Langston 2005).

Anthrax is a disease of mammals and has a worldwide distribution. It is sporadically reported from endemic areas of Australia (AHA 2006) and the USA. It is common throughout tropical Africa, Central America, South East Asia and Middle Eastern countries.

Infected, septicaemic animals are likely to have large numbers of bacteria in the blood. Haemorrhage before death and the opening of carcasses cause sporulation and environmental contamination. Anthrax spores remain viable in favourable conditions for at least 50 years.

Animals are exposed to the spores by ingestion, inhalation or inoculation subcutaneously by biting insects. There is no evidence that *B. anthracis* is transmitted by animals before the onset of clinical and pathological signs (OIE 2007).

In dogs and cats, natural infection occurs by ingestion of meat or hides from infected carcasses. The incubation period is 3-7 days with the subacute to chronic form of anthrax

occurring in dogs and cats. This manifests as fever, anorexia, local inflammation, necrosis, and oedema of tissues of the upper gastrointestinal tract. This causes swelling of the head and neck tissues, and if the swelling is severe, death occurs due to occlusion of the airway, particularly in young animals (Orr et al 1978; Creel 1995). In cases where this does not occur, a fatal bacteraemia may develop, although recovery after a few days of illness is not uncommon. An intestinal form with severe acute gastroenteritis is also seen in carnivores (Coker 2004).

Experimental exposure of dogs to aerosolised anthrax spores caused only short term fever and anorexia in some dogs with lesions restricted to the lungs (Moore & Greene 2006).

Semen is not implicated in anthrax transmission in the literature reviewed.

6.1.5. Hazard identification conclusion

Anthrax is a zoonotic, unwanted organism that may cause severe disease in mammals. It is therefore concluded to be a potential hazard in the commodity.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

Anthrax is a natural disease of herbivores which are most susceptible, with dogs and cats being relatively resistant and incidentally infected. Isolated infections in dogs have been reported during major anthrax outbreaks in farm animals. Infections in captive canids and felids have also been reported after they have been fed raw meat from contaminated carcasses (Moore & Greene 2006).

Imported dogs and cats are generally domestic companion animals from urban environments and are unlikely to be present on farms where outbreaks are occurring and thus unlikely to have ingested uncooked meat or hides that are infected with anthrax. Cats and dogs are very rarely infected in this way, but when they are they may become chronically infected. Rare cases are likely to exhibit the obvious clinical signs of oedema of the pharynx and head tissues or severe gastroenteritis between 3-7 days from exposure and this would diminish the likelihood of infected animals from travelling.

Cats and dogs appear to have a natural resistance and cases of anthrax are extremely rare. Cats and dogs are not mentioned in the *Code* chapter on anthrax. Therefore the likelihood of importing an infected animal is assessed to be negligible.

Since anthrax is not regarded as being transmitted venereally, the risk of entry is assessed to be negligible for semen.

6.2.2. Exposure assessment

Any animal imported that is chronically infected would only be a risk to other animals if it died and released anthrax spores into the environment. There is no evidence that anthrax is transmitted by animals before the onset of clinical and pathological signs (Creel 1995; OIE 2007).

In the extremely unlikely event that a chronically infected dog or cat is imported and should die from anthrax, it is highly improbable that its carcass would be allowed to contaminate the environment. The original outbreaks of anthrax in New Zealand around the 1900s resulted from the importation of thousands of tons of unsterilised animal bones that were applied to pastures as fertiliser (Barry 1954). Despite this widespread practice and several outbreaks, *B. anthracis* never became established. An imported case of anthrax in a cat or dog would not contaminate the environment to the same extent.

The risk of exposure is therefore assessed to be negligible.

6.2.3. Risk estimation

The risk of anthrax being imported in a dog or cat and resulting in establishment of the organism is negligible. The risk estimate for semen is also negligible.

As a result the risk estimate for *B. anthracis* is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justified.

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7. Borreliosis (*Borrelia* spp.)

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Bacterium in the Family: Spirochaetaceae, Genus: *Borrelia*.

The genus *Borrelia* contains at least 37 species which are characterized into two groups; those causing relapsing fever, and those causing Lyme borreliosis. Dogs and cats are only rarely affected by the relapsing fever borreliae group, with the clinical significance of such infections not known (Breitschwerdt 1994).

The genospecies of *Borrelia burgdorferi* sensu lato is a bacterial group of at least 10 species that are causative agents of borreliosis in Europe and the USA (Lyme disease). Organisms from this group are the causative agents of Lyme borreliosis (Branton 1998).

7.1.2. OIE List

Not listed.

7.1.3. New Zealand's status

Borrelia burgdorferi is listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

7.1.4. Epidemiology

Within the *Borrelia burgdorferi* sensu lato group, most species are non-pathogenic for humans, dogs and cats. Three species, however, are clinically important as zoonoses in humans and dogs, *B. burgdorferi* sensu stricto, *B. garinii,* and *B. afzelii* (Greene 2006).

Borreliosis is the most frequent tick-transmitted zoonotic disease in the northern hemisphere affecting humans (up to 155 cases per 100,000 individuals) (Wilske 2005). The bacterium has specific tropism for skin, musculoskeletal tissue, joints and the central nervous system depending on the species involved.

Early symptoms of human borreliosis include a red, enlarging rash from the site of tick bite, and flu-like symptoms. Many complications may follow an untreated case, such as meningitis, Bell's palsy (paralysis of part of the face), heart block and painful joints, muscles and bones (AOCD 2004; Bratton 2005).

Borreliae cannot survive as free living organisms. Small mammals and birds are reservoir hosts and infection is transmitted by certain *Ixodes* species of tick. Haematophagous arthropods, including other tick species, fleas, flies and mosquitoes have been found to be infected in nature. These other arthropods are believed to have acquired infection from feeding on infected vertebrates but they have not been capable of transmitting infection to new hosts experimentally (Piesman 1997). Their role, if any, relative to the known tick vectors, is considered insignificant.
In North America, *B. burgdorferi* sensu stricto is the only pathogenic species found in dogs. *Ixodes scapularis*, *I. pacificus*, and *I. neotomae* are the tick vectors (Greene 2006).

In Japan, dogs may be infected with *B. japonica* and *B. garinii*. In Europe, dogs are mainly infected with *B. burgdorferi* sensu stricto and *B. garinii*. *Ixodes ricinus* in Europe and *I. persulcatus* in Eurasia are the primary tick vectors. Distribution of infection corresponds to the habitat of the ticks (Greene 2006).

In an endemic area dogs may become infected but most remain asymptomatic with approximately 5 % developing disease concurrent with a rising titre. Clinical signs include fever and polyarthritis. Cats appear to be asymptomatic and more resistant than dogs (Hovius 2005).

Cats, dogs and humans are incidental hosts of Lyme borreliosis. Infection is associated with outdoor activities that result in exposure to tick vectors. Cats and dogs are not a direct source of infection to people. They may, however, bring infected ticks into the human household (Hovius 2005; Greene 2006).

Investigation of *in utero* transmission by testing the pups of infected dams failed to isolate *B. burgdorferi* and antibodies were not found in any puppy's heart blood. The same study failed to isolate the organism from infected dogs' urine or bladders concluding that urine is an unlikely source of infection. Keeping healthy dogs in direct contact with the infected dogs for up to a year did not lead to infection or seroconversion (Appel 1993). An earlier study (Burgess 1986) suggested that contact transmission may have occurred between two dogs. However, the organism was not isolated from the in contact dog which would have provided the evidence that transmission had occurred.

Blood transfusion could theoretically be a means of transmission, but this has not been reported in humans or animals (Greene 2006).

Semen intended for artificial insemination might be considered a potential source of infection as the organism survives freezing and storage (Kumi-Diaka 1995). However, there have not been any reports of sexual transmission of the disease and attempts to transmit it venereally in rats and hamsters failed (Moody 1991; Woodrum 1999).

Natural transmission of the organism by any means other than by tick inoculation has not been reported. No references could be found indicating that relapsing fever borreliosis is of any clinical significance in dogs or that they are anything but dead-end hosts.

7.1.5. Hazard Identification conclusion

Since *Borrelia burgdorferi* sensu lato species are exotic organisms that may cause severe illness in humans and animals, they are classified as potential hazards. *Borrelia* belonging to the relapsing fever group are not considered to be potential hazards.

Semen is not a potential hazard since there is no evidence of venereal transmission.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

It is likely that asymptomatic infected dogs and cats from endemic areas may be imported. Attached ticks could also be infected with *Borrelia* spp.

Likelihood of entry is therefore assessed to be non-negligible.

7.2.2. Exposure assessment

The only natural way to transmit infection is through the bite of an infected tick. *Borrelia* spp. would not be able to establish in New Zealand because of the absence of the necessary *Ixodes* spp. tick vectors.

There is no evidence that infected cats and dogs can transmit infection to people or other animals. Animals may, however, bring ticks into a household increasing the exposure to humans. Provided animals do not introduce tick vectors, transmission of *Borrelia* spp. would not occur, even if the imported animals were infected.

7.2.3. Risk estimation

In the absence of vectors in New Zealand, and because no other natural transmission is possible, the risk from importing infected dogs and cats is considered negligible. As a result the risk estimate for *Borrelia* spp. is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justified.

However, the risk of importing ticks attached to animals is non-negligible and it is recommended animals undergo an option in the ectoparasites Section 30.3 that would ensure imported animals are tick-free.

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8. Canine Brucellosis (*Brucella* spp.)

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

The *Brucella* genus is comprised of six classical species based on host preference (Greene & Carmichael 2006). *Brucella canis*, *B. abortus* and *B. suis* are on the preliminary hazard list.

8.1.2. OIE List

Bovine (*B. abortus*) and porcine (*B. suis*) brucellosis are listed.

8.1.3. New Zealand's status

B. canis, *B. abortus* and *B. suis* are listed as unwanted, notifiable organisms (Ministry of Agriculture & Forestry 2008).

8.1.4. Epidemiology

B. canis is probably found throughout most of the world and has been reported from the United States, Canada, Central and South America, some European and African countries, China and Asia. Some island nations such as Australia and New Zealand have been able to maintain freedom (The Center for Food Security & Public Health 2007).

B. canis has a natural host range that is limited to species of *Canidae*. It is zoonotic but humans are rarely infected. Cats have been infected experimentally and may develop a transient bacteraemia but are considered highly resistant to natural infection.

The dog is the natural reservoir host for *B. canis*. However, clinical signs are generally restricted to intact dogs and bitches and there are minimal clinical signs associated with infection in neutered animals, despite a persistent bacteraemia. Neutered dogs are rarely febrile although they may have mild generalised lymphadenopathy. Reproductively intact male dogs develop epididymal swelling and testicular atrophy. In bitches, chronic intracellular infection may be re-activated during pregnancy. Intact bitches may show infertility, abortion, or give birth to stillborn or weak pups. Chronic bacteraemia may lead to clinical illness dependent on where embolic organisms localise. This may cause uveitis, meningitis or discospondylitis (Greene & Carmichael 2006).

Dogs are also susceptible to infection with *B. suis*, *B. abortus* and *B. melitensis* from contacting infected tissues and secretions of farm animals. However, dogs are not important in

the spread and maintenance of these infections since they are self-limiting (Metcalf et al 1994; Greene & Carmichael 2006).

Brucella species are transmitted by contact of a sufficient number of organisms with mucous membranes. The numbers of bacteria are highest in semen and uterine/vaginal secretions of reproductively intact dogs (Metcalf et al 1994). Venereal transmission is therefore the usual means of natural spread in dog populations. Organisms are shed in large quantities in the uterine discharges of parturient or aborting bitches. Inhalation of such large quantities of organisms provides another means of spread to dogs and other susceptible hosts such as humans (Greene & Carmichael 2006).

Brucella spp. can be transmitted by artificial insemination and blood transfusions.

8.1.5. Hazard identification conclusion

Since *Brucella* spp. are exotic unwanted organisms that may cause illness in dogs and humans, they are concluded to be potential hazards.

8.2. RISK ASSESSMENT

8.2.1. Entry assessment

Clinically normal but infected dogs could be imported from endemic areas. Dog semen could be infected. Therefore, the likelihood of entry is non-negligible for dogs and dogs' semen.

Only canids appear to be susceptible to *B. canis* and cats are highly resistant (Greene & Carmichael 2006). Cats are very unlikely to be infected with brucella organisms; therefore the likelihood of entry is assessed to be negligible for cats.

8.2.2. Exposure assessment

Venereal transmission is the usual means of natural spread in dog populations. The use of infected animals for breeding purposes, including artificial insemination, or as blood donors could readily result in transmission of the organism. It might be theoretically possible to expose other animals through iatrogenic means such as from careless hygiene during tatooing or microchip implantation.

Organisms are shed in large quantities in the uterine discharges of parturient or aborting bitches. Therefore, inhalation of organisms in dried uterine discharges provides another means of spread to dogs and other susceptible hosts such as humans. Since abortions and normal births would be followed by excretion of the organism in large numbers, transmission by this method or venereally could ultimately lead to establishment of the disease.

Therefore, the likelihood of exposure of naïve New Zealand dogs and humans is assessed to be non-negligible.

8.2.3. Consequence assessment

Infected animals may require desexing and antibiotic treatment. Production efficiency and profitability of affected breeding kennels would be diminished since dogs would become infertile, abort, or give birth to stillborn or weak pups.

Establishment of the disease could result in rare cases of disease in humans.
Dog owners, veterinary and laboratory personnel would be occupationally exposed to infection, particularly when whelping infected dogs.

Effects on the environment would be negligible since there are no wild dog populations.

Since *B. canis* could establish and result in reproductive disease in dogs and rare human infections the consequences are assessed to be non-negligible.

8.2.4. Risk estimation

The likelihood of introduction is non-negligible for dogs and dogs' semen from countries where *B. canis* is present. The likelihood of exposure is non-negligible and the consequences are considered non-negligible should establishment occur.

As a result the risk estimate for *B. canis* is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

Risk management measures are not justified for cats.

8.3. RISK MANAGEMENT

8.3.1. Options

The *Code* makes no recommendations that would prevent *B. canis* being introduced into an importing country with dogs or dogs' semen.

8.3.1.1. Dogs

Quarantine is not an option since infection is generally subclinical and chronic. No vaccine is available and antibiotic therapy is often ineffective because of the persistent intracellular location of *B. canis* infection.

Diagnostic testing to identify carrier dogs is the only feasible option. Serological testing is the most frequently used diagnostic method (Greene & Carmichael 2006). There are at least seven serological tests that may be employed to detect antibodies to *B. canis*. Each has its advantages and disadvantages dependent on sensitivity, specificity and how early antibodies can be detected post-infection. Haemoculture and detection of bacterial DNA using PCR are the other diagnostic testing options that may be considered, either alone or in various combinations with serological testing.

The following testing options presented in ascending order of stringency are available for the effective management of *B. canis* in the commodity.

Option 1.

Within the 7 days prior to shipment, dogs could be serologically tested. The rapid slide agglutination test is highly sensitive but lacks specificity since reactions to other infecting bacterial organisms such as *Pseudomonas* may occur. It could be used as a screening test. A positive result could be followed by either a tube agglutination test or agar gel immunodiffusion test. Any titre of 1:50 or greater in the tube agglutination test could be

followed by a cytoplasmic agar gel immunodiffusion test which is highly specific. A negative result means the dog is probably not infected or is recently infected and not enough time has elapsed for an immune response to be detectable (Greene & Carmichael 2006). A positive result could disqualify the dog from importation.

Option 2.

If more stringent conditions are required, a genus specific PCR blood test could be added to the serology testing or alternatively the screening serology regime could be repeated after 30 days. This is because serologic test results are often negative during the first 3-4 weeks post-infection despite the presence of a bacteraemia within 2 weeks of infection. This option would more reliably detect dogs that have been recently infected compared to Option 1.

Alternatively if less stringent measures are considered appropriate, then haemoculture or PCR without serology could be utilised. However, bacteraemia although sustained, can be absent or intermittent in chronically infected animals and in those treated with antibiotics (Greene & Carmichael 2006).

8.3.1.2. Dogs' semen

The following options may be suitable for the efficient management of the risk of importing *B. canis* in semen.

Option 1.

Donors could be certified as found to be free from clinical signs of canine brucellosis on the day of collection and could be subjected to the same testing as for the dog, with negative results within 2-4 weeks after semen donation.

Option 2.

Since PCR has been shown to be more sensitive than semen culture (Keid et al 2007) a genus specific PCR on the semen would constitute a more stringent test procedure than serology on the donor dog.

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9. Leptospirosis (*Leptospira* spp.)

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Before 1989 in the accepted taxonomic scheme, the species *Leptospira interrogans* contained all pathogenic serovars. More recently over 200 serovars of *Leptospira interrogans* have now been re-classified into at least 23 new serogroups on the basis of antigenetic relatedness (CFSPH 2005). For the purposes of this risk analysis serovars are written as if they were single species e.g. *Leptospira canicola*, *L. bratislava* etc.

9.1.2. OIE list

Listed under the category of "multiple species diseases".

9.1.3. New Zealand's status

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

Other *Leptospira* spp. are listed as "other exotic organisms" (Ministry of Agriculture & Forestry 2008).

9.1.4. Epidemiology

Leptospirosis is not a single disease but a complex of diseases caused by at least 200 different serovars. Many *Leptospira* serovars are adapted to a particular host species which remain asymptomatic carriers for long periods (months to years).

Leptospirosis occurs world-wide and the endemic serovars that occur in each country differ. The most commonly incriminated serovars associated with canine leptospirosis in the USA are *canicola*, *icterohaemorrhagiae*, *grippotyphosa*, *pomona* and *bratislava* that belong to the serogroups: Canicola, Icterohaemorrhagiae, Grippotyphosa, Pomona and Australis respectively (Miller et al 2007). In Australia, *copenhageni* predominates in southern temperate regions and serovars *australis* and *zanoni* in the tropical environment of northern Queensland. Serovar *canicola* does not occur in Australia (Miller et al 2007). Early Australian reports incriminating serovar *icterohaemorrhagiae* probably reflect infection by serovar *copenhageni*. Both organisms belong to the same serogroup (Icterohaemorrhagiae) and cross-react serologically, but *icterohaemorrhagiae* probably does not occur in Australia (Miller et al 2007).

Leptospirosis is particularly prevalent in tropical, humid climates, marshy or wet areas and in regions with an alkaline soil pH. Certain serovars produce acute haemorrhagic, hepatic or renal involvement. Many animals develop more than one of these clinical manifestations, and disease expression can vary among outbreaks and geographic areas with a given serovar.

Species other than the maintenance host may be more resistant to infection with a particular serovar but if infected are more susceptible to disease. *L. pomona* for example, infects dogs in an endemic situation but only causes occasional cases of disease in the dog. However, it may be responsible for causing sporadic cases of disease in other species such as humans (accidental hosts). In maintenance hosts, *Leptospira* may localise in the kidneys and the animals may continue to excrete the organism in their urine and semen for years.

In New Zealand, the prevalence of the disease in humans is relatively high for a temperate climate country and *L. hardjo* accounts for nearly half the cases (Thornely & Baker 2002). The risk of acquiring leptospirosis is strongly associated with occupational or recreational exposures (Truccolo et al 2002). To reduce the risk to humans that are in contact with cattle, vaccination of cattle against the main serovars occurring in New Zealand is widely practiced.

Dog vaccines that contain four main serovars *L. canicola*, *L. icterohaemorrhagiae*, *L. grippotyphosa* and *L. pomona* are available. After a serosurvey in New Zealand, a vaccine against *copenhageni* in dogs was also introduced. Newer vaccines marketed for dogs and other species have prevented renal colonisation and shedding (Greene et al 2006). Vaccines are used to prevent or manage disease in "at risk" dog populations such as those living in northern New Zealand where the prevalence of infection is comparatively high (Hilbink 1992). However, vaccination will not protect dogs that have been infected prior to vaccination.

Leptospirosis is transmitted between animals by direct or indirect contact. Direct transmission occurs through contact with infected urine, venereal and placental transfer, bite wounds or ingestion of infected tissues. Infection can occur through the skin, particularly via abrasions and wounds or macerated skin. Crowding of dogs, as may occur in kennels, enhances direct spread of leptospirosis through contact with infective urine of dogs and/or rats. Indirect transmission occurs through exposure to water, soil, food or bedding contaminated with infected urine.

Dogs are leptospiraemic during the first week of infection (Greene et al 2006). The incubation period is approximately 7 days but varies according to the infecting serovar and host immunity. Increases in serum antibodies clear the bacteria from most organs, but they may persist in the kidney and be shed for months to years in the case of *L. canicola*. Excretion patterns of other serovars have not been determined in dogs (Greene et al 2006). Diseased animals shed more organisms and are more important sources of infection than chronic carriers (Horsh 1989). Leptospirosis is mainly of concern because it is a zoonotic disease that occasionally causes serious disease in humans (Thornley et al 2002).

The disease can be diagnosed by the isolation of the organism, but because this is a slow process (taking up to 26 weeks dependent on serovar) it is more usually diagnosed by serological methods, with a rising titre signifying recent infection and a stable, often low titre indicating resolution or a chronic infection. The microscopic agglutination test is still the most commonly used test and can be used on a variety of animal species without modification. A number of variations of commercial ELISAs that detect recent and active infections in dogs and humans are also available (Greene et al 2006).

Leptospira spp. are sensitive to several antibiotics (Truccolo et al 2002; Murray & Hospenthal 2004). In particular doxycycline and tetracycline can be used in dogs to clear renal carriers (Greene et al 2006).

Although cats seroconvert after exposure to leptospires, they appear to be less susceptible than dogs to both natural and experimental infections. Clinical signs are mild or not noticeable and leptospirosis is considered a rarity in this species (Torten & Marshall 1994; Greene et al 2006). It is unclear, although unlikely that cats can remain long term shedders of leptospires in their urine since they have not been identified as the maintenance host for any serovar (Wilks 2008).

9.1.5. Hazard identification conclusion

Numerous serovars have been implicated in subclinical and clinical leptospiral infections in dogs. Other than the six endemic serovars, they are exotic, zoonotic organisms that may cause severe disease and are therefore concluded to be potential hazards.

Since cases of leptospirosis in cats are rare and surveillance data collected throughout the world point to a very low incidence of the disease in this species (Torten & Marshall 1994; CDC 2005), leptospires are not considered potential hazards in cats.

9.2. RISK ASSESSMENT

9.2.1. Entry assessment

Worldwide, leptospirosis is relatively common in dogs that are unvaccinated and often these dogs are found to be carriers of live pathogenic leptospires. Dogs can be infected by all known serovars, dependent on the prevailing epidemiological situation (Torten & Marshall 1994).

Clinical signs in dogs vary from no noticeable disease to severe icterohaemorrhagic disease. Acutely infected animals or clinically normal chronic carriers may excrete the organisms in their urine and semen. Therefore the likelihood of entry is non-negligible for dogs and dogs' semen.

9.2.2. Exposure assessment

Infected dogs are proportionally much less of a hazard than, say, cattle as a human public health risk in New Zealand. However, carrier dogs shed the organism in their urine and could potentially infect other animals and humans. Dog owners and veterinary staff could be occupationally exposed. Venereal transmission of the organism also occurs. The likelihood of exposure of New Zealand animals and humans to *Leptospira* is therefore assessed to be low but non-negligible.

9.2.3. Consequence assessment

Introduction of new serovars of *Leptospira* are unlikely to have a big impact on the New Zealand dog population. Sporadic cases of disease may occur dependent on the virulence of the infecting serovar with younger dogs' likely to develop more severe clinical signs.

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

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

The likelihood of establishment of new *Leptospira* serovars is low but non-negligible. Establishment of new serovars could cause sporadic cases of disease in humans. Therefore the consequences of establishment are non-negligible.

9.2.4. Risk estimation

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

9.3. RISK MANAGEMENT

9.3.1. Options

At the OIE General Session in May 2009, the International Committee accepted the recommendation of the TAHSC that the empty *Code* chapter on leptospirosis should be deleted from the *Code*. Therefore the *Code* makes no recommendations that would prevent leptospires being introduced into an importing country with dogs or dogs' semen.

The dog is not a well adapted host for most leptospiral infections but shedding is known to occur for months to years in the case of *L. canicola* infection (Greene et al 2006). Because of the occurrence of long term carriers of infection, quarantine is not a suitable option. Vaccination is not an effective means of eliminating leptospires from the kidneys of chronically infected animals.

Testing urine and semen samples by culture or PCR is problematic because isolation of organisms is slow (may take up to 26 weeks dependent on serovar) and selection of primers for PCR that will recognize all serovars has not yet been achieved. Further studies are needed to determine the sensitivity and reliability of genetic detection as a method for diagnosis.

At this present time, serological testing and antibiotic treatment are considered to be practical safeguards that could be applied. The following options presented are in ascending order of likely efficacy of excluding an animal infected with leptospires.

Option 1.

The OIE *Terrestrial Animal Health Standards Commission* considers that international trade does not increase the risks to human or animal health in regards leptospirosis (OIE 2007).

Diseased animals shed more organisms and are more important sources of infection than chronic carriers (Horsh 1989). The diseased dog would not be eligible for travel since they would not be certifiable as clinically healthy and free from infectious diseases.

It could be appropriate to consider leptospirosis in clinically healthy dogs to be of negligible risk to human or animal health and trade without restriction could be permitted.

Option 2.

The microscopic agglutination test (MAT) is the standard serological test for diagnosing leptospirosis. It is a serogroup specific assay and does not identify organisms at the serovar level. Screening with multiple antigens helps identify all serogroups that may be present dependent on serovars known to be present in the exporting country that are able to infect dogs (Torten & Marshall 1994; Greene et al 2006).

Cross-reactivity between serovars confounds serovar-specific serological diagnosis. Nonetheless, negative serology probably provides a strong assurance that the dog is not currently infected and therefore provides a useful pre-export measure. A negative test (50 % agglutination) at a 1:200 titre in the MAT provides the most appropriate interpretation (Greene et al 2006).

Previous infection or vaccination is usually associated with an MAT titre of less than 1:400. Higher vaccination titres are possible but they generally do not persist for longer than 3 months (Greene et al 2006).

Negative serology to a panel of antigens representing a wide range of serogroups could be justified, even though this measure may mean dogs infected with serovars endemic to New Zealand would be excluded on the basis of serology as well as dogs with a recent vaccination history. For this reason, serologically positive dogs could still be eligible for importation after completing a treatment option for eliminating potential carriers of the organism.

Another serological test that could be applied as a pre-export screening test is the macroscopic slide agglutination test that has been developed for human diagnosis. This test is available as a commercial screening kit that uses a broadly reactive leptospiral antigen. It has been used to detect recent or active infections in people and dogs without modification (Levett & Whittington 1998).

Option 3.

The carrier state in dogs can be treated with appropriate antibiotics, which are highly effective in preventing urinary shedding. Aminoglycosides and doxycycline are considered highly effective at clearing the renal carrier state (Greene et al 2006). Imported dogs could be treated with doxycycline or another effective antibiotic before being shipped.

9.3.1.1. Semen

The following options are in ascending order of likely efficacy of excluding *Leptospira* spp. from the commodity.

Option 1.

Donor dogs could be tested serologically with a variety of antigens that occur in the exporting country and not in New Zealand, with negative results.

This option may create practical problems around the confidence in the number and identity of endemic serovars as well as standardisation of the serological test (Wilks 2008).

Option 2.

Donor dogs could be treated with effective antibiotics prior to semen collection.

Option 3.

Semen diluents containing antibiotics that are effective against *Leptospira* spp. could be used in the preparation of the semen.

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10. Melioidosis (*Burkholderia pseudomallei*)

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

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

10.1.2. OIE list

Not listed.

10.1.3. New Zealand's status

Listed as "Other exotic organism, unwanted" (Ministry of Agriculture & Forestry 2008).

10.1.4. Epidemiology

Melioidosis is a disease of humans and animals that occurs predominantly in the tropical and subtropical regions of Asia and northern Australia (Thomas 1981) and in some foci in Africa (Groves & Harrington 1994; Inglis 2004; Inglis et al 2004). The organism is restricted to latitudes within 20° north or south of the equator (O'Brien et al 2006). A human case has occurred in New Zealand in a traveller returning from Fiji (Corkill & Cornere 1987). The aetiological agent is a saprophyte and opportunistic pathogen that occurs in the environment and is widely distributed in water and soil (Sprague & Neubauer 2004). It has been transmitted to animals via oral mucosa, nasal mucosa, ingestion, parental inoculation, and skin scarification (Groves & Harrington 1994). Infection in natural cases is probably by contact with infected water and mud especially through abrasions and wounds. Water was implicated as a possible source of infections in six locations in one study (Inglis et al 2004).

In animals, clinical melioidosis is most commonly seen in sheep, goats and swine. Cases of canine and feline melioidosis have been reported only rarely in the literature. Dogs and cats are considered fairly resistant to disease (Choy 2000) but clinical signs reported include fever, abscess formation that may affect multiple organs, and lymphadenopathy (O'Brien et al 2006). Zoonotic transmission to humans is extremely rare. There have been three possible zoonotic cases in Australia. However it is not certain if the farm animals involved or the environment have been the source of infection (Choy et al 2000).

10.1.5. Hazard identification conclusion

Burkholderia pseudomalleus is an organism that is geographically confined to tropical and subtropical areas of the world. It has not established in temperate climates. It appears to be an opportunistic pathogen and infection is acquired from the environment. The likelihood that a

clinically healthy animal would introduce the organism and that it would establish is considered to be negligible. Therefore it is concluded that *B. pseudomallei* is not a potential hazard in the commodity.

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11. Mollicutes (*Mycoplasma* spp.)

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agents

Class Mollicutes; Genera *Mycoplasma*, *Ureaplasma* and *Acholeplasma*. At least 17 species have been found in dogs and 10 in cats (Greene 2006). Recently organisms previously classified as *Haemobartonella* spp. have been reclassified as *Mycoplasma* spp. In particular *Haemobartonella bovis* has been re-classified as *Mycoplasma haemofelis* (Neimark et al 2002).

11.1.2. OIE list

Not listed.

11.1.3. New Zealand's status

Mycoplasma felis and *Mycoplasma haemofelis* are present (Anderson & Charleston 1967; Tan & Miles 1973; Thompson 1996). The other species are likely to be present but have not been reported. No Mollicutes that infect cats and dogs are listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

11.1.4. Epidemiology

Mycoplasma felis has been associated with conjunctivitis and anaemia in cats and it is regarded as a causal agent of these conditions (Tan & Miles 1973; Thompson 1996; Greene 2006). Since both species are present in New Zealand they are not potential hazards in the commodity.

Other *Mycoplasma* spp. have not been shown to be primary pathogens. They are found in the upper respiratory tract but not usually in the lungs of healthy animals. However, they may be involved as secondary opportunistic organisms in cases of pneumonia. Infections of the urinary tract are also considered to be opportunistic when conditions allow ascending infections from the distal urinary tract where they normally reside. Infections of the reproductive tract are also regarded as opportunistic. *Mycoplasma* spp. are occasionally found in abscesses resulting from wounds (Walker et al 1995). No evidence could be found of the isolation of *Mycoplasma* spp, other than those mentioned above, from dogs in New Zealand. However, since they occur widely in cats and dogs and thousands of dogs and cats are imported annually, it is likely that they would be found if looked for intensively.

11.1.5. Hazard identification conclusion

Since *Mycoplasma* spp. that have not already been described in New Zealand are not primary pathogens and are probably already present here, they are not considered to be potential hazards in the commodity.

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12. Tuberculosis (*Mycobacterium* spp.)

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Order: *Actinomycetales,* family *Mycobacteriaceae*.

The genus *Mycobacterium* contains over 60 recognised species divided into the obligate intracellular pathogens that cause tuberculosis and leprosy, and the ubiquitous soil saprophytes that occasionally cause subcutaneous infections and rarely systemic disease (Irwin et al 2000; Brodin et al 2002).

Four species that may infect cats and dogs were identified on the hazard list: *Mycobacterium bovis*, *M. tuberculosis*, *M. avium* complex, and *M. lepraemurium*.

12.1.2. OIE List

Mycobacterium bovis is listed.

12.1.3. New Zealand's status

Mycobacterium bovis is endemic, and is the subject of an eradication campaign in cattle and deer in a Pest Management Strategy under the Biosecurity Act 1993.

M. tuberculosis, *M. lepraemurium* and *M. avium* complex are endemic, but not subject to control or eradication.

12.1.4. Epidemiology

M. bovis is primarily a bovine pathogen, causing bovine tuberculosis, which can occasionally infect cats and dogs.

Cats and dogs can be infected by ingestion of meat or milk from infected cattle, through bite wounds from infected rodents, (or possums in New Zealand) and by aerosols from infected animals or humans.

Depending on the route of infection, affected animals may present with gastrointestinal or respiratory clinical signs, or with localised disease affecting the skin. Cutaneous infection is seen most commonly in cats (Gunn-Moore 2008). Disease in dogs is rare, and is primarily respiratory (Buick 2006). Subclinical infection is also possible. No reports could be found that actively or subclinically infected cats or dogs can transmit an infective dose of *M. bovis* to other animals or humans.

Cats and dogs are classified as spillover hosts where disease occurs in the species only as long as there is input from an external source. Therefore, the incidence of tuberculosis in dogs and cats is often a reflection of the local prevalence of tuberculosis in maintenance hosts (Buick 2006).

Specific tests for the diagnosis of tuberculosis in dogs and cats via intra-dermal skin tests and specific serum antibody responses have been found to be ineffective. Aspirates or biopsy samples of affected tissues can be stained with Ziehl Neelsen (ZN) stain to confirm mycobacterial presence. The organism is cultured to determine the species involved. However, many ZN positive samples fail to grow in culture, and those that do take approximately 8 weeks (Gunn-Moore 2008). PCR tests are now available, but only for a limited number of mycobacterial species.

Bovine tuberculosis has been eradicated from many developed countries or is the subject of eradication campaigns. The eradication campaign in New Zealand is challenging because the disease is established in brush tailed possums which continually reinfect cattle. Australia is free from bovine tuberculosis (OIE 2008).

12.1.5. Hazard identification conclusion

M. tuberculosis, *M. lepraemurium* and *M. avium* complex are endemic, and not subject to control or eradication. They are, therefore, concluded not to be potential hazards.

Because *M. bovis* is subject to a control and eradication programme it is considered to be a potential hazard.

12.2. RISK ASSESSMENT

12.2.1. Entry assessment

Cats and dogs recently infected or subclinically infected with *M. bovis* could enter New Zealand since clinical signs may not be evident.

Therefore the likelihood of entry is non-negligible for cats and dogs imported from countries that are not bovine tuberculosis free.

12.2.2. Exposure assessment

Transmission of *M. bovis* from infected cats and dogs to humans or other species has been postulated but not formally reported. An infected cat or dog would need to excrete an infective dose via direct contact (aerosol) or by contaminating fomites.

In general, contamination of feed and pasture appears not to be a significant pathway to transmit the organism, as survival times of infective doses of organisms on fomites are relatively short under natural conditions. Also, animals are not commonly exposed to a dose high enough to be infective by the alimentary route. Infection through the oropharyngeal mucous membrane may be significant, although the infective dose for this route is not known (Morris et al 1994).

There are currently no routine tests to detect subclinical or newly infected cats and dogs for *M. bovis*. However, the disease is endemic in cattle, possums, and deer, and infection in cats and dogs is rare. The entry and exposure pathways are sufficiently unfavourable for establishment that the risk of new foci of *M. bovis* infection from importing newly or subclinically infected cats and dogs is considered negligible.

12.2.2.1. Semen

M. bovis has been listed as an organism that is known to be excreted in bull semen. However, the incidence of cattle excreting the organism in semen is assumed to be low as reported cases in the literature are rare. No cases of *M. bovis* being isolated in, or transmitted through canine semen have been described. Therefore, *M. bovis* is not a potential hazard in dog semen that has been donated from a healthy dog.

12.2.3. Risk estimation

Since exposure and establishment are assessed to be negligible, risk is estimated to be negligible and *M. bovis* is not classified as a hazard in the commodities. Risk management measures are therefore not justified.

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13. Plague (*Yersinia pestis*)

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

The gram-negative bacterium *Yersinia pestis* is the causative agent of plague and belongs to the family Enterobacteriaceae.

13.1.2. OIE List

Not listed.

13.1.3. New Zealand's status

The last case of *Yersinia pestis* infection in humans was reported in 1911 during the last pandemic that started in Hong Kong in 1894. There were 21 cases reported in New Zealand between 1900 and 1911. *Y. pestis* is exotic, and listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

13.1.4. Epidemiology

Plague is a zoonotic disease transmitted and maintained by an obligatory flea-rodent-flea life cycle involving chronically bacteraemic rodent hosts and their fleas. Humans and domestic animals are susceptible hosts for *Y. pestis* (Macey 2006).

Plague exists within particular areas of every continent except Australia. These areas are generally associated with semiarid, cooler climates that are adjacent to deserts. The epidemiology in each area is unique, dependent on the rodent reservoir, flea vector, and environmental factors (Watson et al 2001; Macey 2006).

Many animal species are susceptible and 30-40 rodent species that are relatively resistant to disease serve as bacteraemic natural reservoirs (Macey 2006; Pauli et al 2006).

Transmission of *Y. pestis* occurs primarily through flea bites. Following ingestion of infectious blood, *Y. pestis* may be cleared by some flea species (Macey 2006). In others, (particularly rat fleas of the genus *Xenopsylla*) it replicates in the proventriculus 'blocking' the flea so that it starves. During the subsequent increased feeding attempts the bacterium infects the bite wound of the host (Eisen et al 2007). While many species of flea can be infected with *Y. pestis*, fleas of the dog and cat (*Ctenocephalides* spp.) are considered to be poor vectors (Macey 2006).

Infection of cats and dogs usually results from ingestion of infected rodents or lagomorphs.

Infected dogs may develop mild clinical signs. Clinical illness is rare because dogs are highly resistant to infection. They may, however, transport infected fleas into contact with humans (Watson et al 2001).

In cats (as with humans), three clinical forms of the disease have been recognised; bubonic, septicaemic and pneumonic plague.
If infected by a flea bite, the organism enters mononuclear cells where it replicates and is transported to regional lymph nodes. As replication continues the lymph node becomes a "bubo" which undergoes necrosis and abscess formation after 2-6 days, disseminating the organism to the lungs via the lymphatic or blood stream. The bubo itself may burst and discharge thick creamy pus (Macey 2006).

If the organism is ingested or inhaled, which is common in cats from ingesting infected rodents, buboes of the submandibular and sublingual lymph nodes occur. These may burst. Cats develop high temperatures associated with bubonic plague (Gasper et al 1993; Watson et al 2001; Macey 2006). Infection spreads more rapidly in the infected animal following ingestion than by flea bite inoculation, resulting in a shorter incubation period of 1-3 days (Macey 2006).

In the natural environment, mortality in cats is approximately 50 % (Macey 2006).
Death occurs within 4-9 days of ingestion of an infected mouse (Gasper et al 1993).

Less commonly, infection can also occur through contact of the organism with mucous membranes or broken skin or by inhalation of droplets from animals with pneumonic plague (Macey 2006). Spread by respiratory droplets and possibly bites and scratches from cats to humans and other animals could occur. In one survey, 3 % of human plague cases were attributed to contact with infected domestic cats. From 1977-1998 the CDC confirmed 23 human plague infections acquired through inhalation of *Y. pestis* infected droplets expelled from cats with pneumonic plague (Gage et al 2000). Human patients were occupationally exposed veterinarians and their staff or the owners of the sick cats (Watson et al 2001; Macey 2006). Therefore bacteraemic, clinically ill cats may be an uncommon source of human infection (Watson et al 2001).

13.1.5. Hazard identification conclusion

Y. pestis is a zoonosis that causes severe disease in cats and humans. It is listed as an unwanted exotic organism. Therefore, it is concluded to be a potential hazard.
Introduction of infected fleas is also a potential hazard.

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

Dogs are resistant to infection and disease is rare in this species. Since healthy imported dogs have little likelihood of introducing the organism, the likelihood of entry is assessed to be negligible. Similarly, semen from a healthy donor dog has a negligible likelihood of harbouring *Y. pestis*.

Cats are much more susceptible to plague than dogs, generally developing high temperatures with head and neck buboes. Since infection in cats is acute with obvious and dramatic clinical signs (within 1-3 days) including a mortality rate of 50 % (within 4-9 days); it is unlikely that a clinically healthy imported cat would be incubating the disease. Therefore, there is a very low likelihood of entry when a cat is clinically healthy.

Cats and dogs may however pose a risk of introducing *Y. pestis* through the importation of infected fleas. Therefore, the likelihood of entry is considered to be low, but non-negligible for fleas associated with imported animals.

13.2.2. Exposure assessment

The most common mode of plague transmission to humans is through flea bites. If infected fleas are present on the animal, particularly rat flea species, then human or animal exposure might occur.

Establishment might occur if a susceptible reservoir host here (e.g. rodents, rabbits, and their fleas) were exposed to infected fleas in a suitable ecological niche. Elimination of the disease, if it established, would be difficult and expensive.

13.2.3. Consequence assessment

Plague is notifiable to the World Health Organization in accordance with international health regulations and is one of four internationally quarantinable human diseases (Gray et al 2006). Humans that come into contact with an infected animal would require prophylactic antimicrobial therapy. Plague is fatal in 50 % of infected humans if untreated. Secondary spread of plague from person to person, person to animal and animal to person is also possible (Macey 2006). All contacts may require identifying, tracing, treating and possibly quarantine. In the worst case scenario where an animal was not identified as afflicted with plague, it could have contact with multiple humans and animals. The severity of the consequences would depend on the speed of diagnosis. Introduction of *Y. pestis* could result in the establishment of infected populations of rats and other rodents.

There would probably be significant public concern if even a single case of plague occurred in an imported animal or in an in-contact person.

The introduction of plague is likely to cause significant direct and indirect negative consequences. The consequences are therefore assessed to be moderate to potentially high.

13.2.4. Risk estimation

The risk of introducing *Y. pestis* by importing healthy cats and dogs is remote. The risks are considered low for transported fleas that may be associated with the commodity. Therefore, the risk estimate for the introduction of *Y. pestis* is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

13.3. RISK MANAGEMENT

13.3.1. Options

The *Code* makes no recommendations that would prevent *Y. pestis* being introduced into an importing country with the commodities.

Pre-export certification on the day of travel that cats and dogs are clinically healthy, showing no sign of infectious disease is an important available option whereby the animal conforms to the commodity definition.

An option available to ensure that cats and dogs are not infected with plague is to stipulate certification conditions that the animal has been resident continuously for 1 month in a plague free country or zone before departure and undergone the selected risk management option for excluding fleas. If originating from an endemic region then pre-export quarantine may be an option. Quarantine should be long enough to allow the disease to develop if infected and to protect animals from becoming infected.

Dogs and cats in endemic areas may have antibodies that persist for a year or longer following exposure. Testing for antibodies is therefore not available as a risk management option unless two serum samples are taken 10-14 days apart demonstrating a four-fold rise in titre to distinguish active infection from previous exposure. Since *Y. pestis* has a short incubation period, antibodies are not produced early in the course of the disease, therefore serological testing may be insensitive in recently infected animals.

13.3.1.1. Specific options for cats and dogs

The five options presented are in ascending order of likely efficacy of excluding an animal infected with *Y. pestis*, or carrying infected fleas.

Option 1.

Cats and dogs for export could be:
1) subjected to flea control as outlined in Section 26.3; *and*
2) clinically examined and found to be healthy on the day of shipment

Option 2.

Cats and dogs for export could be:
1) certified by a veterinarian as having been treated with an effective acaracide twice at 2 week intervals during the 4 week period prior to export; and
2) been found to be free from fleas and clinically healthy at each treatment.

Option 3.

Cats and dogs for export could be:
1) held in vermin-proof pre-export quarantine facility for 28 days with effective flea control.

Option 4.

Cats and dogs for export could be:
1) held in vermin-proof pre-export quarantine for 28 days with effective flea control; *and*
2) subjected to a serological test for *Y. pestis* with negative results; *and*

3) in the case of a positive result, subjected to a second serological test 10-14 days later. A rising titre could disqualify the animal from entry while a stable or declining titre could indicate that the animal could be imported.

Option 5.

Cats and dogs for export could be:
1) resident continuously for the 28 days prior to shipment in a country or zone that is free from plague; *and*
2) subjected to the flea control option selected for excluding fleas in Section 26.3.

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14. Salmonellosis (*Salmonella* spp.)

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Modern nomenclature (Wray & Davies 2002; Davies 2004b) classifies the genus *Salmonella* into only two species, *Salmonella enterica* and *S bongor*. *Salmonella enterica* is divided into six subspecies with the subspecies *enterica* containing most of the important pathogens of animals and humans. There are approximately 2,500 known serovars in the *Salmonella* genus (Davies 2004a) and when correct conventions are used serovar names do not have species status and should not be italicised but are capitalised. A serovar such as typhimurium is therefore correctly classified as *Salmonella enterica* subspecies *enterica* serovar Typhimurium. In this chapter the abbreviated new convention is used and the organism is referred to as *S*. Typhimurium.

Salmonella serovars are often further classified as to their definitive phage types (DT).

A large number of the known *Salmonella* serovars are potential pathogens of cats and dogs.

14.1.2. OIE list

Salmonella serovars other than a few species adapted serovars are not listed.

14.1.3. New Zealand's status

S. Abortus ovis, *S*. Dublin, *S*. Gallinarum and *S*. Pullorum are notifiable organisms. *S*. Arizonae, *S*. Enteritidis DT 4, *S*. Typhimurium DT 44 and DT 104 and *Salmonella* spp. (other exotic serovars affecting animals) are unwanted exotic organisms (Ministry of Agriculture & Forestry 2008). *S* Enteritidis DT4 has been isolated several times from humans but not from animals (ESR 2007). *S*. Typhimurium DT 104 is an occasional isolate from humans (ESR 2007) and was isolated from three dogs in a household in which the owners suffered from diarrhoea after returning from an overseas visit (Julian 2002), but has not been isolated from any animal since that time.

All *Salmonella* spp. isolated at medical and veterinary laboratories are sent to the National Reference Centre where they are typed and recorded on a register (ESR 2007). In 2006, which was a typical year, there were 1404 isolates from humans comprising 120 serovars or phage types and 1417 isolates from non-human sources comprising 89 serovars or phage types. Only 12 isolations were from dogs and eight of these were *S*. Typhimurium (ESR 2007).

14.1.4. Epidemiology

Salmonella spp. have been commonly isolated from dogs. Twelve investigations in dogs were reviewed (Morse et al 1976) and prevalences of 0-27.6 % were reported. In the largest investigation 8,157 samples were examined with 27.6 % positive. Another worker reports that

the frequency of isolation from faeces was from 1-36 % in dogs and 1-18 % in cats, but that higher numbers of isolations were made when mesenteric lymph nodes were cultured (Greene 2006). A review indicated that at least 53 serotypes had been isolated from dogs up to 1976 (Morse et al 1976). Dogs and cats are more likely to be infected with *Salmonella* spp. when unprocessed (raw) meat or meat products are fed than when commercially manufactured food is used (Joffe & Schlesinger 2002; Finley et al 2007). It has been suggested that prevalence is decreasing due to the more common feeding of commercially produced foods (Greene 2006). Many different serotypes have been transmitted from dogs to humans (Morse et al 1976).

Information on clinical disease caused by *Salmonella* spp. in dogs in New Zealand is sparse indicating that it is a rare disease. No information was found about surveys to detect subclinically infected dogs. Since thousands of dogs and cats have been imported annually without any testing for *Salmonella*, or measures to prevent introduction of the organism, it is assumed that the species in New Zealand will be similar to other countries, particularly those from which dogs and cats are regularly imported. Humans and dogs also share the same salmonellae (MacDiarmid 2008), and about 2.5 million overseas visitors entered New Zealand during 2007 without salmonella testing (Statistics New Zealand 2008). Similarily, other animal species entering New Zealand such as horses and alpacas, do not require testing.

The prevalence of subclinical *Salmonella* infections in dogs and cats is high. Mortality in acute infections is less than 10 % (Greene 2006). Clinical signs are those of gastroenteritis and less commonly bacteraemia and endotoxaemia, pneumonia or other organ infection and abortion or still births (Greene 2006).

Transmission is by the faecal-oral route and both dogs and humans are commonly infected by ingesting contaminated food. No evidence could be found that dogs' semen can transmit salmonellosis. Venereal transmission is not implicated in the epidemiology of the disease in dogs. The existing IHSs for canine semen impose no specific measures for salmonellosis.

Dogs usually excrete the organism in their faeces for 4-6 weeks. Shedding is continuous during the first week of infection but then becomes intermittent. It can be reactivated by stress or recurrent illness (Greene 2006). However, some dogs may be long term sporadic shedders of the organism (Day et al 1963).

Salmonella Typhimurium is endemic in New Zealand in both animals and humans but the definitive phage type DT 104 has only been isolated from humans, between 1-3 times each year from 2004 to 2007.

14.1.5. Hazard identification conclusion

Thousands of *Salmonella* serovars have been identified and many of these have not been isolated in New Zealand. Therefore, the introduction of a new serovar of *Salmonella* by imported dogs is possible and *Salmonella* spp. are considered to be potential hazards.

No evidence of transmission from dogs' semen could be found. Venereal transmission has not been implicated in the epidemiology of the disease in dogs, therefore dog semen is concluded not to be a potential hazard.

14.2. RISK ASSESSMENT

14.2.1. Entry assessment

A wide range of *Salmonella* serovars may occur in subclinically or clinically infected dogs and cats. Since it would be possible for dogs to be carrying serovars that do not occur in New Zealand the likelihood of introducing a new serovar is non-negligible.

14.2.2. Exposure assessment

Imported dogs and cats could transmit new *Salmonella* spp. to humans, other dogs and cats and other companion and production animals that may contact them. Therefore, the likelihood of exposure is non-negligible.

14.2.3. Consequence assessment

The introduction and establishment of new *Salmonella* serovars that might be potentially pathogenic could result in gradual spread of the organisms in New Zealand and the establishment of production limiting diseases of livestock, human disease and infections of wild and feral animals. The emergence of a new serovar, *S*. Brandenberg, demonstrated how a new *Salmonella* serovar was able to spread through the South Island sheep population (Kerslake & Perkins 2006).

Because of its resistance to antibiotics, establishment of *S*. Typhimurium DT 104 in animal populations, and in particular production animals would have the potential to constitute a dangerous source of infection for humans (Davies 2001; Hogue et al 1997).

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

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

Salmonella are sensitive to several antibiotics, but many antibiotic resistant strains have emerged (Jones et al 2002; Wray et al 1991).

Vaccines are not available for immunising dogs and cats against a wide variety of *Salmonella* serovars.

Introduction of infected dogs and cats could lead to the establishment of new *Salmonella* spp. These are likely to be no more harmful than the endemic serovars. However, the multi-antibiotic resistant strains may have the potential to cause human and animal disease that is difficult and expensive to treat. Therefore the consequences are non-negligible.

14.2.4. Risk estimation

Since entry exposure and consequence assessments are all non-negligible, risk is considered to be non-negligible and exotic *Salmonella* serovars are classified as hazards in the commodity. Therefore, risk management measures can be justified.

14.3. RISK MANAGEMENT

14.3.1. Options

The *Code* makes no recommendations that would prevent *Salmonella* serovars being introduced into an importing country with the commodities.

The following relevant points have been considered when drafting options for the effective management of exotic *Salmonella* serovars in the commodity:

- Many *Salmonella* serovars, including many of the common and significant serovars already occur in New Zealand.
- With the exception of the multi-resistant serovars, imported serovars are not likely to be more pathogenic than the endemic serovars.
- Salmonellosis is not a major disease of dogs and has been described only rarely in New Zealand.
- The pathway of introduction of new *Salmonella* serovars by healthy dogs and cats is likely to be insignificant when compared to other pathways such as human travellers.
- Dogs have not been implicated as playing an important role in the transmission of salmonellae to humans, unlike contaminated animal products for human consumption.
- Dogs and cats can be carriers of infection but shedding of organisms is likely to be intermittent and may be reactivated by stress.
- Treatment and vaccination are not useful methods for preventing the introduction of the organism.

The *Code* 2008 does not recommend measures to prevent the introduction of *Salmonella* serovars when trading in cats or dogs.

The following options, given in order of ascending stringency, are available for managing the introduction of exotic *Salmonella* serovars in the commodity:

Option 1.

Since many *Salmonella* serovars occur in New Zealand and cats and dogs are not regarded as important in the epidemiology of salmonellosis, (mainly food contamination from infected production animals) importation of clinically healthy dogs and cats could be allowed without restrictions.

Option 2.

Feeding of raw meat could be prohibited during the 6 weeks immediately prior to shipment.

Option 3.

Faecal samples could be collected twice within the 6 weeks before shipment with an interval of 3 weeks between sample collections. The samples could be cultured and any *Salmonella* isolated could be fully identified to serovar and in the case of *S.* Enteritidis and *S.* Typhimurium to phage type.

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15. Tularemia (*Franciscella tularensis*)

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

Franciscella tularensis is a gram-negative coccobacillus that causes the zoonotic disease tularemia. Isolates are antigenetically similar and divided into two types. Type A, *Franciscella tularensis* subspecies *tularensis*, is prevalent in North America. Type B, *Franciscella tularensis* subspecies *holoarctica*, is found in Asia, Europe and North America (Radostitis et al 2007).

Type A is associated with tick-borne tularemia in rabbits and type B with mosquitoes and water-borne disease in aquatic rodents. Type A is the most pathogenic and the more virulent for humans. Type B rarely causes disease in higher mammals (Radostits et al 2007).

15.1.2. OIE List

Listed under 'multiple species diseases'.

15.1.3. New Zealand's status

Franciscella tularensis is listed as an unwanted, "other exotic organism" (Ministry of Agriculture & Forestry 2008).

15.1.4. Epidemiology

Tularaemia is a zoonotic disease that is able to infect many species of terrestrial and aquatic animals, birds and insects. The natural reservoir hosts are certain rodents and lagomorphs and their associated parasites that include ticks, mosquitoes, fleas and horseflies (Acha & Szyfres 1987).

Tularaemia occurs in the Northern Hemisphere, predominantly between 20° and 70° latitude (Greene & DeBey 2006). It is endemic in some areas of Northern Europe, south-central and western states of the USA, the Russian Federation and Asia. It does not occur naturally in the United Kingdom. The disease occurs as epizootic outbreaks in some countries or sporadically in others. An unusual report in 2002 of a type B subspecies (*novicida*) infecting a human in Australia has been described (Petersen & Schriefer 2005). However, tularemia is not normally found in the Southern Hemisphere or in the tropics (Morner 2004).

In Eurasia, tularemia has a complex epidemiology involving cricetine rodents (such as voles and lemmings), hare and rabbit reservoirs with transmission by the bites from infected ticks and mosquitoes being important sources of infection (Greene & DeBey 2006).

In North America the principal animal reservoirs are the cottontail rabbit (*Sylvilagus* spp.), wild hares and rodents. It is primarily a tick-borne disease (Hopla & Hopla 1994). Infection can pass

transstadially and transovarially in some tick species. In the US, the wood tick *Dermacentor andersoni*, the American dog tick *D. variabilis*, the Pacific Coast tick *D. occidentalis* and the Lone Star tick *Amblyomma americanum* are the primary vectors for dogs and cats. Cats and dogs may also be infected from hunting or ingesting an infected rodent or rabbit.

However, infection in dogs is rare as they are highly resistant, with infection inducing minor clinical signs (Greene & DeBey 2006). The incubation period is approximately two days and disease if it occurs is self-limiting with clinical signs resolving within 5 days (Gustafson & DeBowes, 1996). Cats, in particular younger cats, are more susceptible. Clinical signs in the cat may be absent or include fever and regional or generalised lymphadenopathy with abscessation and occasionally death (Acha & Szyfres 1987; Greene & DeBey 2006).

Transmission from cats to humans may occur in the absence of clinical signs in the cat (Morner 2004). Humans can be infected from cat scratches or bites. However, the overall prevalence of human cases is low in the US (approximately 200 cases per year) and only 1.6 % of these cases were attributed to cats (Greene & DeBey 2006).

Venereal transmission is not implicated in the epidemiology of the disease.

15.1.5. Hazard identification conclusion

Tularaemia is an exotic OIE listed zoonotic disease that is able to infect many species of terrestrial and aquatic animals, birds and insects. It is therefore concluded that *F. tularensis* is a potential hazard in the commodity.

15.2. RISK ASSESSMENT

15.2.1. Entry assessment

Tularemia is a contagious disease principally of wild rabbits and rodents. Cats and dogs are accidental hosts.

Dogs are rarely infected and are resistant to infection with tularaemia organisms (Hopla & Hopla 1994). Although dogs are not thought to be reservoirs or to maintain the organism in an ecosystem they may harbour infected ticks (Markowitz 1985). Therefore entry is assessed to be negligible for tick-free dogs.

The likelihood that dogs' semen would contain *F. tularensis* is assessed to be negligible since healthy donor dogs are very unlikely to be infected and venereal transmission is not implicated in the epidemiology of the disease.

Cats require high doses of *F. tularensis* to become infected and are reported to be only mildly susceptible to infection (Capellan 1993). Cats' occassionally manifest clinical disease but rarely develop a bacteraemia. No evidence could be found that cats are carriers of *F. tularensis* (Acha & Szyfres 1987; Magnarelli 2007), but infected cats can transmit the organism to humans through biting and scratching.

Cats displaying clinical signs would be excluded from travel. Therefore entry is assessed to be low but non-negligible for clinically healthy cats introduced from endemic regions.

15.2.2. Exposure assessment

Although zoonotic potential exists, very few cases of human infection are thought to have been contracted from cats. Their ability to transmit infection to humans is likely due to the presence of the organism in their mouths or claws following hunting or ingestion of infected rodents or rabbits (Capellan 1992). In the unlikely event that an imported cat subsequently infects a human, this would not lead to establishment of the organism. This is because treatment for humans is effective and person to person transmission has not been reported (The Center for Food Security & Public Health 2005).

Tick vectors present in endemic parts of the Northern Hemisphere include the rabbit tick *Haemaphysalis leporispalustis* and *H. otophila*. Therefore a competent vector may be present in New Zealand in the form of *H. longicornis*. As such, the potential for establishment could exist due to tick, lagomorph and rodent reservoirs being present.

However, the likelihood that *H. longicornis* ticks could become infected from feeding on an imported cat is considered remote. This is because cats are unlikely to infect any arthropod since cats are uncommonly infected and infection should it occur is self-limiting and rarely bacteraemic (Acha & Szyfres 1987; Magnarelli 2007).

Therefore exposure and establishment are assessed to be negligible.

15.2.3. Risk estimation

Since the exposure assessment is negligible, the risk estimate for *F. tularensis* is negligible and it is not classified as a hazard in the commodity. Therefore risk management measures are not justifiable.

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16. Q Fever (*Coxiella burnetii*)

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Coxiella burnetii is an obligate intracellular gram-negative bacterium.

16.1.2. OIE List

Listed.

16.1.3. New Zealand's status

Exotic notifiable disease (Ministry of Agriculture & Forestry 2008).

16.1.4. Epidemiology

Q fever (*Coxiella burnetii* infection) occurs worldwide with the exception of New Zealand (Worthington 2001) and the Nordic countries (Lumio 1981; Jensenius 1997).

Coxiella burnetii probably infects all mammalian species, birds and many arthropods. In animals the infections are of minimal economic importance and rarely cause disease, but *C. burnetii* is a zoonotic organism that sometimes causes serious disease in humans. Most human infections are asymptomatic or present as a mild flu-like condition, but acute or chronic infections sometimes occur and some of these result in serious complications such as myocarditis, endocarditis, hepatitis and renal failure. *C. burnetii* causes sporadic abortions in both humans and animals (Maurin 1999; Arricau-Bouvery 2005).

C. burnetii mostly affects cattle, sheep, goats and humans (Rousset 2004). Dogs and cats do not develop the endocarditis and chronic infections that are sometimes observed in humans (Greene 2006).

Wildlife and farm animal species may be the source of infection for dogs and cats. Transmission to the dog and cat occurs from ingestion or inhalation of organisms while feeding on infected body tissues, milk, placentas or carcasses (Greene 2006).

Cattle, sheep and goats are the principal source of infection for humans. However, since domestic animals such as cats and dogs are susceptible to infection they should be considered as possible sources of infection for other animals and humans (Rousset 2004).

The prevalence of infection in dogs having contact with sheep is much higher than in those with no contact (Boni 1998). Feral cats and stray dogs have a much higher seroprevalance than

domestic pets (Greene 2006). Serological studies in Switzerland and Germany have found significant percentages of dogs (13-45 %) and cats (26 %) to be seropositive (Aitken 1987).

Ticks may also play an important role in maintaining and spreading the organism. At least 40 species of ticks from 11 genera can be infected (Maurin 1999).

Inhalation of aerosols from contaminated secretions or tissues from infected animals is an important means of zoonotic spread. The lungs appear to be the main portal to the systemic circulation. In chronically infected people and subclinically infected animals the uterus and mammary glands are the main site of chronic infection. Reactivation of infection occurs during pregnancy, so shedding occurs mainly at parturition. At that time, large numbers of organism enter the placenta, parturient fluids, faeces, urine and milk (Arricau-Bouvery 2005). *C. burnetii* has been found in semen of bulls and mice (Kruszewska 1993; 1997) and venereal transmission has been demonstrated in mice. However, such transmission has not been established as occurring in other species, including humans, and no information on transmission of the agent in semen of cats and dogs has been found.

Infection in cats and dogs is usually asymptomatic and may be chronic with persistent shedding in the faeces and urine. *C. burnetii* has been found in the blood of experimentally infected cats for 1 month and in their urine for 2 months. Bitches can shed *Coxiella* in their milk for 1 month and in their urine for at least 70 days (Greene 2006). Even so, an Austrian study concluded that pet ownership has no effect on seroprevalence in humans who resided with either cats or dogs or both (Skerget 2003).

Reports of cats and dogs transmitting infection to humans are rare and have always been by exposure to aerosols or fomites that are contaminated with parturient or aborted tissues of infected cats and dogs (Langley 1988; Pinsky 1991; Marrie 1998, 1999; Buhariwalla 1996; Nagaoka 1998).

Infected animals generally remain asymptomatic so the incubation period and the interval to the development of antibodies is uncertain. In humans the incubation period is 1-3 weeks and the development of detectable antibodies takes 2-3 weeks after the onset of symptoms (Maurin 1999). It is assumed that infected cats and dogs will develop antibodies within a similar time interval. Cats have shown a similar reactivity to antigens as humans (Greene 2006).

The infection is diagnosed by serological tests or by isolation of the organism (Arricau-Bouvery 2005). The antibody detection ELISA tends to replace the IFA and CF test as the test of choice for veterinary diagnosis because it is convenient for large scale screening in various animal species (Rousset 2004).

16.1.5. Hazard identification conclusion

C. burnetii is an exotic, notifiable and zoonotic organism. Therefore, it is concluded that it is a potential hazard in cats and dogs. Since venereal transmission has not been established as occurring in dogs, and no information on transmission of the agent in semen of dogs could be found, dogs' semen is concluded not to be a potential hazard.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

Dogs and cats are usually asymptomatic whether they are acutely or chronically infected. Chronic infections can only occur in the unspayed female since the organism resides in the mammary tissue and uterus of such animals. Serological studies have found significant percentages of dogs and cats to be seropositive.

Since acute and chronic infections are asymptomatic it is possible that infected animals could be imported. The likelihood of entry is, therefore, assessed to be non-negligible.

16.2.2. Exposure assessment

In chronic infections of the bitch, the organism can be excreted for at least 70 days in urine (Greene 2006). At parturition large numbers of the organism are shed in the placenta, parturient fluids, faeces, urine and milk. Although cats and dogs may be excreting the organism, infections resulting from exposure to faeces, urine and milk have not been described and a study failed to show pet ownership having an effect on seroprevalence in humans (Skerget 2003).

In humans, inhalation is the primary means of infection. Pregnant infected animals pose the highest risk, as the only described method of transmission from cats and dogs to humans (albeight rarely) is through contact with infected birth products such as placentas or aborted foetuses. Other pets, livestock or wild animals could also be exposed to these infectious birth products.

For these reasons, the likelihood of exposure of humans and animals to *C. burnetii* introduced in unspayed female cats and dogs is assessed to be non-negligible.

The likelihood that spayed females or males could infect other animals or humans is negligible since they do not shed the organism in the numbers found in birth products.

It is not known whether the New Zealand cattle tick can become infected but since at least 40 species of ticks can be infected the likelihood that *Haemaphysalis longicornis* could be infected with the organism is assessed to be non-negligible.

16.2.3. Consequence assessment

Establishment of *C. burnetii* would be likely to have a negligible effect on the livestock industries as infected animals are usually asymptomatic. However, there is a low likelihood that the introduction into a naïve population might cause some abortions. The New Zealand cattle tick could also become infected and might play an important role in the organism becoming endemic.

Establishment of the disease would result in sporadic cases of serious disease in humans.

Owners and veterinarians whelping infected imported cats and dogs would be at immediate risk of infection.

If the organism were to become established in livestock, then abattoir workers, wool sorters, tanners, farm workers, shepherds, dairy workers, and veterinary and laboratory personnel could be occupationally exposed to infection.

Virtually all animals including birds and fish can be infected. These infections are likely to be subclinical; therefore, effects on the environment would not be noticeable.

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

16.2.4. Risk estimation

Since the exposure assessment for spayed females and male cats and dogs is assessed to be negligible, risk from such animals is estimated to be negligible.

However, entry, exposure and consequence assessments are considered to be non-negligible for unspayed female cats and dogs, and as a result, *C. burnetii* is classified as a potential hazard in the commodity. Therefore, risk management measures can be justified.

16.3. RISK MANAGEMENT

16.3.1. Options

The *Code* makes no recommendations that would prevent *C. burnetii* being introduced into an importing country with the commodities. Infected cats and dogs would be asymptomatic carriers of infection and quarantine may not prevent the entry of the organism.

There are no treatment regimes described for cats and dogs that resolve chronic infections.

Cattle, sheep and goats are the principle source of infection for humans. Since domestic ruminants are considered the main reservoir, with cats and dogs very rarely reported to be shedders involved in human infections (Rousset 2004), it may be considered unnecessary to impose restrictions on the importation of cats and dogs.

However, serological testing by an ELISA within 10 days of shipment could significantly reduce the likelihood of the organism being introduced. Animals should also be subjected to all measures proposed in the ectoparasites Section 31.3 of the risk analysis to ensure that infected ticks are not introduced.

The two options presented are in ascending order of likely efficacy of excluding an animal infected with *C. burnetii* or carrying infected ticks.

Option 1.

Suitable measures could be implemented to prevent the importation of ticks on the commodity (see Section 31.3).

NB. Option 1 does not provide protection against the importation of *C. burnetii* except for the prevention of the importation of infected tick vectors.

Option 2.

Unspayed females could be tested by an ELISA, with negative results, within 10 days of shipment and be subjected to the measures required to effectively manage the introduction of ticks (Section 31.3).

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BLOOD PARASITES SECTION

17. Anaplasmosis (*Anaplasma platys*)

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agent

Family *Anaplasmataceae*, genus *Anaplasma*, formerly *Ehrlichia platys*.

17.1.2. OIE Listed

Not listed.

17.1.3. New Zealand's status

Ehrlichia spp. are unwanted organisms (Ministry of Agriculture & Forestry 2008).

17.1.4. Epidemiology

Anaplasma platys was first reported in the United States. Subsequently it has been reported in Greece, France, Italy, Taiwan, Israel, Japan (CDC 2000) and more recently Australia (Brown 2001).

A. platys has been shown by sequencing of the 16S rRNA gene to be related to other ehrlichial species. The natural mode of transmission is presumed to be through the bite of an infected tick *Rhipicephalus sanguineus*. *A. platys* has been recently demonstrated to be present in heavily parasitised free-roaming dogs in the Tanami Desert of central Australia (Brown 2001). *R. sanguineus* is the only tick species in the isolated central areas of Australia where infection has been found. In addition, co-infection with *E. canis* which is known to be transmitted by *R. sanguineus* has been reported from several areas of the world. *A. platys* DNA has been identified in *R. sanguineus* ticks removed from dogs in Japan (Shaw 2005).

Evidence from studies of the tick-borne ehrlichias such as *E. canis*, *A. phagocytophilum* and *E. chaffeenis* indicate that they have each co-evolved with, and are transmitted naturally by, a single genus of ticks (Sumption 2004).

No evidence could be found that dogs' semen can transmit *A. platys*. Venereal transmission is not implicated in the epidemiology of the disease.

Intravenous inoculation of a cat with *A. platys* failed to lead to infection. Humans are not considered susceptible (CDC 2000) since there are no reported cases of infection. The dog is the only known host, although other canidae might be susceptible.

Dogs infected naturally with *A. platys* remain asymptomatic unless they have other concurrent diseases (Hibler 1986). Clinical signs for either agent may be potentiated when *A. platys* infection occurs concurrently with *Babesia canis* or *Ehrlichia canis*. The incubation period in experimentally inoculated dogs is 8-15 days. There are minimal clinical signs in experimentally infected dogs; a slight temperature increase may be noted (Hibler 1986). *A. platys* is not considered a significant disease-causing agent (Arraga-Alvarado et al 2003).

In some geographically restricted areas, strains appear more pathogenic than those found elsewhere. Fever, pale mucous membranes and petechial haemmorhage have been reported in both natural and experimentally infected dogs infected with a Greek and Israeli strain of *A. platys* (Shaw 2005). Doxycycline is an effective treatment for *A. platys*.

17.1.5. Hazard identification conclusion

Since *A. platys* is not present in New Zealand and is an unwanted organism it is classified as a potential hazard. Venereal transmission is not implicated in the epidemiology of the disease, therefore *A. platys* is not considered to be a potential hazard in dogs' semen.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

The only known vertebrate host is the domestic dog. It is likely that some dogs imported from endemic areas could be infected with *A. platys*. Dogs may carry the organism without showing clinical signs.

Cats are not susceptible to infection with *A. platys*.

The likelihood of entry is therefore assessed to be non-negligible for dogs, and negligible for cats.

17.2.2. Exposure assessment

The endemic areas of *Anaplasma* spp. found in Australian cattle do not extend to those where *H. longicornis* is present (Heath 2002). *H. longicornis*, the only tick of livestock found in New Zealand, is incapable of transmitting *A. marginale* or *A. centrale* (Connell 1978; Heath 2002) which are closely related genetically (particularly *A. marginale*) to *A. platys*. Therefore *H. longicornis* probably could not transmit *A. platys*.

The close genetic similarity between *A. platys* and *A. phagocytophilum* causes serological cross reactivity. *A. phagocytophilum* DNA was identified in *H. longicornis* from Korea but the report does not confirm whether the tick can transmit the organism (Kim et al 2003). However, it has been suggested that although natural infection of several genera of ticks by single species of *Ehrlichia* occurs, infected species of ticks may not necessarily be competent vectors, and each species of *Ehrlichia* is only transmitted by a single genus of competent ticks (Sumption 2004). Therefore it seems likely that the competent vectors of the organisms are not *Haemaphysalis* spp. The only known tick vectors for various *Ehrlichia* spp. are *Ixodes* and *Rhipicephalus* species and no ticks from these genera occur in New Zealand.

A. platys is a tick-borne pathogen with *Rhipicephalus sanguineus* the likely vector.Without the presence of suitable vector ticks in New Zealand, even if *A. platys* were to be introduced into the country from imported infected dogs, the likelihood of transmission to another dog is considered to be negligible.

17.2.3. Risk estimation

For dogs, exposure is assessed to be negligible; therefore the risk from dogs infected with *A. platys* is estimated to be negligible.

Cats are not susceptible to infection with *A. platys*, therefore the likelihood of entry is negligible, and the risk is estimated to be negligible.

Since the risk estimate for *A. platys* is negligible, it is not classified as a potential hazard in the commodity. Therefore, risk management measures are not justified.

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18. Babesiosis (*Babesia* spp.)

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agents/vectors/geographic distribution

18.1.1.1. Dogs

Babesia annae: *Ixodes hexagonus*, Northwest Spain.
Babesia gibsoni (many strains): *Haemaphysalis longicornis*, *H. bispinosa*, Middle East, southern Asia, Japan, North Africa, USA, Italy, Australia, southern Europe, Brazil.

Babesia canis: has three antigenically distinct subspecies that are transmitted by various tick species:

- *Babesia canis canis*, *Dermacentor reticulatus*, widespread in Europe and foci in Asia.
- *Babesia canis vogeli*, *Rhipicephalus sanguineus*, Europe, Australia, Japan, Brazil, Africa, USA.
- *Babesia canis rossi, Haemaphysalis elliptica*, southern Africa (the most virulent of the subspecies).

Babesia conrade, a new species with unknown tick vectors has recently been described in North Carolina and California respectively.

18.1.1.2. Cats

Babesia felis: vector unknown, South Africa and Sudan.
Babesia canis subsp. *presentii*: vector unknown, Israel
(Irwin 2005; Schoman 2006; Taboada 2006).

18.1.2. OIE List

Cat and dog *Babesia* species are not listed.

18.1.3. New Zealand's status

Babesia spp. are listed as unwanted notifiable organisms
(Ministry of Agriculture & Forestry 2008).

18.1.4. Epidemiology

Babesiosis is a tick-borne disease. The main tick vectors of the various *Babesia* species are listed above (Section 17.1.1). *B. canis* and *B. gibsoni* are the two species that cause canine babesiosis worldwide. These parasites are transmitted transovarially and transstadially and ticks are believed to remain infective for several generations.

Prevalence in endemic regions around the world varies widely, with the highest prevalence rates reported from animal refuges, greyhound kennels and in fighting breeds of dogs. Surveys of some endemic countries have indicated *B. gibsoni* prevalence ranges from 17 % to 55 % (Rajamanickam 1985; Macintire 2002; Miyama 2005).

Although the UK is not considered endemic with babesiosis, *B. canis canis* infection has been increasingly diagnosed there since the introduction of the Pet Travel Scheme. A recent case of *B. canis* has been diagnosed in an untravelled British dog (Holm 2006).

Babesia spp. can also be transmitted by blood transfusions and transplacental transmission has been demonstrated experimentally and is suspected to occur naturally. There is circumstantial evidence that *B. gibsoni* may be transmitted by dog bites, as many infected dogs are fighting breeds such as American pit bull terriers and the Tosa breed in Japan (Irwin 2005; Birkenheuer 2005).

Venereal transmission is not implicated in the epidemiology of babesiosis and viable protozoa are present only in the bloodstream of animals in the active stages of the infection (Radostits 2007).

In endemic areas, *Babesia* species mostly cause disease in young dogs, although dogs of any age can be affected. The incubation period varies from 10-21 days for *B. canis* and 14-28 days for *B. gibsoni* (Schoman 2006). The pre-patent period (time from infection to the appearance of the organism in the bloodstream) was 2 days in a *B. gibsoni* infected dog tested by PCR (Fukumoto 2001). PCR was sensitive enough to detect DNA from 2.5 µl of blood sample with a parasitaemia of 0.000002 % (Fukumoto 2001). PCR is almost certainly able to detect infection much earlier than serology or examination of blood smears by microscopy and is also able to determine species (Birkenheuer 2003).

The severity of infection depends on the species of *Babesia* and the animal's age. Clinical signs vary widely from acute to chronic or subclinical, depending on species. The dominant species in South Africa (*B. canis rossi*) is very virulent whereas *B. canis vogeli* causes mild or inapparent disease with low parasitaemia. *B. canis canis* infection results in an intermediate pathogenicity between that of *rossi* and *vogeli* subspecies. Young dogs or those that have not been previously exposed to infection are more likely to display severe clinical signs.

Clinical signs are a result of red blood cell lysis as the organism parasitises these cells. Pale mucous membranes, tachycardia, weakness, splenomegaly and fever are seen. Icterus and death occurs in approximately 12 % of dogs infected with *B. canis rossi*, but only around 1 % with *B. canis vogeli* infection (Reyers 1998).

B. gibsoni infections may follow a hyperacute, acute, or chronic course. The acute course is most common with fever, lethargy, and haemolytic anaemia. The hyperacute state, in which the animal quickly goes into shock, is rare. Subclinical infections have been reported and infected animals may remain life-long carriers (Jefferies 2003; Schoman 2006) despite conventional treatments.

Babesiosis in cats is less common and not as well researched as in dogs. *B. felis* is recognised as the cause of feline babesiosis in domestic cats in parts of South Africa and the Sudan (Jacobson 2000; Taboada 2006). South Africa appears to be the only country where feline babesiosis is recognised as a clinical entity in domestic cats. It manifests as an asymptomatic low grade disease due in part to the cat's better tolerance of anaemia than the dog (Irwin 2005). A survey carried out in South Africa noted favourable responses to treatment but with recurring chronic infections. The mortality rate was estimated to be around 15 % in cats (Jacobson 2000).

Few drugs have been shown to be able to eliminate *Babesia* parasites. In the case of *B. canis* imidocarb dipropionate as either a single dose at 7.5 mg/kg or 7 mg/kg given twice 14 days apart has been shown to eliminate the *Babesia* infection and eliminates the infectivity of ticks engorging on treated animals for up to 4 weeks after treatment (Penzhorn 1995; Schoman 2006; Taboada 2006).

B. gibsoni is difficult to clear with conventional treatments and dogs usually become chronic carriers. The first treatment shown to be effective against *B. gibsoni* was demonstrated in a small pilot study which used a combination of atovaquone and azithromycin. However, further studies are needed as not all infections were eliminated and atovaquone is difficult to obtain in some countries (Birkenheuer 2004).

An improved ELISA has been developed that can differentiate between *B. gibsoni* and *B. canis* infections on serology alone (Verdida 2004). Similarily, PCR methodology can determine the species involved (Birkenheuer 2003).

Parasitaemia may be below the microscopic detection limit in chronic cases due to the cyclical nature of the organism as it is not always circulating in the blood. The high sensitivity of newly developed PCR techniques allows the detection of low parasitaemia in subclinically infected cases (Ano 2001; Fukumoto 2001) early detection of infection and characterisation of the species and subspecies present (Birkenheuer 2003).

18.1.5. Hazard identification conclusion

Babesia spp. are unwanted notifiable organisms that may cause significant animal illness, and it is concluded that they are potential hazards in the commodity.

Dogs' semen is concluded not to be a potential hazard since venereal transmission is not implicated in the epidemiology of babesiosis and viable protozoa are present only in the bloodstream of animals in the active stages of the infection (Radostits 2007).

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

Untreated animals infected with *Babesia* spp. are likely to be long-term carriers. Imported animals could also be infested with ticks infected with *Babesia* spp. The likelihood of entry is therefore assessed to be non-negligible.

18.2.2. Exposure assessment

Imported animals could become infested with the New Zealand 'cattle tick' *Haemaphysalis longicornis*. This could result in ticks becoming infected.

The endemic cattle tick is known to be a potential vector for *B. gibsoni* and could possibly be a vector for other *Babesia* spp. For example, *B. canis* is known to be transmitted by a tick of the same genus, *Haemaphysalis leachi* and, therefore, *H. longicornis* might also be a suitable vector for *B. canis* as well.

Imported animals could also be infested with infected ticks capable of establishing and transmitting *Babesia* spp. to the resident cat and dog population.

For these reasons, the likelihood of exposure of New Zealand cats, dogs and ticks to *Babesia* spp. is assessed to be non-negligible.

18.2.3. Consequence assessment

Exposure of New Zealand ticks to imported cats and dogs from *Babesia* infected countries could result in the establishment of babesiosis here. Alternatively, infected ticks on imported cats and dogs could infest New Zealand animals. Naïve New Zealand cats and dogs will be fully susceptible to babesiosis. This would lead to sporadic cases, in exposed cats and dogs with consequent morbidity and mortality and the necessity for treatment and tick control.

Babesia spp. of cats and dogs are not known to be able to infect any other species within New Zealand, including humans. The consequences for humans and New Zealand wildlife are therefore assessed to be negligible.

Since introduction of animals infected with *Babesia* spp. could lead to establishment of a debilitating disease, particularly of rural dogs that are more likely to come into contact with ticks, the consequences are assessed to be non-negligible.

18.2.4. Risk estimation

Release, exposure and consequences are all assessed to be non-negligible, therefore the risk estimate is non-negligible and *Babesia* spp. are classified as a hazard in the commodity. Therefore risk management measures can be justified.

18.3. RISK MANAGEMENT

18.3.1. Options

Although babesiosis is a listed disease of cattle, the *Code* makes no recommendations that would prevent *Babesia* spp. being introduced into an importing country with cats and dogs.

Since animals infected with *Babesia* spp. may be asymptomatic life-long carriers, isolation of imported animals in quarantine is not an option. Treatment for many of the *Babesia* species cannot be relied upon to eliminate infections. Therefore testing animals prior to importation is the only feasible option.

Although *B. gibsoni* infection is refractory to treatment, efficacious treatment is available for *B. canis*. Animals imported from countries where *B. canis* is endemic could be subjected to treatment with imidocarb dipropionate to eliminate infection and give post treatment protection from re-infection (Taboada 2006). *B. canis* test-positive dogs could be treated with a single IM dose of 7.5 mg/kg imidocarb dipropionate no more than 28 days and no less than 21 days from departure.

During the pre-export period the animal could be maintained tick-free to prevent infection, with enough time elapsing for infections to become detectable by appropriate diagnostic tests. By ensuring freedom from tick infestation for 30 days prior to export, any recently infected animal will have sufficient time to seroconvert prior to testing. This could be achieved by

having the animal certified by a veterinarian as been treated with an effective acaracide twice at 2 week intervals during the 4 week period prior to export, and been found to be free from ticks and clinically healthy at each treatment.

Using both antibody and antigen tests would maximise sensitivity in identifying both chronic and recent infections.

By utilizing an ELISA or IFA test for the species of *Babesia* that occur in the country of origin, chronically infected animals would be identified as antibody titres remain detectable for prolonged periods despite low parasitaemia. However, as antibody titres may be low in the early stages of infection, some recently infected animals may test negative (Bobade 1989). A group-specific PCR probe sensitive for the genus *Babesia* could be employed to detect these infections.

Whole cell ELISA methods are considered more sensitive than IFAT methods. False positive serological tests for *B. gibsoni* may result from infections with *Toxoplasma gondii* and *Neospora caninum* as well as for *B. canis* especially at lower serum titres (Taboada 2006). PCR assays of infected dogs detected 100 % of infections by testing at 30 day intervals (Birkenheuer 2004).

Therefore, for animals that are serologically positive for *B. gibsoni* but PCR negative could still be truly infected due to the intermittent presence of the organism in blood, or actually false positive due to toxoplasma cross reaction, for example. A second PCR carried out 30 days after the first PCR could be used to determine the animal's true health status.

In the case of countries where both *B. gibsoni* and *B. canis* occur, an antibody ELISA that can differentiate between the two species could be used. This would allow the importation of an animal serologically positive to *B. canis* and negative to *B. gibsoni* so long as it has been treated with imidocarb diproprionate and is PCR negative after treatment. To safeguard against false-positive PCR results from DNA remnants of *B. canis*, treatment for *B. canis* could be administered no more than 28 days, and no less than 21 days prior to travel.

Sensitive screening of *Babesia* spp. could therefore be achieved by serological and PCR tests on a blood sample taken from the animal within 10 days of scheduled departure.

Animals should be subjected to measures proposed in the ectoparasites Section 31.3 of the risk analysis to ensure that ticks are not introduced. If ticks are detected on arrival in New Zealand the animal should be held at a transitional facility and require further testing and treatment for *Babesia* spp. and be treated to eliminate ticks.

The following options, given in ascending order of stringency, are available to effectively manage the risk of importing *Babesia* spp. in the commodity.

Option 1.

Dogs to be imported could be:

1) certified by a veterinarian as having been treated with an effective acaracide twice at 2 week intervals during the 4 week period prior to export, and been found to be free from ticks and clinically healthy at each treatment; *and*
2) show no clinical signs of babesiosis on the day of export; *and*
3) be subjected to measures for effective management of ticks; *and*
4) if ticks are detected on arrival in New Zealand the animal should be directed to a transitional facility and may require further testing and treatment for *Babesia* spp. and should be treated to eliminate ticks.

NB. Option 1. does not provide meaningful protection against the importation of *B. gibsoni* or *B. canis* except for the prevention of the importation of tick vectors.

Option 2.

1) subjected to the measures for tick control in Option 1; *and*

2) subjected to a serological test (IFAT or ELISA) for the *Babesia* spp. that occur in the country of origin and/or a *Babesia* genus or species specific PCR within 10 days of shipment; *and*
   a) test negative dogs could be imported; *and*
   b) *B. canis* test positive dogs that are negative for *B. gibsoni* could be treated with a single IM dose of 7.5 mg/kg imidocarb dipropionate no more than 28 days and no less than 21 days before shipment; *and*
   c) be subjected to the option selected for ticks (Section 31.3) to prevent their importation;
   d) *B. gibsoni* test positive dogs could be disqualified; *and*
   e) serologically positive but PCR negative dogs should be re-tested with the PCR after 30 days.

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19. Ehrlichiosis (*Ehrlichia canis*)

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agent

Family *Anaplasmataceae*, genus *Ehrlichia*, species *canis* (NCBI 2006).

19.1.2. OIE List

Not listed.

19.1.3. New Zealand's status

Ehrlichia spp. are unwanted and exotic organisms (Ministry of Agriculture & Forestry 2008).

19.1.4. Epidemiology

Ehrlichia canis is geographically widespread in tropical and semi-tropical regions of the world, reflecting the distribution of the tick vector *Rhipicephalus sanguineus* (Waner 2000).

E. canis affects only members of the family Canidae. Although a strain of *E. canis* has been isolated from an asymptomatic human in Venezuela, *E. canis* is not considered a zoonotic disease (The Center for Food Security & Public Health 2005).

E. canis is transmitted by the tick *R. sanguineus*, with no reports of natural transmission by any other means. Experimental transmission by *Dermacentor variabilis* has been demonstrated (Johnson 1997) but there is no reference to the New Zealand cattle tick *Haemaphysalis longicornis* being able to transmit infection. Available evidence for the well studied tick-borne ehrlichias such as *E. canis*, *A. phagocytophilum* and *E. chaffeenis* indicates that they have each co-evolved with, and are each transmitted naturally only by, a single genus of ticks (Sumption 2004). An infected blood transfusion may iatrogenically transmit the organism (Shaw 2005).

In dogs, infection with *E. canis* causes a variety of clinical signs that include depression, lethargy, fever, weight loss and, occasionally severe bleeding disorders leading to death (Waner 2000).

After an incubation period of 8-20 days, the acute phase of infection lasts 1- 4 weeks. Few dogs succumb to acute disease and clinical signs usually resolve within 1- 2 weeks without treatment. Death is rare during this phase (Shaw 2005; Neer 2006).

Some dogs that recover clinically from the acute phase remain subclinically infected for months or years. During this subclinical phase dogs may clear the organism, remain infected but asymptomatic, or develop chronic disease.

Clinical signs in the chronic phase vary from mild to severe. However, chronic infections may also be asymptomatic. Dogs can therefore remain asymptomatic carriers for some time. It is not known what percentage of chronically infected dogs develop clinical illness.

Ehrlichial DNA in spleen aspirates taken from four subclinical phase dogs, 34 months after infection, suggests that the spleen is the organ likely to harbour *E. canis* and is the last organ to harbour the organism before elimination. Two of the dogs were PCR positive on blood testing but it is not known if the parasitaemia would have been infective to ticks or to other dogs by blood transfusion. The organism could not be observed by examining the blood microscopically, probably because the numbers were too small (Harrus 1998).

Experimental attempts to transmit *E. canis* with adult *R. sanguineus* ticks that fed to repletion as nymphs on dogs during the subclinical and chronic phases of infection were unsuccessful. Transmission by adult *R. sanguineus* occurs only when nymphs or larvae have fed on dogs in the acute phase of infection (Johnson 1997). An earlier study also found that ticks could become infected during the acute canine infection, but not by engorging on chronically infected dogs (Lewis 1977). It is likely that for any tick to become infected it would have to ingest an infected leukocyte during the acute phase of infection (Neer 2006). The organism is transmitted transstadially but not transovarially. As ticks become infected only by feeding on acutely infected dogs this precludes chronically infected dogs from being the major natural reservoir of the organism (Hibler 1986).

Doxycycline given for 10 days at 5 mg/kg once daily is highly effective at clearing infection from dogs treated during the acute phase. A dose of 10 mg/kg daily for 10 days is effective in dogs with asymptomatic chronic disease (Hibler 1986; Waner 2000; Greig 2006). In the chronic severe stage of disease treatment with antibiotics is unrewarding and the prognosis is poor (Waner 2000).

19.1.5. Hazard identification conclusion

Since *E. canis* is not present in New Zealand and is an unwanted organism that may cause severe disease in dogs, it is concluded to be a potential hazard in the commodity.

Because *E. canis* is transmitted by the tick *R. sanguineus*, with no reports of natural transmission by any other means, dogs' semen is concluded not to be a potential hazard.

19.2. RISK ASSESSMENT

19.2.1. Entry assessment

Dogs may carry this organism in any phase from acute to chronic without showing clinical signs. It is likely that infected dogs from endemic areas could be imported into New Zealand.

The major vertebrate hosts for *E. canis* are members of the family Canidae, e.g. fox, coyote, jackal and domestic dog. Attached *R. sanguineus* ticks may also be infected with *E. canis*.

Cats may be rarely infected naturally by *E. canis* as shown by seropositivity and the presence of *E. canis* DNA. However, naturally occurring disease has not been confirmed.

Likelihood of entry is therefore assessed to be non-negligible for dogs, and negligible for cats.

19.2.2. Exposure assessment

E. canis would not be able to establish in New Zealand in the absence of *R. sanguineus*. The organism is maintained and is naturally transmitted to dogs solely by that species of tick. The only other means by which infection could be transmitted is by blood transfusion. In the unlikely event that an infected imported animal were to be used as a blood donor, the recipient dog may become infected. However, this would not lead to establishment. There has been one reported case of *E. canis* in an asymptomatic human,. Therefore, if transmissible to humans it appears that this is extremely rare and causes no ill effects.

R. sanguineus ticks have been shown to be infected from feeding on dogs in the acute phase of infection only (Lewis 1977; Johnson 1997; Waner 2000). Since rickettsaemias are cyclic and low in mammalian hosts, they are often considered dead-end hosts except in the acute phase of infection. Amplification of *Ehrlichia* probably occurs in the tick.

The exotic tick *Dermacentor variabilis* has been shown to be able to transmit infection experimentally. New Zealand's endemic tick *H. longicornis* may feed on dogs, but is mainly associated with cattle and is not a known vector of *Ehrlichia* spp. nor is it able to transmit the closely related *Anaplasma* spp. (see Anaplasmosis, chapter 16).

The likelihood of *E. canis* establishing is assessed to be negligible since the vector *R. sanguineus* is not present in New Zealand.

Infected *R. sanguineus* ticks on imported animals could transmit infection. Measures should be implemented to ensure that ticks are not introduced on imported cats and dogs.

19.2.3. Risk estimation

For dogs, exposure is assessed to be negligible; therefore the risk from importing dogs infected with *E. canis* is estimated to be negligible.

Cats are rarely infected naturally with *E. canis*. Therefore, likelihood of entry is negligible and the risk is estimated to be negligible.

Since the risk of introducing *E. canis* is regarded as negligible for imported cats and dogs, risk management measures are not justified for *E. canis* itself. However, measures to ensure ticks are not introduced are strongly recommended (see Section 30.3).

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20. Filariosis (*Filaria* and *Brugia* spp.)

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Nematode roundworms belonging to the Order Spirurida, Superfamily Filaroidea: Family: Onchocercidae. There are approximately 200 species of these filarial nematodes. The important exotic filarial species infecting cats and dogs are: *Dirofilaria immitis*, *Dirofilaria repens*, *Brugia malayi* and *Brugia pahangi*.

20.1.2. OIE List

Not listed.

20.1.3. New Zealand's status

Dirofilaria immitis is listed as a notifiable, unwanted organism (Ministry of Agriculture & Forestry 2008).

20.1.4. Epidemiology

20.1.4.1. Heartworm

The most important filarial parasite in the dog and cat is *Dirofilaria immitis*, known commonly as heartworm. Heartworm is widely distributed in Asia, Australia, Europe and the Americas. The prevalence ranges from several percent in cooler areas e.g. 1 % in South Australia (Copland 1992) to virtually 100 % in tropical regions of the Northern Territory of Australia (McSporran 1994). The prevalence is dependent on the density of infected vectors, the presence of water and average daily temperatures.

A survey of 18,000 American veterinary clinics in 2001 identified more than 240,000 dogs and 3000 cats infested with *D. immitis* (McCall 2005).

Heartworm infested dogs were imported into New Zealand on a number of occasions prior to imposition of safeguards in 1994, but the organism has never established. The results from a survey of 880 healthy dogs tested during 1989-1990 from Northland supported New Zealand's claim to be free from *D. immitis* (McKenna 2000).

Worldwide there are over 70 species of intermediate mosquito hosts for *D. immitis*, mostly in the genera *Culex*, *Ades*, *Anopheles* and *Mansonia* (Weinland 1969; Ferasin 2005). Three

potential vector species are found in New Zealand; *Aedes notoscriptus*, *Culex quinquefasciatus* and *Aedes australis*.

The mere simultaneous presence of vectors and infested dogs is not sufficient to enable establishment of the parasite. For instance, *Aedes australis* is restricted to Southland where temperatures would not allow larval development (McSporran 1994; Holder 1999). The successful completion of the life cycle depends on environmental temperatures that allow the development of infective larvae within mosquito hosts.

Adult *D. immitis* are usually found in the caudal pulmonary arteries. The 1st stage larvae (microfilaria) are released into the circulation and ingested by the mosquito. Mosquitoes are obligatory intermediate hosts in which microfilaria develop into the 3rd stage infective larvae. The maturation time is dependent on the environmental temperature. The 3rd stage larvae enter the mammalian host during subsequent blood feeding and over 2-4 months undergo further development and migration to the pulmonary arteries. The young worms then take 2-4 months to mature and can survive for several years as adults. Therefore, the time from infection to development of mature worms is about 7 months.

While maturation in mosquitoes takes 8 days at 30°C (average daily temperature) it takes approximately 1 month at 18°C. Maturation cannot occur below a threshold temperature of 14°C (Ferasin 2005; AHS 2005). It is, therefore, possible that transmission of infective larvae could occur in the summer months in the northern parts of the North Island where the necessary climatic conditions may be met. However, establishment of the parasite would also be dependent on a number of other factors, such as population density of mosquito hosts and infected dogs.

Disease occurs mostly in dogs and less commonly in cats and ferrets. Humans can be infected, however they are incidental hosts and the resulting infestation is non-patent.

Cats are considered to be an aberrant host for *D. immitis* but spillover infections occur in regions where there is a high density of infested dogs and vectors. Infestation occurs at a lower incidence and is generally less severe. Most importantly, microfilaraemia is uncommon (fewer than 20 % of infested cats), and is low or transient when present. Humans and cats are considered dead end hosts, as the parasites rarely undergo final maturation to complete their biological cycle (McSporran 1994; AHS 2005).

Clinical signs are related to damage to the pulmonary arteries and subsequent right-sided heart failure. Many heartworm infestations are clinically inapparent for 2 or more years. During this time circulating microfilaria produced by the mature worms are infective to feeding mosquitoes (Nelson & Couto 1992; Ferasin 2005).

Traditionally, diagnosis has relied on the identification of microfilaria in a concentrated blood sample. Antibody tests are available, but have in general been superseded by antigen ELISA kits which detects specific circulating proteins released by the reproductive tract of the mature female worm (Ferasin 2005). These tests are unable to detect immature female and male-only infestations and they sometimes fail to detect light infestations (1-4 adult worms) (Atkins 2003). Sensitivities approach 98 % in heavy infections but decrease to 35 % in dogs with low worm burdens. Specificity approaches 100 % for all the available kits (Ferasin 2005). As mature worm burdens are rare in the cat, the antigen tests have much lower sensitivities in this species. Combining a microfilaria blood filter test with an antigen ELISA does not necessarily increase sensitivity (McSporran 1994) because occult infestations (no microfilaraemia) occur

in male-only, low worm burden and immature female worm infestations. However, several studies conclude that the ELISA methods are better able to detect cases with a low number of worms than microfilarial concentration tests (Courtney 1993; Martini 1996).

The earliest that heartworm antigen and microfilaria can be detected is about 5 and 6.5 months post-infection respectively. Circulating antigen may precede but sometimes lags behind the appearance of microfilaria by a few weeks. In low worm burdens, or in animals receiving chemoprophylaxis, antigenaemia may be delayed for approximately 9 months post infection. However, microfilaraemias are transient and low in number in these animals (McCall 2005).
Tests for detection of microfilaria may also be influenced by time of sampling, as microfilaraemia may have circadian rhythms with minimal numbers occurring late morning (Rhee 1998; Hayasaki 2003). There is no justification for testing dogs prior to about 7 months of age for microfilaria. Any microfilaria present in dogs younger than 7 months of age will be from transplacental infection and not from patent heartworm (AHS 2005). These microfilaria cannot complete their life cycle unless taken up by the mosquito, undergo development within the mosquito to the infective larval stage and are then subsequently re-inoculated into a dog.

The current treatment for adult worms in dogs is melarsomine, an arsenical drug. Treatment should proceed in several stages to prevent complications of pulmonary embolism, a common sequel to large numbers of worm deaths. The most commonly used heartworm chemoprophylactics are the macrocyclic lactones (ivermectin, milbemycin oxime, moxidectin and selamectin). These drugs possess anthelmintic activity against microfilariae (1st stage larvae), 3rd and 4th stage larvae, and, in some instances, young adult heartworms. Macrocyclic lactones have ability to kill tissue migrating 4th stage larvae up to the 6th week of infection (McCall 2005; Ferasin 2005). The drugs ability to kill larvae when initial infection may have occurred up to 6 weeks earlier is referred to as “reach-back” efficacy. The American Heartworm Society considers that a single dose of a macrocyclic lactone has reach-back efficacy assured for 1 month and remains high for at least the second month (AHS 2005).

Therefore infective larvae up to 2 months post-infection can be eliminated with either ivermectin at 6 µg/kg, milbemycin oxime at 0.5 mg/kg, or moxidectin at 2-4 µg/kg. Ivermectin can be close to 100 % effective up to 3-4 months post-infection (McCall 1996; Ferasin 2005). In some countries a sustained-release injectable moxidectin is available which protects against infestation after exposure for at least 6 months (Lok 2005).

20.1.4.2. Other filarial infestations

Dirofilaria repens, a mosquito-borne zoonotic parasite, infects domestic dogs in tropical and sub-tropical regions which include southern Europe, Africa and Asia. The life cycle is similar to that of *D. immitis* except adults reside in the subcutaneous tissues. Transmission is by *Armigeres subalbatus* and certain species within the genera *Aedes* and *Mansonia*, none of which are present in New Zealand (Dissanaike 1997; Holder 1999; Anayanwu 2000; Gratz 2004). The use of macrocyclic lactones is also effective in preventing infestation (Ferasin 2005) and treatment of pre-existing infestation is the same as that for *D. immitis*.

Lymphatic filariasis (*Brugia* spp.) is a tropical disease caused by the presence of nematodes residing in the lymphatic vessels of humans and animals.

Brugia malayi almost exclusively parasitises humans, but dogs and cats are able to be infested in endemic areas. Transmission is principally by *Mansonia* and *Anopheles* spp. mosquitoes. Dogs and cats are not considered important epidemiologically, with the organism being

maintained in monkeys and humans. The subperiodic form of *B. malayi* (whereby filaraemia is present during the day) has led to transmission by *Mansonia* spp. to several non-human primates and cats, including wild cats (Acha & Szyfres 1987).

Brugia pahangi may parasitise animals, with cats and dogs being able to act as a reservoir of infection for intermediate mosquito hosts (Snowden 1989). Distribution of this organism coincides with that of *B. malayi*. The vectors for *B. pahangi* are *Mansonia* spp. and *Armigeres subalbatus* (Acha & Szyfres 1987), which are not found in New Zealand (Holder 1999).

20.1.5. Hazard identification conclusion

It is concluded that *Dirofilaria immitis* is a potential hazard in the commodity. Other filarial infestations are confined to tropical and sub-tropical regions and no known vectors are present in New Zealand. Therefore, other filarial organisms are not considered to be potential hazards in the commodity.

Venereal transmission has not been implicated in the epidemiology of filarial infestations and dogs' semen is concluded not to be a potential hazard.

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

Infested dogs and cats, particularly those with low worm burdens may show no noticeable clinical signs. The adult worm life expectancy is 5 years in the dog and 2 years in the cat. Therefore it is likely that dogs and cats imported from endemic areas may be infested.

The likelihood of entry of *D. immitis* is therefore considered to be non-negligible.

20.2.2. Exposure assessment

Cats are rarely infested with *D. immitis* and infestations seldom lead to a microfilaraemia. Since they are considered dead end hosts, the likelihood that cats will infect mosquitoes is considered to be negligible.

Although an increase in the movement of dogs has been implicated in the spread of heartworm in Australia and North America (McSporran 1994), temperatures in New Zealand are marginal for larval development.

It is considered that establishment, if it were to occur, would do so where an average temperature of 18° C or above is maintained for 3 to 4 months (McSporran 1994). Imported infected dogs residing in such climates with a suitable density of intermediate host and other dogs could establish the disease. Climatic conditions suitable for larval development occur in the northern parts of the North Island, including the Auckland region.

As suitable hosts, vectors and climatic conditions are present in some areas, transmission and establishment of the organism is theoretically possible. Therefore, the likelihood of exposure of dogs in northern New Zealand is assessed to be non-negligible.

20.2.3. Consequence assessment

Heartworm causes cardiovascular and pulmonary damage in dogs which can result in death. The disease can be prevented and treated. Establishment would place a small financial burden on dog owners in Northland as chemoprophylaxis during the summer months would be required. Specialised working dogs such as police, customs, MAFBNZ dogs, farm dogs and guide dogs would all be placed at risk.

Eradication is feasible unless a reservoir of microfilaraemic domestic and wild canids establishes that is beyond the reach of veterinary treatment.

20.2.3.1. Other consequences

Human pulmonary dirofilariasis is reported to be a rare zoonosis but it may be more common than generally recognised (Theis 2005). In this condition a single nematode that rarely reaches maturity dies and causes small lung infarcts. The lesions are benign, but on thoracic radiography they appear as nodules which may be misdiagnosed as severe disease. This could prompt unnecessary further diagnostic and therapeutic procedures (Miyoshi 2006).

Because significant adverse health consequences potentially affecting companion animals and humans are likely, the consequences of *D. immitis* introduction are assessed as non-negligible.

20.2.4. Risk estimation

As the exposure assessment for cats is assessed to be negligible, risk is estimated to be negligible.

Entry, exposure and consequence assessments are all non-negligible for dogs. The risk is therefore estimated to be non-negligible and risk management measures can be justified.

20.3. RISK MANAGEMENT

20.3.1. Options

A report commissioned by MAF and completed in 1993 led to the imposition of the current heartworm safeguards for dogs in 1994 (McSporran 1994). Safeguards in current IHSs apply to all countries and consist of adult worm antigen testing, microfilarial concentration testing and pre-export treatment for early larval infestation. Since 1994, many advances have been made in diagnostic antigen testing.

Microfilarial concentration testing may be useful for validating a positive antigen serological test, but offers no further information than the antigen test alone. Even in areas where the prevalence of heartworm infestation is high, many (20 %) infected dogs may not be microfilaraemic. The current generation of antigen tests identify most occult (microfilaria negative) infestations consisting of at least one mature female worm and are nearly 100 % specific (AHS 2005). Heartworm antigen can be identified about 6 weeks earlier than the production of microfilaria. For these reasons the microfilarial concentration test has limitations as a screening test for importing dogs.

The earliest that heartworm antigen and microfilaria can be detected is about 5 and 6.5 months post-infection respectively. Circulating antigen may precede but sometimes lags

behind the appearance of microfilaria by a few weeks. In low worm burdens, or in animals receiving chemoprophylaxis, antigenaemia may be delayed for approximately 9 months post infection. However, microfilaraemias are transient and low in number in these animals (McCall 2005).

Tests for detection of microfilaria may also be influenced by time of sampling, as microfilaraemia may have circadian rhythms with minimal numbers occurring late morning (Rhee 1998; Hayasaki 2003). There is no justification for testing dogs prior to about 7 months of age for microfilaria. Any microfilaria present in dogs younger than 7 months of age will be from transplacental infection and not from patent heartworm (AHS 2005).

Dogs with adult heartworm infections are highly likely to be infectious to intermediate hosts. Owners of antigen-positive dogs could have their animal treated so that they can be imported when they become antigen negative.

Antigen testing identifies adult female worms and does not identify intermediate larval stages. For recently infested animals macrocyclic lactones could be utilized as a pre-export treatment to eliminate early larval infections. The reach back ability is up to 4 months in the case of ivermectin, but approximately 2 months in general. The American Heartworm Society considers that a single dose of a macrocyclic lactone has reach-back efficacy assured for 1 month (AHS 2005).

The options available for excluding *D. immitis*, in ascending order of likely efficacy, are:

Option 1.

Dogs could be certified as showing no clinical signs of heartworm on the day of shipment.

Option 2.

Dogs older than 5 months of age (earliest time post-infestation that heartworm antigen can be detected) on the scheduled date of export, could be subjected to
an antigen ELISA with negative results within 1 month of travel; *and*

Within 48 hours of departure all dogs could be treated with:

Either:	ivermectin at 6 mcg/kg
Or;	milbemycin at 0.5 mg/kg
Or;	moxidectin at 2-4 mcg/kg
Or;	injectable sustained release formulation moxidectin at the recommended dose rate
Or,	selamectin at 6 mg/kg

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21. Leishmaniosis (*Leishmania* spp.)

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

Protozoan parasites of the genus *Leishmania* in the family *Trypanosomatidae* (Ross 1969).

The most common agents causing canine leishmaniosis worldwide are *L. infantum* (Old World) and *L. chagasi* (New World). These two species have been found to be identical and these names should be regarded as synonyms (Gradoni 2004).

21.1.2. OIE list

Listed under 'other diseases'.

21.1.3. New Zealand's status

Leishmania spp. are unwanted, notifiable organisms (Ministry of Agriculture and Forestry 2008).

21.1.4. Epidemiology

L. infantum occurs in India, China, parts of Africa, the Middle East and the Mediterranean region (Old World). *L. chagasi* is endemic in parts of Central and South America (New World) (Gradoni 2004). Leishmaniosis occurs in over 100 countries with climates that are warm-temperate through sub-tropical to tropical. Leishmaniosis is most common around the Mediterranean area and in South America. Trypanosomatids require an insect host to complete their life cycles. These zoonotic diseases are naturally transmitted by sandflies. During a blood meal, the sandflies ingest leishmanial bodies, which develop in the insect mid-gut. Large numbers of infectious organisms then pass to the pharynx and salivary glands, from where they are injected into a mammalian host during feeding (Ross 1969).

The genus *Phlebotomus* in the Old World and genus *Lutzomyia* in the New World are the vectors (Gradoni 2004). It has been suggested that the tick *Rhipicephalus sanguineus* may transmit leishmaniosis. A study in Brazil has shown that it is possible for *R. sanguineus* to be infected by *L. infantum* from feeding on infected dogs. More research is required into the role ixodid ticks may have as possible vectors in the epidemiology of leishmaniosis (Coutinho et al 2005).

Wild and domestic dogs are the main reservoir hosts, with *L. infantum* (Old World) and *L. chagasi* (New World) primarily affecting humans, dogs and certain rodents/carnivores. Cats may be incidental hosts, but are rarely infected. If infected they show both systemic and

cutaneous signs of disease (Baneth 2006). Cats were refractory to experimental infection with a Kenyan strain of *L. donovani*. They appear to have a high degree of natural resistance which may have a genetic basis (Mancianti 2004) but this has not been substantiated.

In dogs, *Leishmania* infection usually causes chronic systemic disease. The incubation period varies from 3 months to 7 years. The disease often manifests as skin lesions but it can also cause chronic renal failure and death. The organisms are more resistant to treatment in dogs than humans and infection is rarely completely eliminated and relapses are common. No effective prophylactic treatment or vaccination is available (Baneth 2006).

Dogs act as a significant reservoir host for human infection. There is a low incidence of human *L. infantum* associated with disease in native inhabitants in endemic areas. The likelihood of infection in healthy people is very low.

The most important route of natural transmission for human or canine leishmaniosis is through the bite of infected sandflies. Although people are often bitten by sandflies infected with *Leishmania*, most do not develop the disease. However, among persons who are immunosuppressed (e.g. due to Human Immunodeficiency Virus infections, immunosuppressive treatments for cancer etc.), cases quickly evolve to a full clinical presentation of severe leishmaniasis (WHO 2006).

It is estimated by the World Health Organization that 500,000 new human cases of the potentially fatal visceral form occur worldwide per year, particularly in children and immunosuppressed adults.

There has been a single reported case (1938) of experimental transmission from dog to human by direct contact and a single suspected case of dog to dog transmission following several years of co-habitation (Longstaffe 1986; Harris 1994). Transmission of *L. infantum* has been reported in dogs that received blood transfusions from infected canine donors (Baneth 2006). However, in the USA, where imported *L. infantum* leishmaniosis has apparently become endemic amongst some foxhound kennels, it is suspected that non-vector transmission may be occurring (Duprey et al 2006).

The diagnosis of leishmaniosis in several foxhounds in a kennel in New York (1999) led to the screening of 10,531 foxhounds by the IFA test. This identified infected dogs in 69 kennels in 21 states and two Canadian provinces. Five hundred and seventy pet dogs not associated with foxhounds were all negative (Owens et al 2001).

Suspected transmission in these circumstances includes fighting/biting, reusing needles for injection, blood transfusions, and *in utero* infection. Experimental infection of pregnant bitches with *L. infantum* has shown maternal *in utero* transmission as demonstrated by positive PCR results in a caesarian-delivered pup but not placental tissues at necropsy (Rosypal et al 2005). However, another published paper concluded that *L. chagasi* is not transmitted vertically in dogs (Andrade et al 2002).

A recent experimental study (Silva et al 2009) speculates that *L. chagasi* may be sexually transmitted from naturally infected dogs to susceptible bitches in the absence of biological vectors. However, in that study the dogs used for mating the bitches had advanced signs of visceral leishmaniosis. Although these dogs were PCR positive on semen testing, indicating the presence of *Leishmania* DNA, no amastigotes could be identified in the mated bitches by either histopathology or immunohistochemistry. Therefore, the study did not prove

transmission by this route. Venereal transmission has never been reported in dogs, and there is no compelling evidence to suggest that it is possible (Diniz et al 2005; Silva et al 2008). Although extremely rare cases of veneral transmission have been reported in humans, it is not considered a sexually transmitted disease of humans.

21.1.5. Hazard identification conclusion

Leishmania species are not present in New Zealand and are unwanted notifiable organisms. They are concluded to be potential hazards in the commodity.

Dogs' semen is concluded not to be a potential hazard.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Dogs may be chronically infected with leishmaniosis with no clinical signs. Attached ticks could also be infected with *Leishmania* spp. Therefore, it is likely that infected dogs could be imported from endemic areas. The likelihood of entry is therefore assessed to be non-negligible.

21.2.2. Exposure assessment

Leishmaniosis is generally a tropical/sub-tropical zoonosis and dogs act as a reservoir of the parasite for humans where there is a competent vector. *Leishmania* spp. would not infect humans in New Zealand because of the absence of suitable vectors. Sandflies that occur in New Zealand are black flies (Simuliidae) and not phlebotomine flies.

Establishment of the disease in the UK, where the vectors are also lacking, has not occurred despite importation of 165,000 cats and dogs with no safeguards for *Leishmania* over a 4.5 year period under the newly introduced Pet Travel Scheme (Guitton 2005). In 2005, five cases of leishmaniosis were recorded through the DACTARI scheme[B]. These were all dogs which had either been imported from endemic countries or were born and usually resident within the UK but had visited endemically infected countries (McCormack 2006). The scheme depends on voluntary submission of cases, there is no requirement for diagnostic laboratories to report infection, therefore the number of cases is probably an under estimation. One hundred and thirty one cases of *Leishmania* were diagnosed by PCR and/or serology by the University of Bristol during 2005 and 2006 (Shaw 2007). The DACTARI scheme and University of Bristol results therefore show that leishmaniosis has been introduced in dogs, but despite this and the large number of infected dogs imported, it has failed to become established in the pet dog population.

However, in the USA, imported *L. infantum* leishmaniosis has become endemic amongst some foxhound kennels. It is suspected that non-vector transmission may be occurring (Duprey et al 2006). The means of transmission is not fully understood; dog fighting/biting, the reuse of needles, *in utero* transmission to pups, blood transfusions and travel to where vectors are present have been suggested as possible infective pathways.

[B] Dog and Cat Travel And Risk Information (DACTARI) a voluntary scheme where practitioners may report cases of exotic diseases diagnosed in the UK.

The likelihood that infected dogs could expose local animals to *Leishmania* is therefore assessed as non-negligible.

It has been demonstrated that *Rhipicephalus* spp. ticks can be infected. Therefore ticks attached to imported dogs could introduce the agent. It is likely that these ticks could establish in New Zealand (Heath 1980; Loth 2004). The likelihood that infected ticks introduced on imported dogs could expose local animals to *Leishmania* is also assessed to be non-negligible.

21.2.3. Consequence assessment

The likelihood of people becoming infected in areas without sandfly vectors is remote, even when they live in close contact with infected dogs.

The likelihood of establishment as an arthropod-borne disease is considered to be negligible since the vectors are not present in New Zealand. However, from the USA paradigm it is possible that dogs managed in a similar fashion to infected foxhound kennels may be at an increased risk of infection. Transmission and subsequent endemic disease might develop in such kennels. Clinical manifestations in foxhounds included chronic wasting with severe muscle atrophy, polyarthritis and renal failure. Dogs would require veterinary treatment which would need to be repeated when relapses occurred. The consequences of disease in the greyhound racing industry for example would be likely to have significant welfare and economic effects. Any trade restrictions imposed would cause further economic loss to the industry.
If infected ticks were to establish and prove to be competent vectors, then dogs could become infected from tick exposure. *Leishmania* could become endemic in areas that have tick and dog populations. During the incubation period, and despite treatment of clinical cases, dogs would remain a source of infection to vectors as treatment rarely clears *Leishmania* infection (Baneth 2006).

The consequences of importing dogs infected with *Leishmania* spp. are assessed as non-negligible. Also, the introduction of infected ticks attached to these animals is non-negligible.

21.2.4. Risk estimation

Dogs
In the absence of a suitable vector, the risk of imported infected dogs transmitting the disease to humans is estimated to be negligible. The risk of dog to dog transmission is however estimated to be non-negligible. As a result the risk estimate for *Leishmania* spp. is non-negligible and it is classified as a hazard in the commodity. Therefore, risk management measures can be justified.

Cats
Cats are incidental hosts which are rarely infected and appear to have a high degree of natural resistance. The risk is estimated to be negligible and risk management measures are not justified.

21.3. RISK MANAGEMENT

21.3.1. Options

Although listed under "other diseases" the *Code* makes no recommendations that would prevent *Leishmania* spp. being introduced into an importing country with the commodities.

Diagnosis is covered in the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* and is usually based on serological tests. Three serological methods (indirect fluorescent immunoassay (IFI), enzyme-linked immunosorbent assay (ELISA) and direct agglutination test (DAT)) are commonly employed in the diagnosis of canine leishmaniosis (Gradoni 2004).

Treatment is not reliable in dogs and no effective vaccine is available. Since infection is chronic with an incubation period of up to several years possible, quarantine is not an option.

Pre-arrival serological testing will detect animals that may have subclinical infections. Veterinary examination to ensure animals remain free from signs of disease on the day of testing and on the day of travel could be required.

The options available for excluding leishmaniosis, in ascending order of likely efficacy, are:

Option 1.

Dogs could be certified as showing no clinical signs of leishmaniosis on the day of shipment.

Option 2.

Dogs could:

1) be subjected to a serological test with negative results within 10 days of travel; *and*
2) be subjected to the measures required to effectively manage the introduction of ticks (Section 31.3).

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22. Rickettsiosis (*Rickettsia* spp.)

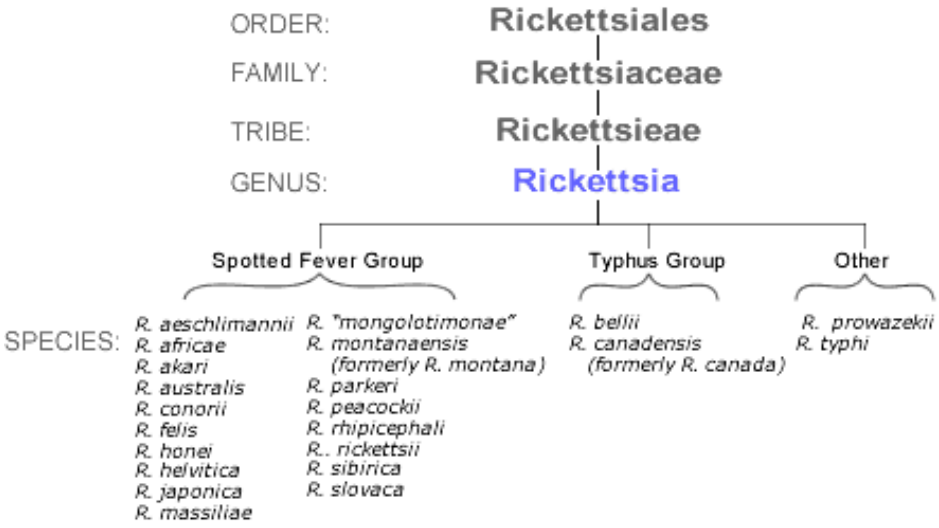
22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

Family Rickettsiaceae, genus *Rickettsia*, spotted fever group, typhus group/scrub typhus group. The taxonomy of rickettsiae has recently undergone significant reorganisation.

Between 1984 and 2005, eleven more species or subspecies of spotted fever group rickettsiae were identified as emerging agents of tick-borne rickettsiosis. Seven of these species had been isolated from ticks and later found to be pathogenic to humans (Parola 2005).

Classification is being continually modified, and experts in the field of rickettsiology frequently disagree over species definitions (Parola & Davoust 2005). This chapter will use the taxonomic grouping as described in Todar's Online Textbook of Bacteriology (Todar 2008).



The related *Coxiella burnetii* and *Ehrlichia* spp. are discussed in separate chapters. The discussion that follows covers the agents listed in the preliminary hazard list.

22.1.2. OIE List

Not listed.

22.1.3. New Zealand's status

Most *Rickettsia* spp. are exotic, unwanted organisms (Ministry of Agriculture & Forestry 2008). *R. felis* and *R. typhi* are endemic (Kelly 2005).

22.1.4. Epidemiology

The rickettsiae are zoonotic organisms that have been found on every continent except Antarctica. They are divided into three groups: spotted fever group (18 species), typhus group (three species) and the other group (two species). The spotted fever group is mostly transmitted by ticks. The remaining groups have mite, flea or louse vectors (Todar 2008).

Rocky Mountain spotted fever, (*R. rickettsii*) Mediterranean spotted fever (*R. conorii*), Queensland tick typhus (*R. australis*), Japanese spotted fever (*R. japonica*) and North Asian tick typhus (*R. sibirica*) are similar diseases caused by agents of the spotted fever group. They are transmitted by their particular tick species in geographically distinct regions around the world.

The most important *Rickettsia* with respect to human disease are *R. rickettsii*, the aetiological agent of Rocky Mountain spotted fever (RMSF) in humans and dogs and *R. conorii* the aetiological agent of Mediterranean spotted fever (Boutonneuse fever).

R. rickettsii is the most severe and most frequently reported rickettsial disease in the USA. The major vectors transmitting *R. rickettsii* infection to people and dogs are the American dog tick (*Dermacentor variabilis*) and Rocky Mountain wood tick (*Dermacentor andersoni*) (Greene & Breitschwerdt 2006).

R. conorii is mainly found in the Eastern Hemisphere (Africa, India, Black Sea countries, and Mediterranean countries) and is transmitted by the tick *Rhipicephalus sanguineus* (Gilot et al 1990). It also causes significant human illness but unlike RMSF infection, Mediterranean spotted fever is asymptomatic in dogs although they do seroconvert. Cats and other domestic animals can also be seropositive but information on the disease in these animals and potential serologic cross-reactivity is limited.

In humans, RMSF is frequently severe enough to require hospitalisation. Up to 20 % of untreated cases and 5 % of treated cases have fatal outcomes. Initial symptoms include rash and myalgia (Chapman et al 2006). Clinical and subclinical illness has been reported in dogs, with clinical signs being similar to those seen in people. Infected dogs recover if mildly affected or if treatment is instituted early. Dogs may die in the acute stage of illness due to organ failure (Greene & Breitschwerdt 2006).

Direct dog to human transmission does not occur. Dogs do not develop a rickettsiaemia of a magnitude or duration capable of infecting large numbers of ticks (Sexton et al 1994; Greene & Breitschwerdt 2006). It has been shown that dogs are rickettsiaemic for up to 10 days following experimental inoculation with *R. rickettsia*, but no ticks could be infected by

feeding on these dogs. However, three of 348 ticks (0.9 %) were infected after feeding on dogs which were infected naturally by tick bite (Norment & Burgdorfer 1984).

Humans and dogs are considered accidental hosts of spotted fever group rickettsia. Small mammal reservoirs which develop a sufficient rickettsiaemia to infect ticks maintain the cycle in nature. Rodents, voles, squirrels, chipmunks and larger mammals, including raccoons and opossums (*Didelphis* sp.), are sources of infection for ticks (Greene & Breitschwerdt 2006).

Dogs did not develop detectable rickettsaemia, fever or other observable clinical signs when infected by tick bite or inoculation with *R. montana* or *R. rhipicephali*, also members of the spotted fever group (Norment 1984).

R. akari causes rickettsial pox in humans. It is rarely diagnosed in humans in the urban USA. The agent is maintained in a mite/mouse cycle with humans and dogs serving as accidental hosts. Infection is from the bite of a rodent mite, *Liponyssoides* spp. It manifests in humans as a mild self-limiting skin rash. Dogs can be naturally infected and seroconvert. However, infection is of no consequence (Comer et al 2001). The disease is rare and of negligible significance and therefore is not considered further.

In general, spotted fever group rickettsiae are maintained in natural cycles between ticks and small wild mammals. Ticks are the natural hosts serving as both reservoirs and vectors. Dogs, cats and humans are accidental hosts and are not involved in the natural transmission cycle.

The typhus and scrub typhus groups make up the remaining rickettsias. This group has flea, louse or mite vectors. *Orientia tsutsugamushi*, the causative agent of scrub typhus in humans, is transmitted by the bite of *Leptotrombidium* mite larvae. This mite is both vector and maintenance host. Geographically specific foci of scrub typhus are determined by the distribution of the vector and rodents of the family Muridae (rats and mice) which are common hosts for trombiculid mites.

Scrub typhus is reported only in the Asia-Pacific region. The mite vector is dependent on temperature and humidity for survival. Antibody to the rickettsia was discovered in US Army tracker dogs in Vietnam. No signs of clinical disease were noted in the seropositive dogs (Alexander et al 1972). Dogs experimentally infected with *O. tsutsugamushi* did not show clinical illness (Greene & Breitschwerdt 2006).

No *Rickettsia* spp. in the typhus/scrub typhus group cause disease in dogs and cats and no evidence could be found that cats and dogs can act as reservoirs of infection for arthropod vectors.

Venereal transmission has not been implicated as occurring in dogs or humans for rickettsial infections.

22.1.5. Hazard identification conclusion

Exotic *Rickettsia* spp. are unwanted organisms, and it is concluded that they are potential hazards in dogs and cats.

Venereal transmission has not been implicated in the epidemiology of the disease; dogs' semen is concluded not to be a potential hazard.

22.2. RISK ASSESSMENT

22.2.1. Entry assessment

Apart from *R. rickettsia*, rickettsial infections in dogs and cats do not cause clinical signs and therefore go unnoticed (Comer et al 2001). Dogs and cats are not normally part of the life cycle of *Rickettsia* spp. It is likely that animals that have been infected could be imported. However, periods of rickettsiaemia are short and no long term carrier state has been described. The likelihood of entry is therefore low but non-negligible.

22.2.2. Exposure assessment

Dogs and cats infected with rickettsia are not a direct source of infection for other animals and remain rickettsiaemic for short periods only. The disease agents are only transmitted by vectors such as ticks, mites, lice and fleas. Therefore, importation of animals that are not infested with arthropod vectors would not transmit the organism to any other animal including humans. The likelihood of exposure is therefore negligible for ectoparasite-free animals.

22.2.3. Risk estimation

As the exposure assessment is negligible, the risk is assessed as negligible for imported cats and dogs that are not externally parasitised. As a result the risk estimate for rickettsial organisms is negligible and they are not classified as a hazard in the commodity. Therefore, risk management measures are not justified.

However, measures to ensure that ectoparasites are not introduced on dogs and cats entering New Zealand are justified and the options described in the relevant ectoparasite sections should be implemented.

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23. Canine Chagas Disease (*Trypanosoma cruzi* and *rangeli*)

23.1. HAZARD IDENTIFICATION

23.1.1. Aetiological agent

Protozoan Family Trypanosomataceae, genus: *Trypanosoma*, species *cruzi* and *rangeli*.

23.1.2. OIE List

Not listed.

23.1.3. New Zealand's status

Trypanosoma spp. are unwanted notifiable organisms
(Ministry of Agriculture and Forestry 2008).

23.1.4. Epidemiology

Trypanosoma cruzi, the causative agent of Chagas disease in humans, is found only in the Americas, being widespread in South and Central America. Trypanosomes are maintained in a wide variety of wild and domestic animals and infection in humans is severe and difficult to treat (Bradley et al 2000).

T. cruzi is transmitted by the cutaneous inoculation of faeces from infected *Triatoma* insects that are obligate blood feeders known colloquially as 'kissing bugs' or 'Mexican bed-bugs'. The vector insect becomes infected by ingesting circulating trypomastigotes within the blood meal.

The triatomid species that are important in human infections feed on people and domestic reservoir species such as dogs and cats, defecating soon after taking their blood meal (Barr et al 1995). The organism transforms to the infective form in the vector's hindgut and is passed in the faeces. The victim then scratches or rubs the infective faeces into the bite wound or mucous membranes (Barr 2006).

Domestic dogs and cats are considered important reservoir hosts for the parasite (Castanera 1998).

Rare sources of infection include blood transfusions and there have been several cases of transmission of *T. cruzi* to laboratory workers by accidental inoculation (Herwaldt 2001).

T. cruzi has been reported to cause disease in dogs but not in cats, although they may be asymptomatically infected. Acute disease occurs mainly in dogs under 1 year of age. Clinical signs are associated with right-sided heart failure or neurological signs. Survivors of the acute

myocarditis become asymptomatic and generally, by 4 weeks post-infection, dogs have an undetectable parasitaemia (Barr 2006). They then remain asymptomatic for months or years. During this time, however, myocardial degeneration continues and dogs eventually die of heart failure.

T. rangeli infects humans and domestic animals in Central and South America. It has an overlapping distribution and has similar triatome vectors. It is non-pathogenic in vertebrate hosts but infected animals develop antibodies that cross react with *T. cruzi* antibodies and can lead to false positive reactions that interfere with diagnosis of *T. cruzi* infections (Grisard 1999).

23.1.5. Hazard identification conclusion

Since *T. cruzi* is not present in New Zealand and is a notifiable unwanted organism, it is concluded to be potential hazard. *T. rangeli* is not a pathogen and is therefore, not considered to be a potential hazard in the commodity.

23.2. RISK ASSESSMENT

23.2.1. Entry assessment

Dogs and cats may be chronically infected without showing clinical signs.

Likelihood of entry is therefore assessed as non-negligible.

23.2.2. Exposure assessment

Dogs cohabitating with people in endemic areas may serve as reservoir hosts for the *Triatoma* species that are the disease vectors. However, the haematophagous insects necessary for organism development and transmission do not occur in New Zealand.

As establishment and natural transmission of the organism is not possible without the haematophagous triatome vectors, the risk of exposure is assessed to be negligible.

23.2.3. Risk estimation

Since exposure assessment is assessed as negligible, the risk from cats and dogs infected with *T. cruzi* is estimated to be negligible.

Since the risk from *Trypanosoma cruzi* has been assessed as negligible for imported dogs and cats, risk management measures are not justified.

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24. Surra (*Trypanosoma evansi*)

24.1. HAZARD IDENTIFICATION

24.1.1. Aetiological agent

Protozoan in the family *Trypanosomataceae*, genus *Trypanosoma*, species *evansi*.

Trypanosoma evansi is closely related to and difficult to distinguish morphologically from the tsetse-transmitted African trypanosomes. It is thought to have evolved from *T. brucei* (Queiroz 2000).

24.1.2. OIE list

Listed under the category of 'equine diseases'.

24.1.3. New Zealand's status

Trypanosoma spp. are listed as unwanted, notifiable organisms (Ministry of Agriculture and Forestry 2008).

24.1.4. Epidemiology

Trypanosoma evansi is the most widely distributed of the pathogenic animal trypanosomes. It is present in many parts of the tropics, including Central and South America, India, the Philippines, Indonesia, Mauritius, North Africa and China (Connor 2004; Luckins 2004).

Humans are not susceptible to infection with *T. evansi*. Infection is often rapidly fatal in camels, buffaloes, horses, cattle, llamas and dogs. Mild and subclinical infections can also occur in these species (Luckins 2004).

Surveys in India and Brazil found the prevalence in dogs was 4.7 % and 23 % respectively (Singh 1993; Herrera et al 2004).

The organism has a direct life cycle and, unlike tsetse-transmitted trypanosomes, there is no biological cycle within the insect vector. *T. evansi* is transmitted mechanically by haematophagous insects, primarily *Tabanus* (main vector) and *Stomoxys* species (Savani 2005). *Stomoxys calcitrans*, the most common species of this genus, is found worldwide including New Zealand, and can transmit *T. evansi* mechanically (Mihok 1995; Sumba 1998; Queiroz 2001).

Disease in dogs is characterized by pyrexia associated with parasitaemia, together with progressive anaemia (main outcome of infection), loss of condition and dullness. Oedema of

the lower parts of the body, urticarial plaques and petechial haemorrhages of the serous membranes are often observed. Recurrent episodes of fever coinciding with parasitaemia occur during the course of the disease which progresses to death. An experimental study involving 50 dogs found the incubation period to be 1.7 to 4 days with a survival time of 14-41 days (Shien 1976). A dog imported to the Netherlands from Nepal developed clinical signs three weeks after importation (Hellebrekers 1982).

Another study determined the interval between parasitaemic episodes to be 3-11 days with the incubation period 3-7 days. However, in this study all dogs survived, manifesting a chronic form of disease lasting at least 70 days post-inoculation (Arora 1995).

The parasite is known to be capable of localising extravascularly in tissues including the central nervous system. In dogs this may cause nervous signs that resemble rabies. Therefore, it may be possible for the organism to also be present in dogs' semen.

Experimental infection of cats resulted in severe disease (Wongyounoi 1990) with cyclical parasitaemia every 14-15 days coinciding with clinical signs. Young cats did not survive the first peak of parasitaemia (Choudhury & Misra 1972). The incidence and course of natural infection in cats is not known.

24.1.5. Hazard identification conclusion

Since *T. evansi* is an unwanted notifiable organism which may cause significant animal illness, it is concluded to be a potential hazard.

24.2. RISK ASSESSMENT

24.2.1. Entry assessment

Although dogs and cats are highly susceptible and show obvious clinical signs, often resulting in death, subclinical and chronic infections also occur.

The likelihood of entry is therefore assessed to be non-negligible.

Since the organism may localise extravascularly, it may be theoretically possible for *T. evansi* to be present in dogs semen. However, a clinically healthy donor dog is unlikely to be infected and there are no reports of spread by this means.

The likelihood of entry in dogs' semen is therefore assessed to be negligible.

24.2.2. Exposure assessment

Surra is a tropical disease and the principal vectors are *Tabanus* spp. flies which are not present in New Zealand. The hosts usually affected are buffalo, cattle, horses and camels in tropical regions of the world. The competent vector *Stomoxys calcitrans* is, however, typically found in greater numbers in warmer parts of New Zealand. North of Auckland would be the most likely area for establishment to occur initially. The feeding hosts for *S. calcitrans* are cattle, horses, sheep, dogs and humans (Tenquist 2001).

Stomoxys flies are most common around sites where composting of organic material such as dairy farm silage, horse manure and straw occurs. The flies probably disperse only as far as required to obtain blood meals (Todd 1964). Horses have a high seroprevalence in endemic areas which suggests they are preferred hosts for biting flies and are more exposed to *T. evansi* than other species (Herrera et al 2004). Horses are highly susceptible to infection and an infected dog or cat in or around stables could lead to disease transmission to susceptible horses via *S. calcitrans*.

Since there would be many susceptible hosts, dispersion of the organism by *S. calcitrans* could be widespread and result in surra becoming endemic. However, *S. calcitrans* is widely distributed around the world in countries where surra does not occur. This indicates that it is probably unlikely to establish in New Zealand as it has not done so in any other temperate climate despite *S. calcitrans* being present (*Tabanus* spp. are the main vectors).

Therefore the likelihood of transmission and establishment is assessed as very low but non-negligible.

24.2.3. Consequence assessment

T. evansi has a wide host range including horses, cattle, llamas, dogs, cats, sheep, goats, pigs and deer. Infection results in severe disease and death. Direct consequences would result from production losses, mortalities, and costs of treating animals and controlling the vector.

The organism does not infect humans but wild animals such as deer and goats may become infected if they are in close proximity and bitten by infected *S. calcitrans*.

Significant adverse consequences potentially affecting horses, cattle, deer and companion animals are likely should *T. evansi* be introduced. Therefore the consequence assessment of importing such animals is assessed to be non-negligible.

24.2.4. Risk estimation

Since entry, exposure and consequence assessments are assessed to be non-negligible, risk is estimated to be non-negligible. *T. evansi* is therefore classified as a hazard in the commodity and risk management measures can be justified.

24.3. RISK MANAGEMENT

24.3.1. Options

The *Code* makes no recommendations that would prevent *T. evansi* being introduced into an importing country with the commodities. However *T. evansi* is covered in the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*. Diagnosis of surra is usually based on the demonstration of the parasites in blood, supplemented by serological tests.

Surra is a tropical disease principally vectored by *Tabanus* spp. flies. The organism is unlikely to establish in New Zealand because the main vectors are not present, restrictions on importation of cats and dogs may therefore not be necessary. *T. evansi* has never spread to temperate climate countries.

Infection is chronic therefore quarantine is not an option. Treatment is not an option as chemotherapeutic agents have been experimental in nature and no reliable treatment for cats and dogs is available (Galhotra 1986; Greene & Matete 2006).

Pre-arrival serological testing, direct blood examination and veterinary examinations will detect animals that may be incubating disease or have mild or subclinical infections. Animals should therefore remain free from clinical signs of disease over the testing period and on the day of travel. Since infected cats and dogs may have low parasitaemia in which it is difficult to demonstrate the parasites, blood concentration methods should be used.

The following options, given in order of ascending stringency, are available for managing the introduction of *T. evansi* in the commodity:

Option 1.

Cats and dogs could be found to be clinically healthy on the day of export, showing no clinical signs of *T. evansi* infection.

Option 2.

Within 2 days of departure, dogs and cats could undergo direct examination of the blood by a concentration method recommended by the OIE, with no parasites observed.

Option 3.

Dogs and cats could be tested for antibody by an OIE described method and a direct examination of the blood by a concentration method with negative results within the 10 days prior to departure.

Option 4.

Cats and dogs be eligible for importation only from countries that are free from surra.

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25. Nagana (*Trypanosoma brucei*)

25.1. HAZARD IDENTIFICATION

25.1.1. Aetiological agent

Tsetse-transmitted trypanosomosis is a disease complex caused by several species of protozoan parasites of the genus *Trypanosoma* which are transmitted by biting flies of the genus *Glossina* (tsetse flies). *Trypanosoma brucei* comprises a group of indistinguishable heamoparasites including *T. brucei rhodesiense* and *T. brucei gambiense* which are human pathogens and *T. brucei brucei* which is an animal pathogen (Greene 2006).

T. vivax and *T. congolense* are also tsetse fly transmitted animal parasites.

25.1.2. OIE list

Listed within the category of 'cattle diseases'.

25.1.3. New Zealand's status

Trypanosoma spp. are listed as unwanted notifiable organisms (Ministry of Agriculture and Forestry 2008).

25.1.4. Epidemiology

Human African trypanosomosis or "sleeping sickness" is a fatal disease caused by trypanosomes belonging to the species *Trypanosoma brucei* subspecies *rhodesiense* and *gambiense* (Schlater 2004). *Trypanosonma brucei brucei*, *Trypanosoma congolense* and *Trypanosoma vivax* infect animals but not humans. Dogs can be infected by both the human and animal pathogens. The disease in animals is known as "nagana". Humans and animals in 36 African countries between 14º North and 29º South latitude are affected (Matete 2003).

Trypanosomes are transmitted by tsetse flies of the genus *Glossina*. Mechanical transmission by other vectors and vertical transmission does not occur in nature except rarely in the case of *T. vivax*. In the case of biological transmission the organism has a developmental stage in the fly vector which then enters the salivary glands and is inoculated into a new host during feeding (Greene 2006). *T. vivax* has spread to South America where it is apparently transmitted mechanically by biting flies (Connor & Van den Bossche 2004).

Sleeping sickness in humans is endemic in sub-Saharan Africa and is caused by *T. brucei rhodesiense* in East Africa and *T. brucei gambiense* in West Africa. Disease is restricted to the distribution of the tsetse fly vector and is not known outside Africa (Brun 2005).

Dogs are susceptible to these trypanosomes and act as important sentinels for human infection. It is thought that during sleeping sickness epidemics in humans the domestic dog will be the first casualty, rapidly succumbing to disease long before it is noticed in humans (Matete 2003).

Clinical signs in acutely affected dogs are severe. Fever, oedema of the subcutaneous tissues and corneas, purulent ocular and nasal discharges and neurological deterioration similar to rabies is seen. Dogs are not considered reservoir hosts as they are unlikely to maintain infection in nature as dogs have a course of disease lasting 2-4 weeks until death (Greene 2006).

Little information is available on natural infection in cats. Clinical disease has been induced experimentally in this species (Mortelmans & Neetens 1975). A study which experimentally inoculated a cat and a dog resulted in death on day 37 and 28 respectively after infection. The study also demonstrated oral transmission to cats and dogs from being fed infected goat meat. Although infected, the cats and dogs remained asymptomatic until sacrifice 60 days later (Moloo, Losos & Kutuza 1972).

25.1.5. Hazard identification conclusion

Since *Trypanosoma* spp. are not present in New Zealand and are listed as notifiable unwanted organisms, they are concluded to be potential hazards.

25.2. RISK ASSESSMENT

25.2.1. Entry assessment

Natural infection with African trypanosomes usually results in obvious clinical signs and rapid death in dogs and cats. The incubation period is short as dogs act as sentinels for human infections. Some may be chronically affected and asymptomatic, particularly ‘indigenous village dogs’.

Cats and dogs infected by the oral route appear to remain asymptomatic for at least 60 days.

Likelihood of entry is therefore assessed to be non-negligible.

25.2.2. Exposure assessment

African trypanosomosis is an arthropod-borne protozoan. The arthropod vectors are *Glossina* tsetse flies which are restricted to the sub-Saharan African continent.

Since New Zealand lacks the vector, transmission and establishment are not possible. Therefore the risk of exposure is assessed as negligible.

25.2.3. Risk estimation

Since exposure is assessed as negligible, the risk from dogs and cats infected with African *Trypanosoma* spp. is estimated as negligible. Since the risk estimate is negligible, it is not a hazard in the commodity. Therefore risk management measures are not justified.

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ECTOPARASITES SECTION

26. Fleas

26.1. HAZARD IDENTIFICATION

26.1.1. Aetiological agent

Fleas (order Siphonaptera) are small wingless obligate blood-feeding insects. There are about 2500 described species (Wall & Pitts 2005). The preliminary hazard list identifies the following flea species: *Archaeopsylla erinacei*, *Chaetopsylla globiceps*, *Echinophagia gallinacea*, *Hystrichopsylla talpae*, *Nosopsyllus fasciatus*, *Paraceras melis*, *Spilopsyllus cuniculi*, *Tunga penetrans* and *Xenopsylla cheopis*.

26.1.2. OIE List

Not listed.

26.1.3. New Zealand's status

None are listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008). Some of the above species have been reported in New Zealand associated with birds. For example *Nosopsyllus fasciatus* is associated with pigeons, Weka, and North Island fantails (Heath & Bishop 1998). *Xenopsylla cheopis* has been found on gulls and associated with intercepted rats around New Zealand ports but has not established (Heath & Bishop 1998; Kelly et al 2005).

The common cat and dog flea (*Ctenocephalides felis* and *Ctenocephalides canis*) are endemic.

26.1.4. Epidemiology

Fleas are ubiquitous insects and are the most common ectoparasite of companion animals. A limited number of flea species are commonly found on dogs and cats. *Ctenocephalides felis* and *Ctenocephalides canis* are the major species found worldwide (including New Zealand) but *Pulex irritans*, *Ceratophyllus gallinae*, *Archaeopsylla erinacei*, and *Echinophaga gallinacea* are also common species found on companion animals.

However, although fleas are host-preferential rather than host-specific they will feed on any available animal (Kelly et al 2005). Other species of flea other than the usual flea species may therefore be present on an animal. Fleas are capable of transmitting several exotic disease

causing agents, including *Yersinia pestis* and *Francisella tularensis*. They also act as intermediate hosts for cestode and filarial infections (Wall & Pitts 2005).

Fleas must be associated with the host for survival. After feeding on the host's blood, mating occurs and the females lay eggs that drop off in the host's environment (Greene 2006). The eggs cannot withstand major variations in temperature and will not survive below 50 % relative humidity. At 24° C and 78 % relative humidity (most household conditions) *Ctenocephalides felis* will complete its development cycle in 3-5 weeks. Hatched larvae will only survive at temperatures between 13° C and 35° C and mortality is high below 50 % relative humidity. Once fully developed the adults emerge from the pupal cuticle, but this can be delayed up to 1 year at low temperatures. However, at optimum temperatures emergence is rapid. Warm, mobile objects in close proximity induce the emerged flea to jump. Once on the host, feeding and mating take place and egg laying begins (Wall & Pitts 2005; Greene 2006).

The use of insecticides and insect growth regulators with convenient formulations have allowed for control of fleas because of their efficacy and ease of administration. A wide range of products are available, many with long-acting adulticidal activity that also have contact ovicidal and/or larvicidal activity.

26.1.5. Hazard identification conclusion

Fleas are commonly found on cats and dogs, and since they may pose health risks to humans and animals they are concluded to be potential hazards.

26.2. RISK ASSESSMENT

26.2.1. Entry assessment

Fleas are common ectoparasites of cats and dogs that may act as hosts to many different species of flea. Entry is therefore assessed to be non-negligible.

26.2.2. Exposure assessment

The common endemic cat and dog flea have a wide distribution over New Zealand, demonstrating that other species of fleas will be able to survive and become widely dispersed. Since animals are domiciled within houses, this environment may be particularly suitable for fleas to complete their life cycles and increases exposure to human occupants.

Therefore the likelihood of exotic flea species being able to establish is considered to be non-negligible.

26.2.3. Consequence assessment

The major consequences of exotic flea establishment are:
1) The direct effects of parasitism.
2) The possible introduction of exotic flea-borne disease harboured within the flea.

1) Direct effects of parasitism
The parasitic effects of fleas in heavy infestations cause anaemia as a result of blood ingestion, debilitation and skin disease associated with flea allergy dermatitis and bacterial pyoderma.

2) The possible introduction of exotic flea-borne disease harboured within the flea.
Fleas may transmit several exotic disease causing agents, including *Yersinia pestis* and *Francisella tularensis*. They also act as intermediate hosts for cestode and filarial infections. The effects of such organisms on the health of humans and animals may be severe. The consequences are therefore assessed to be non-negligible.

26.2.4. Risk estimation

As entry, exposure and consequence assessments are all assessed to be non-negligible for infested animals, the risk is estimated to be non-negligible. Fleas are classified as a hazard in the commodity; therefore risk management measures can be justified.

26.3. RISK MANAGEMENT

26.3.1. Options

The use of insecticides and insect growth regulators with convenient formulations have allowed for control of fleas because of their efficacy and ease of administration.
The available options for excluding fleas, in ascending order of likely efficacy, are:

Option 1.

Within the 3 days prior to travel, animals for export are to be treated with an effective insecticide.

Option 2.

Treatment as in option 1. *and* inspection, with certification that the animal is free from fleas within 3 days of travel.

Option 3.

Treatment as in option 1. *and* be inspected and found to be free of fleas at the point of departure.

Option 4.

Treatment as in option 1. *and* be inspected and found to be free of fleas at the port of entry before being given biosecurity clearance.

Option 5.

Treatment as in option 1. *and* be inspected and found to be free of fleas at the point of departure *and* be inspected and found to be free of fleas at the port of entry before being given biosecurity clearance.

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27. Leeches

27.1. HAZARD IDENTIFICATION

27.1.1. Aetiological agent

Leeches are annelids and regarded as anatomically and behaviourally specialised earthworms. There are three groups of leeches; jawed leeches *Gnathobdellida,* jawless leeches *Rhyncobdellida* and leeches with no jaw or teeth, *Pharyngobdellida*. The preliminary hazard list identifies *Myxobdella annandalei*. However, there are many other species of exotic leech that are not listed.

27.1.2. OIE List

Not listed.

27.1.3. New Zealand's status

There are eight genera of terrestrial and freshwater leeches with 11 known species in New Zealand. The terrestrial species mostly feed on the blood of birds (Miller 1997). No leeches are listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

27.1.4. Epidemiology

Most leeches are sanguivorous on preferred hosts. However, they are not host-specific and pets might sometimes be infested in endemic areas. Infestation is generally visible, even if attached up the nostril. However, reported cases of internal nasal infestations are extremely rare. Most cases reported are human infestations.

27.1.5. Hazard identification conclusion

Since leech infestation of animals might occur in endemic areas, it is concluded that leeches could be associated with the commodity and are therefore potential hazards.

27.2. RISK ASSESSMENT

27.2.1. Entry assessment

The pharyngobdellida swallow their prey whole which consists of small invertebrates. These leeches are unlikely to be associated with the commodity, however, jawed leeches that are sanguivorous could be.

The first report of nasal infestation of a cat with the parasitic leech *Dinobdella ferox* has only recently been described (Chang et al 2006). This infestation had gone unnoticed for at least 1 month. The only other nasal infestation described was a dog imported to Germany from Nepal and found to have *Myxobdella annandalei* (Gothe et al 1991).

Although leech infestation is a rare event, the entry assessment is considered to be non-negligible for animals exported from endemic areas.

27.2.2. Exposure assessment

Since there are 11 known species present in New Zealand it is concluded that some environments could be suitable for establishment of new species. Leeches are hermaphrodites that can survive long fasting periods in unsuitable environmental conditions by burrowing into the soil. The likelihood that establishment would result from importing infested cats or dogs is probably very low. However, since the environment could be suitable and some leeches are fairly robust survivors the exposure assessment is considered to be non-negligible.

27.2.3. Consequence assessment

Leeches are not known to transmit diseases. However, they may act as intermediate hosts for some nematode parasites. There are generally no health consequences beyond bite irritation and bleeding with wounds sometimes becoming infected. Allergy to leech bite has also been reported. Any blood loss is generally insignificant to the host.

Establishment would require recreational bush walkers to wear clothing to act as a barrier to leech attachment.

Since there could be health implications for humans and animals if an exotic leech species established and eradication would be impossible, the consequences are assessed to be non-negligible.

27.2.4. Risk estimation

Entry, exposure and consequence assessments are all assessed to be non-negligible. As a result the risk estimate for leeches is non-negligible and it is classified as a hazard in the commodity. Therefore risk management measures can be justified.

27.3. RISK MANAGEMENT

27.3.1. Options

The available options for excluding leeches, in ascending order of likely efficacy, are:

Option 1.

Animals could be clinically examined prior to export to provide assurances of freedom from leeches due to their gross visibility.

Option 2.

Further to option 1, point of entry inspection could also be adopted as for ticks and myiasis to alert the examiner to any other ectoparasitic infestation such as leeches should they be present (refer to tick and myiasis sections for further details). If any are found, treatment by applying ivermectin solution, topical anaesthetics or insecticides to paralyse leeches thereby facilitating removal could be performed (Mahato 1990; Chang 2006).

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28. Lice

28.1. HAZARD IDENTIFICATION

28.1.1. Aetiological agents

Lice are host-specific wingless insects in the order Phthiraptera (Soulsby 1968). The preliminary hazard list identifies the dog louse *Heterodoxus spiniger* as an exotic species.

28.1.2. OIE List

Not listed.

28.1.3. New Zealand's status

The common lice species found on cats and dogs are endemic. None are listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

28.1.4. Epidemiology

Heterodoxus spiniger is a biting louse of dogs and a few wild canids, mostly in tropical regions. As with other lice, transmission is by direct contact with an infested animal (Soulsby 1968). Louse eggs or nits are glued to hairs of the host near the skin surface and are translucent, and suboval. The three nymphal stages, of increasing size, are smaller than adults but otherwise resemble them in behaviour and appearance. About 3-4 weeks are required to complete one generation.

Dogs in poor health may become heavily infested and the coat may become rough, dry and matted. The constant crawling and piercing or biting of the skin causes nervousness and infested dogs may injure themselves from biting and scratching (Soulsby 1968).

Lice are active and although they are small they can be seen moving through the hair (Merck 2008).

Louse control requires treatment with an effective insecticide. There are many effective acaracides that treat lice infestations. Dogs can be treated with dips, washes, sprays, or dusts. Effective compounds include permethrin, pyrethrins, rotenone, methoxychlor, lindane, diazinon, malathion or coumaphos. Doses of ivermectin high enough to be effective against lice are not recommended in dogs (Merck 2008).

28.1.5. Hazard identification conclusion

Heterodoxus spiniger is exotic and since infestation may cause irritation that may require treatment, this agent is concluded to be a potential hazard.

28.2. RISK ASSESSMENT

28.2.1. Entry assessment

Heterodoxus spiniger is found on dogs in tropical areas. Therefore there is a low but non-negligible likelihood that such dogs imported from these regions could harbour the organism. The likelihood of entry is therefore assessed to be non-negligible.

28.2.2. Exposure assessment

Since imported dogs infested with lice would directly contact indigenous dogs, the likelihood of exposure is assessed to be non-negligible.

28.2.3. Consequence assessment

Treatment for lice is readily available and efficient and would not require any further effort than that for fleas.

Dog lice will not infest and establish in other animals or humans, and as such there are no consequences for human health or the environment. However, louse infestation is contagious, may result in irritation, and treatment does incur some costs. Therefore, consequences are assessed to be minor, but non-negligible.

28.2.4. Risk estimation

Release, exposure and consequence assessments are all assessed to be non-negligible for infested animals. Therefore, the risk is estimated to be non-negligible for lice and they are classified as a hazard in the commodity. Therefore, risk management measures can be justified.

28.3. RISK MANAGEMENT

28.3.1. Options

Since there are a large number of effective, convenient acaracide treatments available to eliminate lice infestations, imported animals could be treated in conjunction with other treatments for external parasites. Lice are active and can be observed moving through the hair. Therefore, inspection could easily ensure freedom before departure.

The available options for excluding lice, in ascending order of likely efficacy, are:

Option 1.

Within the 3 days prior to travel, animals for export are to be treated with an effective insecticide.

Option 2.

Certification that the animal has undergone pre-export treatment with an effective acaracide within 3 days of travel, and been inspected and found to be free of lice.

Option 3.

The animal has undergone pre-export treatment with an effective acaracide within 3 days of travel, and been inspected and found to be free of lice at the point of departure.

Option 4.

Undergo option 3, *and*; be found to be free of lice upon inspection at the port of entry before being given biosecurity clearance.

Option 5.

Treat with an effective acaracide at the port of entry before being given biosecurity clearance.

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29. Mites

29.1. HAZARD IDENTIFICATION

29.1.1. Aetiological agents

Mites and ticks belong to the subclass Acarina in the class Arachnida which includes spiders and scorpions. Mites are minute relatives of ticks (Soulsby 1968). The preliminary hazard list includes *Lynxacarus radovskyi*, *Pneumonyssus caninum* and *Trombicula autumnalis*. Other Trombiculids such as *Leptotrombidium deliense* are also considered here as they are vectors of scrub typhus, an acute rickettsial disease of humans (Lerdthusnee et al 2002).

29.1.2. OIE List

Not listed.

29.1.3. New Zealand's status

No mites are listed on the unwanted organisms register (Ministry of Agriculture & Forestry 2008). No reports could be found of the above listed mites in New Zealand therefore they are considered to be exotic organisms.

29.1.4. Epidemiology

The family Trombiculidae contains the mites whose parasitic larvae are called 'harvest mites', 'chigger mites' and various other names (Soulsby 1968). The nymphs and adults of *Trombicula autumnalis* are free-living, whereas the larvae parasitise any animal and humans. In Britain, the larvae are common in the late summer and autumn (Soulsby 1968). The larvae may cause generalised pruritis and lesions in the interdigital spaces of infested dogs. They are red, yellow or pink in colour and easily visible.

Rickettsia akari is a rare cause of rickettsial pox in humans. It is maintained in a mite/mouse cycle with humans and dogs serving as accidental hosts. Infection is from the bite of a rodent mite, *Liponyssoides* spp. which has been discussed in the Rickettsiosis chapter of this risk analysis.

Orientia tsutsugamushi, the causative agent of scrub typhus in humans, is transmitted by the bite of *Leptotrombidium* mite larvae (Kumar et al 2004). This mite is both vector and maintenance host. The distribution of scrub typhus reflects that of the mite and and its rat/mice hosts. The disease is reported only in the Asian-Pacific region where the mite vector occurs in tropical climates where the temperature and humidity favour its survival (Wang et al

2002). Antibody to the rickettsia was discovered in US army tracker dogs in Vietnam. No signs of clinical disease were noted in the seropositive dogs. Dogs experimentally infected with *O. tsutsugamushi* did not show clinical illness.[C]

Lynxacarus radovskyi is a mite of domestic cats discovered in Hawaii in 1974. The most evident clinical signs are hair loss and intense pruritis that is proportional to the number of mites and length of time of infestation (Faustino 2004). Cases have been diagnosed occasionally in Brazil, USA, Oceania and Australia (Bowman & Domrow 1978). The only reported case in New Zealand was a cat from Samoa that was held in quarantine (Heath & Mariadass 1999).

Nasal infestation of dogs by the mite *Pneumonyssus caninum* has been reported on numerous occasions from dogs in the USA. This mite has also been reported in Canada, Australia, South Africa, Japan and Europe. Infections in dogs are usually subclinical or present with only mild clinical signs (sneezing). A severe rhinitis may occasionally occur (Fraser 1991).

The adult mite has a pale yellow body and most infections are found at necropsy. A few cases have been reported in which the mite has been found on the nose of sleeping dogs. It is believed that dog to dog transmission is by the direct transfer of larvae from one dog to another. Treatment appears easily achieved by the subcutaneous administration of ivermectin at 200 mcg/kg (Bowman 2000). The mite has never been reported in New Zealand dogs, although it is present in many countries around the world including Australia (Stone 2005).

Mites generally, are easily diagnosed on clinical examination and control requires treatment with an effective insecticide. There are many effective acaracides that treat mite infestations.

29.1.5. Hazard identification conclusion

The larvae of *Leptotrombidium deliense* may transmit scrub typhus to humans. Other mite species may cause generalised pruritis and skin lesions in infested animals. Mites are therefore concluded to be potential hazards.

29.2. RISK ASSESSMENT

29.2.1. Entry assessment

Mites are common ectoparasites of dogs and cats and are widely distributed. The likelihood of entry is therefore assessed to be non-negligible.

29.2.2. Exposure assessment

Leptotrombidium mites are unlikely to establish because of their requirement for a humid and tropical environment. However, infested dogs will be in direct contact with humans and therefore increase the risk of scrub typhus being transmitted to humans.

Since imported animals infested with mites could directly contact indigenous animals, the likelihood of exposure is assessed to be non-negligible.

[C] Refer to Chapter 21, Rickettsiosis for further information.

29.2.3. Consequence assessment

Importation of new species of mites such as *Pneumonyssus caninum* that are of minor importance overseas are unlikely to have significant health effects on dogs in New Zealand. Treatment is available for infested dogs.

An animal could be carrying mites infected with *O. tsutsugamushi* and therefore pose a health threat to humans. Infestation of dogs and cats could cause pruritis and skin lesions, and costs for treatment would be incurred. Therefore consequences are assessed to be non-negligible.

29.2.4. Risk estimation

Entry, exposure and consequence assessments are all assessed to be non-negligible for mite infested animals. Therefore the risk is estimated to be non-negligible, and risk management measures can be justified.

29.3. RISK MANAGEMENT

29.3.1. Options

Treatment of external mites is readily available and efficient and would not require any further effort than that of flea control in most cases. Since there are a large number of effective acaracide treatments available to eliminate mite infestations, imported animals could be treated in conjunction with other treatments for external parasites. Mite infestation can be easily diagnosed by observing mites on clinical examination. Therefore, inspection could ensure freedom before departure.

A dog infested with nasal mite may display mild clinical signs such as sneezing or a more severe rhinitis. Animals displaying such clinical signs could be examined more specifically for nasal mites before travelling and treated if diagnosed.

The available options for excluding mites, in ascending order of likely efficacy, are:

Option 1.

Certification that the animal has undergone pre-export treatment with an effective acaracide within 3 days of travel, and been inspected and found to be free of mites.

Option 2.

Certification that the animal has undergone pre-export treatment with an effective acaracide within 3 days of travel, *and* been inspected and found to be free of mites on the day of departure.

Option 3.

Certification that the animal has undergone pre-export treatment with an effective acaracide within 3 days of travel, *and*; been inspected and found to be free of mites upon inspection at the port of entry.

Option 4.

For countries where nasal mite is endemic, restrictions could be as for option 3. but additionally the animal could be examined by nasal endoscopy or treated by subcutaneous administration of ivermectin at 200 mcg/kg.

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30. Myiasis (Fly Larvae Infestation)

30.1. HAZARD IDENTIFICATION

30.1.1. Aetiological agent

Myiasis is a disease caused by the invasion of the tissues or open cavities (e.g. external ears, mouth, nares) of animals by dipteran larvae (Acha & Szyfres 1987). There are many species of fly that cause myiasis and the preliminary hazard list identifies *Cochliomyia hominivorax*, *Chrysomya bezziana*, *Dermatobia hominis*, *Lucilia caesar*, *Cordylobia anthropophaga*, *Cuterebra* and *Wohlfahrtia* as species that infest cats and/or dogs.

30.1.2. OIE list

New World screwworm (*Cochliomyia hominivorax*) and Old World screwworm (*Chrysomya bezziana*) are listed under diseases of multiple species (OIE 2007).

30.1.3. New Zealand's status

Cochliomyia hominivorax and *Chrysomya bezziana* are listed as unwanted, notifiable organisms. *Cuterebra* spp. are listed as "other exotic organisms" (Ministry of Agriculture & Forestry 2008).

30.1.4. Epidemiology

Both the New World screwworm fly (NWS) *Cochliomyia homnivorax*, and the Old World screwworm fly (OWS) *Chrysomya bezziana*, are obligate parasites of warm-blooded animals, including humans and rarely birds. They are blowflies of the family Calliphoridae, but unlike most other species of blowfly, screwworms lay their eggs at the edges of wounds on live mammals or at their body cavities. Within 24 hours of eggs being laid, larvae (maggots) that are screw-shaped hatch and burrow into the wound in a characteristic screwworm fashion. This results in severe tissue destruction and infested wounds emit an odour that is highly attractive to other gravid female flies (Acha & Szyfres 1987). If untreated, the destructive activity of the larvae may lead to the death of the animal within a very short time.

The larvae reach maturity about 4-8 days after hatching from the egg and leave the wound, falling to the ground into which they burrow and pupate. Adult flies emerge from the pupae in 1 week (at 28°C) to 2 months time dependent on temperature and humidity (Acha & Szyfres 1987; Ausvetplan 1996). Freezing or sustained soil temperatures of 8°C or less kill the pupae (Merck 2006).

The optimal temperature range for the fly is 20-30°C and this has had a major influence on their distribution. Flies will not move at temperatures below 10°C, and in the range 10-16°C they are very sluggish and probably will not mate. At no stage in the fly's life cycle is it resistant to freezing and over-wintering in frost areas does not occur (Ausvetplan 1996).

The OWS fly distribution covers the tropical areas of Africa, the Indian subcontinent, Southeast Asia and Papua New Guinea (Acha & Szyfres 1987). However, it has never become established in Australia (Ausvetplan 1996). The NWS fly is endemic in parts of Central and South America as far south as Argentina. It has been eliminated from the USA, Mexico and several Central American countries, where it was previously endemic, by use of the sterile insect technique. An outbreak in Libya in 1988 was also eradicated by applying the sterile insect technique (Ausvetplan 1996). Like OWS, NWS has never established in Australia.

Dermatobia hominis, the tropical warble fly, lives in humid forested areas and is one of the most important parasites of cattle in Latin America, where it is distributed between southern Mexico and northern Argentina (Acha & Szyfres 1987). Larval stages are found in many hosts, including humans, but cattle and dogs are infested most commonly (Soulsby 1968). The adult fly fastens its eggs to different types of insects of which 49 (mostly mosquitoes and muscoid flies) have been described as vectors of *D. hominis* in Latin America. These vectors then transport the eggs to warm-blooded hosts where they hatch as the insect vector feeds. The warble fly larvae penetrate the skin of the animal within a few minutes of hatching and remain in the subcutaneous tissue for 4-18 weeks (Acha & Szyfres 1987) where they form 'warbles' which are connected by breathing holes through the skin to the air. When mature, the larvae leave the host and drop to the ground, burrow, and pupate (Soulsby 1968).

The African tumbu fly *Cordylobia anthropophaga*, is responsible for boil-like myiasis in both humans and animals in Africa, particularly in the sub-Saharan regions. Female flies produce 100-500 banana-shaped eggs, usually depositing them in dry, shady, sandy soil that has often been contaminated by urine or faeces (Soulsby 1968; Merck 2006). Eggs are never deposited on the skin of the host. Eggs hatch after 1-3 days, and the larvae can survive up to 15 days while waiting for a host, penetrating the host in as little as 25 seconds. After penetration, larvae reside in a cavity in the dermis and hypodermis (Merck 2006). This cavity communicates to the external environment by means of a central breathing pore. A single larva is found in each cavity, within which the larva develops. Larvae require 7-15 days to mature and then emerge through the breathing pore, dropping to the ground, where they pupate. Adult flies emerge 10-20 days later. The dog is the most affected domestic animal and is the usual definitive host. However, many mammals, including humans, can be infested (Acha & Szyfres 1987).

Cuterebra (order Diptera, family Cuterebridae) cause opportunistic, parasitic infestations of cats and dogs. These are dipteran parasites of the Western Hemisphere with some 34 different species being present in North America (Bowman 2000). The flies deposit eggs around the openings of animal nests, burrows and along runways of their normal hosts (rodents and lagomorphs) or on stones or vegetation in these areas. Animals become infested as they pass through contaminated areas; the eggs hatch in response to heat from a nearby host. The larvae enter the host's body through the mouth or nares during grooming or, less commonly, through open wounds. After penetration, the larvae migrate to various species-specific subcutaneous locations on the body, where they mature and communicate with the air through a breathing pore. After approximately 30 days, the larvae exit the skin, fall to the soil, and pupate (Bowman 2000).

The gray flesh fly, *Wohlfahrtia vigil* causes cutaneous myiasis in North America. Larval stages are maggot-like in appearance and are adapted to maintain an attachment to living tissues with strongly developed oral hooks. *Wohlfahrtia vigil* is larviparous i.e. it deposits larvae (not eggs) on healthy, uninjured skin of suitable hosts. Larvae penetrate the unbroken skin and form a boil-like swelling. Development to the infective third-larval stage is usually completed in 9-14 days. The parasites then drop to the ground and pupate, approximately 11-18 days later, depending on temperature.

W. magnifica occurs in the European and African Mediterranean area, the Middle East, the Russian Federation and China. The fly is attracted to open wounds and, being larviparous, deposits larvae in these wounds. It is an important disease of sheep in southern parts of the Russian Federation (Acha & Szyfres 1987).

The following larval dipterans are often referred to as facultative myiasis-producing flies: *Musca domestica* (the house flies) *Calliphora*, *Phaenicia*, *Lucilia*, and *Phormia* spp. (the blow flies or bottle flies) and *Sarcophaga* spp. (the flesh flies). Their adult stages are synanthropic flies, i.e. they are often associated with human dwellings and readily fly from faeces to food. Larval stages are usually associated with skin wounds of any domestic animal that have become contaminated with bacteria or with a matted hair coat contaminated with faeces. In facultative myiasis, the adult flies are attracted to a moist wound, skin lesion, or soiled hair coat. As adult female flies feed in these sites, they lay eggs. The eggs hatch, producing larvae that move independently about the wound surface, ingesting dead cells, exudate, secretions, and debris, but not live tissue. This condition is known as fly strike. Unless appropriate therapy is administered, the infested animal may die, generally from shock, intoxication, or infection. A distinct, pungent odour permeates the infested tissue and the affected animal (Merck 2006).

30.1.4.1. Diagnosis

Myiasis is easily diagnosed from a careful clinical examination of the skin, any open wounds and around body cavities.

C. bezziana (OWS) produce a particularly vile myiasis. Female flies are attracted to open wounds, and larvae burrow deep into the wound which results in severe tissue destruction. Infested wounds emit an odour that attracts more flies.

The presence of a superficially situated dermal swelling with a central opening, especially if more than one is present, may lead to a tentative diagnosis of myiasis due to *C. anthropophaga* or *D. hominis* (Soulsby 1968).

Cats and dogs are abnormal hosts for *Cuterebra* spp. and aberrant migration can involve the head, brain, nasal passages, pharynx and eyelids. In the skin, typical *Cuterebra* lesions are fistulous swellings about 1 cm in diameter, around the head, neck and trunk. The hair is often matted, and a subcutaneous swelling is present beneath the lesions. Purulent material may exude from the lesion. Some infested cats and dogs may display clinical signs of respiratory disease, such as sneezing or nasal discharges. If larvae migrate to the brain, neurological signs may occur (Bowman 2000).

The first indication that an animal is infested with *Wohlfahrtia vigil* is exudation of serum and matting of the hair coat over the site of penetration. The presence of a dermal swelling with a central opening may lead to a tentative diagnosis of myiasis due to *W. vigil*. On the third or

fourth day, the larvae produce abscess-like lesions. The hair coat often becomes parted over the summit of the lesions and reveals an opening 2-3 mm in diameter. The posterior aspect of the larva is visible in these openings, through which it breathes. The penetration of the skin by the larvae, their development in the subcutaneous tissues, and secondary bacterial infection produce intense irritation and inflammation (Merck 2006).

30.1.4.2. Treatment

Treatment and control measures for myiasis in cats and dogs are limited. With most myiasis infestations, removing maggots from existing deep tissue pockets may need surgical exploration, debriding and flushing. This would involve sedating or anaesthetising the animal (Merck 2006).

Larvae of *C. anthropophaga* and *Wohlfahrtia* species can be removed by coating the breathing pore with a thick, viscous compound, such as heavy oil, or liquid paraffin. Clogging the pore causes the larva to become hypoxic and leave the cavity in search of oxygen (Merck 2006).

30.1.5. Hazard identification conclusion

Myiasis is a debilitating, serious disease of warm-blooded animals. *Cochliomyia hominivorax* (NWS) and *Chrysomya bezziana* (OWS) are listed as unwanted, notifiable organisms. *Cuterebra* spp. are listed as 'other exotic organisms'. All the listed agents that cause myiasis are therefore concluded to be potential hazards.

30.2. RISK ASSESSMENT

30.2.1. Entry assessment

Cats and dogs coming from endemically affected countries could be infested with myiasis. Myiasis is generally clinically evident on careful examination of the skin, particularly under any matts, open wounds and around body cavities. Some infested cats and dogs may display clinical signs of respiratory disease, such as sneezing or nasal discharges with *Cuterebra* infestation. If larvae migrate to the brain, neurological signs may occur (Bowman 2000).

Pre-export veterinary examination on the day of travel that certifies the animal is clinically healthy should exclude such infested animals from travel. However, the animal may be infested immediately prior to departure, or en-route to New Zealand with clinically undetectable larvae. The likelihood that infested animals will be imported with myiasis is therefore considered to be extremely low but non-negligible.

30.2.2. Exposure assessment

New Zealand animals could become infested if larvae in infested imported animals were able to complete their life cycle and the resulting adult flies mated sucessfully. However, it is unlikely larvae in imported cats and dogs would leave their hosts naturally since infestation is clinically obvious, and veterinary treatment would most likely be sought. If veterinary intervention did not occur, New Zealand's climate is probably not suitable for the pupal development of the tropical myiasis fly species. Facultative myiasis-producing flies such as *Lucilia* spp, of which some are already present in New Zealand, are more likely to establish.

The likelihood that New Zealand animals will be exposed to exotic myiasis is therefore considered to be low but non-negligible.

30.2.3. Consequence assessment

If the parasites were to establish it would have severe economic effects on New Zealand's primary industries due to production losses and treatment costs.
Occasional infestations of humans would require medical treatment. The consequences for feral and wild animals are likely to be non-negligible since parasites are not host specific, generally affecting any warm-blooded mammal and birds.

Since there could be severe negative effects on animal production and cases of myiasis in many animal species, including humans, the consequences are considered to be non-negligible.

30.2.4. Risk estimation

Since entry, exposure and consequence assessments are all assessed to be non-negligible, risk is estimated to be non-negligible. Risk management measures can therefore be justified.

30.3. RISK MANAGEMENT

30.3.1. Options

OWS and NWS are OIE listed, and the *Code* makes recommendations for the safe importation of animals. Therefore, all cats and dogs introduced from countries that are infested with screwworm could be subjected to measures that are based on the recommendations of the OIE (Option 2). These recommendations would also mitigate the risks from other dipteran larval infestations.

Post-arrival quarantine for 30 days would allow development of larval stages and detection of infestation.

The available options for excluding myiasis, in ascending order of likely efficacy, are:

Option 1.

When importing cats and dogs from countries considered infested with any of the following: New World or Old World screwworm, *Dermatobia hominis*, *Lucilia* spp, *Cordylobia anthropophaga*, *Cuterebra* or *Wohlfahrtia* species:

Animals for export could be subjected to a close inspection of the skin for wounds with egg masses or larvae immediately prior to shipment. Only animals that are free from infestation and that have a dry, unsoiled and unmatted hair coat would be eligible for shipment.

Option 2.

As for option 1. *and* in addition, the inspection could be repeated at the arrival point in New Zealand. This inspection could identify any infestation acquired en route and be integrated with tick inspections.

Option 3.

As for option 2. *and* in addition, post-arrival quarantine with daily inspections for 30 days.

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31. Ticks

31.1. HAZARD IDENTIFICATION

31.1.1. Aetiological agent

Phylum: Arthropoda, Class: Arachnida, Subclass: Euarachnida, Order: Acarina, Suborder: *Ixodoidea* which is subdivided into two families, the Argasidae and the Ixodidae.

Ticks of medical importance are classified into two families; Argasidae (soft ticks) and Ixodidae (hard ticks). Argasid ticks have soft leathery bodies and feed for 5-25 minutes. There are approximately 170 species in this group. The Ixodidae family contains around 650 species, which are characterized by a hard body plate and a prolonged feeding time (Grattan-Smith et al 1997). For example *Rhipicephalus sanguineus* may take up to 21 days to engorge (Soulsby 1969).

31.1.2. OIE List

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

31.1.3. New Zealand's status

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

All exotic ticks are notifiable under the Biosecurity Act 1993 (Ministry of Agriculture & Forestry 2008).

31.1.4. Epidemiology

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

The life cycle of ticks may be classified according to their location when they moult between life stages, either on the host (one-host tick) or off the host (multi-host tick).
All twelve exotic tick species intercepted in New Zealand have been three-host ticks, requiring a different host for every life stage: larva, nymph, and adult (Loth 2005). An infected tick may carry a particular tick-borne pathogen for life. A female tick can transmit some blood-borne pathogens to her eggs by transovarial transmission (through the eggs to the

next generation of larvae) while other pathogens may only be transmitted transstadially. Other pathogens can be transmitted transovarially and transstadially. As each subsequent life stage must find a host and feed, it is possible to transmit tick-borne organisms to multiple hosts.

Dogs are the preferred host for *Rhipicephalus sanguineus* (known as the 'brown dog tick') and are also suitable hosts for a variety of tick species which are more adapted to other mammalian species, e.g. *Haemaphysalis longicornis* (known as the 'cattle tick').

The tick species most likely to be associated with imported dogs are *Rhipicephalus sanguineus*, *Ixodes holocyclus*, and *Haemaphysalis longicornis*. These are all ixodid, three host ticks. Although *H. longicornis* is present in New Zealand a number of diseases potentially vectored by this tick are not. Therefore *H. longicornis* on imported dogs poses a potential risk of introducing diseases not presently in New Zealand. Although other species of tick have occasionally been identified in New Zealand, these three species are the most commonly introduced, mostly from Australia.

Rhipicephalus sanguineus is a vector for a wide range of infectious agents, particularly those causing disease in dogs such as *Babesia canis*, *Babesia gibsoni*, *Ehrlichia canis*, haemobartonellosis and hepatozoonosis (Irwin & Jefferies 2004; Loth 2005).
R. sanguineus also has the potential to transmit the zoonotic *Borrelia burgdorferi* the cause of Lyme disease which is the most common tick-transmitted disease in humans in the Northern Hemisphere (up to 155 cases per 100,000 individuals) (Wilske 2005).

31.1.5. Hazard identification conclusion

Since ticks are unwanted notifiable organisms that pose important health risks to humans and animals, they are concluded to be potential hazards.

31.2. RISK ASSESSMENT

31.2.1. Entry assessment

Dogs are recognised as a significant pathway for the introduction of ticks. Most ticks arriving in this country are attached to dogs or humans or their clothing (Loth 2005). Over the last 25 years annual exotic tick interceptions have steadily increased (Loth 2005). Suggested causes for the increasing number of tick interceptions and incursions include use of acaracides with less than full efficacy against ticks and an increase in dog imports, passenger arrivals and imported goods (Loth 2005).

During the period February 2001 to November 2004, there were 17 pre- and post-border tick interceptions. Fourteen of these (82 %) were attached to dogs, the remaining three were associated with humans or their possessions. Of the 14 reported cases involving dogs, 8 were intercepted at airports by detecting the ticks during dog inspections (Waite 2005).

Since dogs are a significant pathway for the introduction of ticks, entry is assessed to be non-negligible. Cats also host ticks, however their grooming allows removal and they are not as important a pathway as dogs. However, ticks do parasitise cats and are therefore assessed to have a non-negligible likelihood of introducing ticks.

31.2.2. Exposure assessment

Ticks can survive long periods and have many susceptible hosts. The host animals may disperse ticks widely from the point of initial introduction. The cattle tick *H. longicornis* has a distribution over the North Island and parts of the northern South Island (Loth 2004) specifically Golden Bay/Takaka, demonstrating that ticks can survive and become widely dispersed. It is considered that New Zealand's mainly moist-temperate climate provides an ideal environment for all but the most strictly tropical or arid region tick species (Heath 2001).

R. sanguineus (Brown dog tick) is the most commonly intercepted exotic tick and most have come from Australia (Loth 2005). It is a tropical/subtropical tick and is most commonly found between latitudes 50° North and 35° South of the equator (Roberts 1970; Brown 2005).

In New Zealand, it is likely that *R. sanguineus* could establish in Northland and possibly some other areas with sufficient high mean temperatures (Loth 2004; McColl & Tenquist 1980). *R. sanguineus* can establish in heated houses outside its usual latitudes if suitable hosts are present. It readily infests homes and persists without the need to maintain a population outdoors (Irwin & Jefferies 2004). On three occasions in 1979, 2000 and 2004, a New Zealand house became infested with *R. sanguineus*. Each time the tick was eradicated by applying treatments to the house, household effects, surrounds and animals (Loth 2005). The most recent incursion in 2004 resulted in property fumigation with surveillance carried out on dogs in the local area (Stone 2005).

Ixodes holocyclus (paralysis tick), the second most intercepted tick species, inhabits the east coast of Australia as far south as Victoria. As a similar climate exists in regions of the North Island of New Zealand, it is reasonable to conclude that it could establish itself there (Loth 2005).

Other species of ticks that could be introduced from temperate climate countries are also likely to be capable of establishing.

Therefore, the likelihood of exotic tick species attached to dogs being able to establish within New Zealand is considered to be non-negligible.

31.2.3. Consequence assessment

The major consequences of exotic tick establishment are:
1) The direct effects of parasitism and toxicity.
2) The possible introduction of exotic tick-borne disease harboured within the tick.
3) An increased risk of introduced exotic diseases being able to establish in New Zealand if suitable tick vectors are established here.

1) Direct effects of parasitism

The parasitic effects of ticks in sufficient numbers can include anaemia as a result of blood ingestion, debilitation and skin disease associated with hypersensitivity and bacterial pyoderma (Irwin & Jefferies 2004). *I. holocyclus*, the Australian paralysis tick is one of the most toxic of all the worlds paralysing ticks. It is the cause of paralysis and death in pets, domestic production animals, mice and humans (Grattan-Smith et al 1997). Tick paralysis has been estimated to affect up to 20,000 domestic farm animals and 75,000 pets annually in Australia (Grattan-Smith et al 1997; Merial 2008).

2) Exotic disease associated with ticks
The species of tick and tick-borne disease a dog may carry into New Zealand is dependent on the species in the country of origin and the existence of tick-borne disease in that country. Multiple organisms may be present in one tick, e.g. *Ixodes ricinus* (widespread in the UK and Europe) may harbour tick-borne encephalitis virus, *Borrelia burgdorferi* (Lyme disease) and *Rickettsia helvetica* simultaneously (Grattan-Smith et al 1997).

Since most imported ticks are *R. sanguineus* arriving attached to dogs from Australia, the most likely risks are the tick-borne diseases known to cause disease in Australian dogs, such as babesiosis, ehrlichiosis and heptazoonosis (Jefferies et al 2003). *R. sanguineus* also has the potential to transmit the zoonotic *Borrelia burgdorferi* or Lyme disease and important rickettsial diseases such as Boutonneuse fever and Rocky Mountain spotted fever. Tick species from the Northern Hemisphere could also introduce and transmit Lyme disease (Wilske 2005).

3) Increased biosecurity threat if exotic tick vectors exist in New Zealand
Q fever is a zoonotic disease caused by *Coxiella burnetii*, which is present in most countries apart from New Zealand (Hilbink et al 1993). Based on notification data from Australia, Q fever is known to be active in southern Queensland/northern New South Wales where it is the cause of significant human illness and subsequent economic loss (Garner 1997).

Q fever has been transmitted between guinea pigs by *I. holocyclus*, and *R. sanguineus* (Williams & Sanchez 1994). *H. longicornis* (as[D] *H. bispinosa*) can be infected with *Coxiella burnetii* but infection could not be transmitted to guinea pigs (Smith 1942).
If new tick species were to become established in New Zealand the likelihood of exotic tick-borne diseases establishing here at some point in the future is increased over the likelihood that exists now. For example, the absence of Q fever in New Zealand may be attributable to the limited vector potential of *H. longicornis*. The introduction and establishment of *R. sanguineus* would greatly increase the risk of many exotic dog diseases establishing in New Zealand.

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

31.2.4. Risk estimation

Entry, exposure and consequence assessments are all assessed to be non-negligible for infested animals. As a result the risk estimate for ticks is non-negligible and they are a hazard in the commodities. Therefore risk management measures can be justified.

[D] *Haemaphysalis bispinosa* was the name incorporating *Haemaphysalis longicornis* in early studies until morphological characteristics to differentiate the two species were identified by Hoogstraal et al (Hoogstraal et al 1968).

31.3. RISK MANAGEMENT

31.3.1. Options

Important points when considering options for the effective management of the importation of ticks in the commodity are:

In 2004, MAF Biosecurity New Zealand commissioned a review of the published literature on the relative efficacy of acaracides. A recognised expert evaluated a series of studies, looking closely at repellent effects, efficacy and duration of protection of acaricides belonging to seven chemical classes, either alone or as mixtures (Heath 2004). This report supported fipronil spot-on as the best acaricide to be used as a component of control measures. However, there is no known acaracide, or acaracide combination, that is consistently 100 % effective for all ticks for any time period. An area of particular concern with fipronil was the poor control of ticks in the ears of dogs. Ticks may be resistant to a range of acaracides. Tick inspections are therefore an important adjunct to acaracide treatment since treatment alone cannot be relied upon.

Tick inspections are also important for the management of the risk posed by *B. gibsoni*. [E]

The available options for excluding ticks, in ascending order of likely efficacy, are:

Option 1.

Treatment of dogs and cats to be exported with fipronil or other effective acaracides, within the 7 days immediately prior to export.

Option 2.

As for option 1. but in addition, inspection by a trained MAFBNZ inspector at the point of entry. The inspection could be carried out to ensure that animals are well groomed and free from ticks; *and*

the contents of the container in which the animal arrived is free from ticks.

Animals found to be infested with ticks could be transferred to a transitional facility and treated with a different acaracide from that used previously. It could be held for 48 hours following treatment so as to allow the active ingredient to kill any undetected ticks. At the facility the container could be thoroughly steam cleaned to remove any remaining ticks. The container and bedding could then be destroyed or treated with an acaracide. All ticks found could be sent to a laboratory for identification.

A biosecurity clearance could be given 48 hours after treatment when the inspector is satisfied that the animal and container are tick-free.

Fractious, unmanageable and dangerous dogs or cats could be directed to a transitional facility for inspection by a veterinarian in the presence of the owner, after having been tranquillized if

[E] Import risk analysis: *Babesia gibsoni* in dogs (*Canis Familiaris*) and dog semen (MAF 2003). Bridging document, Import requirements for *Babesia gibsoni* (Aug 2004).

necessary. If ticks were to be found, the animal could be held in the transitional facility and treated and inspected as described above.

Option 3.

As for option 2. but the pre-export treatment of cats and dogs could be the same as an option in the babesiosis chapter for excluding ticks, the animal to be certified by a veterinarian as having been treated with an effective acaracide twice at 2 week intervals during the 4 week period prior to export, and been found to be free from ticks at each treatment.

Option 4.

Post arrival quarantine.

A quarantine period of a sufficient duration to allow the free-living stages to detach and be captured would ensure that no ticks were introduced on imported dogs and cats (Heath 2002). This option may be regarded as excessively restrictive on trade between Australia and New Zealand. It would also require specialised facilities to ensure that ticks are captured and destroyed.

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ENDOPARASITES SECTION

The section on endoparasites is divided into three major sections:
- nematodes and acanthocephalans
- trematodes
- cestodes
- All references are collated at the end of this section.

32. Nematodes and Acanthocephalans

32.1. HAZARD IDENTIFICATION

32.1.1. Aetiological agents

The following nematode parasites were identified in the preliminary hazard list:

Nematodes
Ancylostoma spp.
Ancylostoma braziliense
Ancylostoma ceylanicum
Angiostrongylus spp.
Angiostrongylus (parastrongylus) cantonensis
Angiostrongylus vasorum
Capillaria spp.
Capillaria (Pearsonema) plica
Capillaria (Pearsonema) feliscati
Crenosoma spp.
Crenosoma vulpis
Cyathospirura spp.
Cyathospirura seurati (dasyuridis)
Cylicospirura spp.
Cylicospirura heydoni
Cylicospirura felineus
Cylicospirura subaequalis
Dioctophyma spp.
Dioctophyma renale
Dracunculus spp.
Dracunculus medinensis
Dracunculus insignis
Filaroides (Andersonstrongylus) spp.
Filaroides milksi
Filaroides hirthi
Gnathostoma spp.
Gnathostoma spinigerum
Gurtia spp.

Gurtia paralysans
Lagochilascaris spp.
Lagochilascaris minor
Lagochilascaris major
Mammamonogamus spp.
Mammomonogamus ierei
Mammomonogamus auris
Physaloptera spp.
Physaloptera praeputialis
Physaloptera rara (felidis)
Physaloptera canes
Spirocerca spp.
Spirocerca lupi
Spirura spp.
Spirura rytipleurites
Strongyloides spp.
Strongyloides planiceps (cat)
Strongyloides felis
Strongyloides stercoralis
Strongyloides tumefaciens
Thelazia spp.
Thelazia callipaeda
Thelazia californiensis
Toxocara spp.
Toxocara malaysiensis
Toxocara mystax
Trichuris spp.
Trichuris felis (serrata, campanula)

Acanthocephalans
Macracanthorhynchus ingens.
Oncicola spp.
Oncicola canis
Oncicola pomatostomi

32.1.2. OIE list

None of the species in Section 32.1.1 are listed.

32.1.3. New Zealand's status

All the species listed in 32.1.1 have been identified in the preliminary hazard list as species that do not occur in New Zealand.

32.1.4. Epidemiology

Relevant information on each of the species is given below:

Angiostrongylus vasorum or French heartworm is a parasite of foxes and dogs. The adult worms develop in the pulmonary arteries and lay eggs that lodge in lung capillaries, hatch and develop into larvae that break out into airspace and are passed out in the faeces. Snails feed on dog faeces and frogs on snails and foxes and dogs on snails or frogs which are paratenic hosts

(Chapman et al 2004; Conboy 2000; Conboy 2004). The parasites occur in France, England, Europe, Africa, former Soviet Union countries, South America and Canada. Diagnosis is by identification of the larvae in faeces using the Baermann technique (Chapman et al 2004; Conboy 2000; Conboy 2004). Levamisole, ivermectin, fenbendazole and milbendazole (Chapman et al 2004; Conboy 2000) have been used successfully for treatment.

Angiostrongylus cantonensis is a parasite of rats. Larvae of the parasite may infest aberrant hosts including humans. In humans, migration of larvae to the brain may rarely cause serious disease. However, even if infected, most people recover fully without treatment. The larvae do not complete their development in dogs (Wallace & Rosen 1969) The mature (adult) form of the parasite is found only in rodents. Since the natural host are rats and dogs and cats are aberrant hosts in which the larvae are unable to complete their life cycle, this parasite is not considered to be a potential hazard in the commodities.

Ancylostoma brazilliense and *Ancylostoma ceylanicum* are hookworm parasites that are closely related and have a similar life cycle to *Ancylostoma caninum* the universally distributed hookworm of dogs. *Ancylostoma brazilliense* occurs in cats and dogs in tropical and subtropical areas and *Ancylostoma ceylanicum* occurs in some parts of Asia (Zajac & Conboy 2006e). *Ancylostoma brazilliense* is zoonotic and can cause eosinophilic enteritis and cutaneous larva migrans in humans (CDC 2004b). *Ancylostoma* spp. infestations are diagnosed in cats and dogs by floatation methods on faeces (Foreyt 2001b; Zajac & Conboy 2006e) and can be treated with a variety of drugs including oxibendazole, fenbendazole, ivermectin, milbemycin and pyrantel (CDC 2004b; Foreyt 2001b).

Capillaria plica and *Capillaria feliscati*. Adult *Capillaria feliscati* are found in the bladder of cats and *Capillaria plica* in dogs (Bedard et al 2002; Kirkpatrick & Nelson 1987; Senior et al 1980). They are rare parasites and seldom cause clinical signs (Bedard et al 2002; Companion Animal Parasite Council viewed 9/11/2007a). The life cycle is not fully understood but eggs are excreted in urine but do not develop to larvae unless ingested by an earthworm. Cats are infected by eating earthworms or by uptake of larvae in contaminated materials. Larvae reside in the intestinal wall for short period and then migrate to the bladder where they develop into adults (Bedard et al 2002; Companion Animal Parasite Council 2007a). Eggs are excreted in the urine about 2 months after infestation but then the number of eggs excreted declines and ceases over a period of 84 days. Ivermectin is the best drug for treatment (Bedard et al 2002; Kirkpatrick & Nelson 1987; Senior et al 1980).

Cyathospirura seurati (*Cyathospirura dasyurids*), *Cylicospirura heydoni*, *Cylicospirura felineus*, *Cylicospirura subequalis*. *Cylicospirura* spp. are found in fibrous nodules in the stomachs of cats while *Cyathospirura seurati* may occur in nodules or free in the stomach. The parasites are predominantly parasites of dasyurid marsupials (Ladds et al 2006) and feral cats (Coman et al 1981; Gregory & Munday 1976; Milstein & Goldsmid 1997; Ryan 1976) and more rarely dogs and dingoes (Coman 1972) in Australia. Other species of *Cylicospirura* are found in wild cat species such as cougars, bobcats and ocelots in North America (Pence et al 2003b; Rickard & Foreyt 1992; Tiekotter 1985). Reports of the parasites in domestic cats are rare (Junker et al 2006). No reports on treatment were found. *Cylicospirura advena* has been found in New Zealand (Clark 1981). It is concluded that infestations of domestic cats and dogs are rare and of little consequence. Presumably eggs or larvae are shed in the faeces and could be found by examination of faeces by standard methods. There is no reason to believe that the parasites would not be susceptible to anthelmintic drugs.

Dracunculus medinensis (Guinea worm) is primarily a parasite of humans. Occasional infestations occur in other animals including dogs and cats (Bimi et al 2005). The adult parasite is a large worm found in subcutaneous tissue. Female parasites can grow up to 800 mm in length, males do not exceed 40 mm. On maturity a blister forms in the skin that bursts and when the parasite is exposed to water the parasite discharges 1st stage larvae into the water. The larvae are ingested by copepods (*Cyclops* spp.) and the development of the parasite continues in these intermediate hosts. When infected copepods are ingested by humans, the larvae migrate through the lymphatics to the subcutaneous tissues where they develop into adults. The parasite is found in Africa, Asia, and the Middle East but efforts to eradicate the parasite have greatly reduced the numbers of cases in endemically infected countries (Kelly & Pereira 2006). Infestations occur primarily in areas of low rainfall where people use untreated water directly from rivers and pools for drinking, bathing and washing (Anonymous 2007b). Education and improvements in hygiene have led to a great reduction in the numbers of cases in humans (Kelly & Pereira 2006). Although copepods capable of acting as intermediate host do occur in New Zealand (Kiefer 1931-2) the use of safe water and general hygiene as well as the general climatic conditions make the likelihood that the parasite could establish here negligible.
In addition, the likelihood that a dog or cat from an under-developed area where untreated water is used would be imported into New Zealand is negligible.

Dracunculus insignis has a similar life cycle to *Dracunculus medinensis* but occurs in the USA and Canada in a variety of aquatic or semi-aquatic wild carnivores and occasionally in dogs. It does not infest humans. The intermediate hosts are copepods and frogs are often paratenic hosts. Frogs are eaten by the definitive hosts. Larvae do not develop at water temperatures up to 15° C but develop rapidly at 24° C. The adult parasites occur in the subcutaneous tissues of the legs. The females are up to 28 cm in length and the males up to 4 cm. They do not cause mortality in wild carnivores. Dogs usually develop swellings on the legs which form a blister. On contact with water the blister bursts and the worm releases her larvae. Diarrhoea, vomiting, dehydration and asthma may occur in infested dogs (Department of Natural Resources 2007; Fargo 2003). Since the parasites have remained confined to North America it is likely that the New Zealand environment, with its paucity of potential wild animal hosts and its temperate climate, would not be suitable for the host to establish. The likelihood of introduction and establishment is considered to be negligible.

Filaroides hirthi, *Filaroides milksi* and *Filaroides osleri* occur world-wide including New Zealand. *Filaroides hirthi* and *Filaroides milksi* are described as rare infestations and even more rarely cause disease (Zajac & Conboy 2006e). Both species occur in the respiratory system of dogs in which they complete their entire life cycle. Infestation is acquired through ingestion of saliva or regurgitated stomach contents (Pinckney 2004). Larvae are found in faeces and may be demonstrated by the floatation method in zinc sulphate in preference to the Baermann technique (Pinckney 2004). Treatment with ivermectin, thiabendazole, fenbendazole, albendazole or levamisole led to cessation of egg shedding for *Filaroides osleri* but the nodules formed by the worm did not resolve (Pinckney 2004). Since *Filaroides hirthi* and *Filaroides milksi* do not form nodules but are present in the lung tissue, treatment is likely to be effective.

Crenosoma vulpis is a lungworm that causes eosinophilic bronchitis in wild canids, especially foxes. It occurs predominantly in Atlantic Canada and the north eastern USA, but also occurs in Europe and Asia. Gastropods and terrestrial snails are the intermediate hosts. It occurs at low prevalence in dogs. Signs of infestation may include coughing, dyspnoea and exercise intolerance. Diagnosis can be made by examination of faeces by the Baermann or floatation

techniques (Zajac & Conboy 2006e). Milbemycin, fenbendazole, levamisole and ivermectin have been used for treatment. Shedding of larvae ceased after treatment with milbemycin (Bihr & Conboy 1999; Conboy 2004; Shaw et al 1996).

Dioctophyma renale has a wide range of mammalian hosts including dogs, horses, wolves, cheetahs, mink, swine and humans (Ravani 2003). In America, mink are commonly infested (Mech & Tracy 2006). The most common hosts are fish eating mammals. The parasite occurs in Europe the Americas, Africa and Australia, but has not been described in New Zealand. Adult parasites occur in kidney, abdominal cavity or urinary tract. Eggs are passed in the urine and hatch in water. Annelid worms, such as earthworms and leeches, act as intermediate hosts. Fish commonly serve as paratenic hosts with the third stage larvae encysting in the musculature of the fish. Third stage larvae in water may be taken up directly by mammalian hosts. The larvae penetrate the gut and develop in the peritoneum and then enter the kidney (usually the right kidney) where mating takes place and eggs are excreted in the urine. Adult females may reach 1 metre in length but males only reach about 20 cm (Ravani 2003). The infested kidney may be completely destroyed by the growing parasite but the other kidney may compensate sufficiently for the hosts needs and there may be no obvious signs of infestation. The only generally practised treatment is surgical removal of the parasite. The parasite could enter New Zealand in any of its mammalian hosts, including humans, and up to the present time there have been no measures to prevent its entry in IHSs for animals. It is unlikely to establish here as there are few fish-eating carnivores likely to act as definitive hosts and establish a sustainable parasite cycle.

Gnathostoma spinigerum is a parasite of dogs and cats where the adult parasite is found in the stomach wall. Eggs are passed in the faeces and develop into free-swimming 1st stage larvae. *Cyclops* spp. ingest the larvae and act as first intermediate hosts while at least 48 species of vertebrates act as paratenic hosts, including fish, reptiles, birds and mammals. Swamp eels are common hosts and a king cobra was found to be infected with over 1,000 larvae (Rojekittikhun 2002b; Tseng 2003). Humans are commonly infested aberrant hosts that are infested from eating fish and serious disease is caused if the migrating larva infect the brain. When a definitive host ingests infective larvae they penetrate the gut and the life cycle is completed when the larvae moult in the definitive host's tissues and then develop to the adult stage in the stomach wall (Rojekittikhun 2002b; Tseng 2003). The parasite is found in Asia, particularly in Japan and Thailand and has emerged as a problem in Mexico (CDC 2004a). Faecal examination has been used as the diagnostic method for prevalence surveys in dogs and cats (Rojekittikhun 2002a). Diagnosis can be made by faecal examination but eggs cannot always be found. There is little information about treatment in animals but in humans ivermectin and albendazole have been recommended (Anonymous 2004; CDC 2004a; Nontasut et al 2000).

Gurlia paralysans is an obscure parasite of cats. Following its initial description as a parasite found in the veins draining the lumbar region of the spinal chord (Wolfhugel 1934), nothing substantial appears to have been written about it. Eggs have been found in blood of infested animals but no larvae have been described. It was initially suggested that the life cycle could involve insects, lizards and cats but it remains unresolved. It appears to be confined to South America (Chile, Argentine). Since it is a curiosity parasite of little significance, probably confined to South America, it is not regarded as a potential hazard.

Lagochilascaris minor is a parasite that is confined to South America, Mexico and the Caribbean (Olle-Goig et al 1996). The definitive host is unknown. Accidental hosts include humans and cats. In cats, the parasite is found in abscesses which contain adult worms and

eggs (Sakamoto & Cabrera 2002). Up until 1986, 28 cases had been described in humans (Rosemberg et al 1986) and it has been described sporadically since then. Most human cases occur in people in rural areas who hunt and eat rodents. When rodents such as guinea pigs and agoutis are dosed with eggs, the eggs hatch and penetrate the gut and develop into encysted third stage larvae in the muscles. When cats are dosed with eggs, these fail to develop. However, when encysted larvae were fed to cats they developed into adults which were found in abscesses in the tonsillar region or subcutaneous tissues (Campos et al 1992; Paco et al 1999). However, whether cats act as true definitive hosts is not clear.

Lagochilascaris major has a similar life cycle to *Lagochilascaris minor*. Eggs fed to hamsters and vesper mice developed into 3rd stage encysted larvae in muscles and when these were fed to cats they developed into adults in a sac in the semi lunar fold of the palatine tonsil but the sacs did not fistulate. Adult worms were also found in the middle ear (Pena et al 2002). A natural case presenting as a subcutaneous abscess in a dog contained about 100 adult worms (Craig et al 1982). Natural cases also occur in cats. The normal host is unknown but may be the *Didelphis* opossum (Dell'-Port et al 1988). The parasite has been found in the Americas and in lions in Africa. *Lagochilascaris major* parasites have been found in the faeces of experimentally infected cats, but since the worm occurs in abscesses that may not be fistulated this could be an unreliable method of diagnosis. Cats experimentally infected with *Lagochilascaris major* were treated with fenbendazole, which eliminated parasites from the tonsil but did not kill parasites in the middle ear. Various treatments including fenbendazole and ivermectin have been used to successfully treat human cases of *Lagochilascaris minor* infestation (Bento et al 1993; Monteiro et al 2004).

Mammomonogamus spp. Species in this genus are not well defined and there is still confusion about their taxonomy. Information on the species is hard to find and fragmentary, indicating that the organisms are of little importance. The life cycle has not been clearly defined. Cases in cats occur in the Caribbean (Cuadrado et al 1980) but it has not been described in dogs (Bowman 2000). It is possible the cat species *Mammomonogamus ierei* is a synonym of the ruminant species *Mammomonogamus nasicola* and *Mammomonogamus laryngeus* (Bowman 2000). The parasite infests the nares of cats and eggs are found in the faeces (Anderson 2000; Bowman 2000). No signs are reported for infested cats but histologically there is evidence of chronic inflammation of the nasopharynx (Bowman 2000). Humans are accidental hosts and have been treated with mebendazole. *Mammomonogamus auris* is found in the middle ear and has been reported from Japan (Sugiyama et al 1982).

Physaloptera praeputialis, Physaloptera rara (felidis) and *Physaloptera canis* are common parasites of the stomach and duodenum of wild animals, cats and dogs but are considered to be of minor clinical significance (Zajac & Conboy 2006e). They may cause intermittent vomiting in infested animals (Clark 1990; Theisen et al 1998) and mild pathological changes have been described in the stomach of cats (Naem et al 2006).Various species of beetles are intermediate hosts and several reptiles, frogs and other animals may be paratenic hosts (Clark 1990; Theisen et al 1998; Zajac & Conboy 2006e). Eggs are excreted in the faeces but cannot be reliably demonstrated by the usual floatation methods as the eggs sink. Direct smear examination (Clark 1990) or sedimentation methods are used for diagnosis (Zajac & Conboy 2006e). Pyrantel pamoate, fenbendazole and ivermectin have been used successfully for treatment (Theisen et al 1998).

Spirocerca lupi occurs world-wide but mostly in warm climates (Zajac & Conboy 2006e). It has been described in New Zealand but is thought not to have established here (McKenna 1997). Adult parasites are found in granulomas in the stomach, oesophagus and rarely in the

aorta of dogs and wild canids (Bark 2003; Zajac & Conboy 2006e). The intermediate hosts are dung beetles but various vertebrates that eat dung beetles, such as rodents, birds, amphibians and reptiles act as paratenic hosts (Zajac & Conboy 2006e). The parasites may cause thrombi when located in the aorta or other arteries (Gal et al 2005) Aberrant larval migration may cause varying signs and lesions, particularly neurological signs, when aberrant migration of larvae involves the brain (Bark 2003; Du Plessis et al 2007). The eggs may be found in faeces by faecal sedimentation and less reliably by floatation (Zajac & Conboy 2006e). The most successful drugs for treatment are the avermectins, with doramectin demonstrated to be effective (Bark 2003).

Spirura rytipleurites. Varieties of this parasite occur in cats and hedgehogs. Search for information on the parasite in three electronic databases yielded no useful information. The genus was not mentioned in three textbooks on veterinary parasitology. It is concluded that the parasite is a curiosity parasite of no importance.

Strongyloides spp. *Strongyloides planiceps* (*cati*), *Strongyloides felis*, *Strongyloides stercoralis, Strongyloides tumefaciens*. *Strongyloides stercoralis* is a parasite of humans, primates and dogs. The other three species are parasites of cats (Nolan 2001). The parasite is found in the crypts of the small intestine but has an unusual life cycle. Only parthenogenic females which shed larvae into the intestines are found in the definitive host. The larvae hatch in the soil and develop into male and female adults, the females significantly outnumbering the males (Nolan 2001). In some circumstances the larvae in the intestine develop into second and third stage larvae and again penetrate the intestine and establish an auto infectious cycle. After mating, the adults lay eggs in the soil. The eggs hatch and develop into 3rd stage larvae and then penetrate the skin of their hosts and migrate through the blood to the intestines to complete their life cycle (Nolan 2001). Infestation can be subclinical but respiratory infestation may develop with heavy infestations of migrating larvae, and enteritis may be associated with adult worm infestations (Zajac & Conboy 2006e). Infestation can be diagnosed by examination of faeces for larvae either by floatation in zinc sulphate solution (but not salt solution) or by the Baermann technique (Nolan 2001; Zajac & Conboy 2006e). Anthelmintic treatment with albendazole, thiabendazole, fenbendazole or ivermectin will remove adult parasites but will not kill migrating larvae. Follow-up examination is necessary to determine whether larvae have survived and developed into adults (Nolan 2001).

Thelazia callipaeda and *Thelazia californiensis* are parasitic worms found in the eyes of various animals including cats and dogs (Oranto et al 2003) and humans. The parasites occur in the ocular secretions and are seen on the surface of the eye or under the nictitating membrane. Infestations are usually subclinical but irritation of the eye may occur. Diagnosis is by visual inspection of the eye. Several muscid flies have been implicated as vectors (Soulsby 1969b; Zajac & Conboy 2006e), but one study indicates that *Musca domestica* is not a vector (Oranto et al 2005). Topical applications of local anaesthetics or insecticides or systemic treatment with ivermectin have been used (Nash Viewed 9/11/2007a), but the parasite is not economically important and often not diagnosed or left untreated. Spot-on treatment with imidacloprid 10 % and moxidectin 2.5 % has also been successful (Bianciardia & Otrantob 2004).

Toxocara malaysiensis is found in cats and is similar to *Toxocara canis* with which it is easily confused (Gibbons et al 2001). *Toxocara mystax* also occur in cats. The life cycle of some *Toxocara* spp. may involve a somatic cycle in which larvae resident in organs can be reactivated during pregnancy and infest the foetuses. The somatic cycle occurs more commonly in adult hosts. Alternatively, larvae can migrate through the lungs and hence via

the trachea to the stomach and intestines where they develop into adults and produce eggs (tracheal life cycle) which is the main cycle in young animals (Soulsby 1969c). Various vertebrates (mice) and invertebrates (earthworms and cockroaches) may act as paratenic hosts. However, prenatal infestation of kittens does not occur with *Toxocara mystax*.

Toxocara cati and *canis* occur commonly in New Zealand and cause potentially serious illness in humans (particularly children). Other *Toxocara* spp. would compete for the same environmental niche. Diagnosis of *Toxocara* spp. can be made by demonstration of eggs in faeces by floatation methods (Zajac & Conboy 2006e). However, if somatic larvae are present in animals with no adult parasites in the gut faeces examination will not be useful. Several of the common anthelmintics such as levamisole, thiabendazole and ivermectin can be used for the treatment (Abo-Shehada & Herbert 1984; Carrillo & Barriga 1987; McTier et al 2000; Payne & Ridley 1999; Schnieder et al 1996).
Trichuris felis (*Trichuris serrata*, *Trichuris campanula*). Information on this parasite in three electronic databases is fragmentary and sparse. Most information is on the closely related species *Trichuris vulpis* of dogs. Information on the internet that is not in scientific journals or scientific format indicates that it is an uncommon parasite of little clinical significance (Companion Animal Parasite Council viewed 9/11/2007b; Nash viewed 9/11/2007b). The life cycle of *Trichuris* spp. is direct and diagnosis can be made by examination of faeces by floatation methods (Zajac & Conboy 2006e). Treatment of *Trichuris vulpis* includes dichlorovos, fenbendazole, mebendazole and ivermectin (Foreyt 2001b).

Acanthocephalans

Macracanthorhynchus ingens. The natural host of this parasite is the raccoon (Richardson & Barger 2005) and it is a rare parasite of dogs (Pearce et al 2001), coyotes, other animals and humans (Dingley & Beaver 1985). The intermediate host is a millipede (Pearce et al 2001) and other animals may be paratenic hosts. The parasite is rare in dogs which are not its natural host. The natural host does not occur in New Zealand and the intermediate host is not common here either. For these reasons this parasite is not considered to be a potential hazard in the commodity.

Oncicola canis is a parasite of coyotes in the southern United States (Foster et al 2003; Radomski & Pence 1993). It is a rare intestinal parasite of dogs, and has been described in hog nosed skunks (Neiswenter et al 2006), ocelots, bobcats and mountain lions (Pence et al 2003a). The life cycle is not known but it is suggested in unreferenced information on the internet that it has an arthropod intermediate host or hosts and that armadillos and turkeys may be paratenic hosts, and that it is a rare condition of dogs and is of no clinical importance (Anonymous 2007c; Bates 2004). Encysted larvae have been found in armadillos (Chandler 1946) and in young turkeys (McDougald 2003). The likelihood that this rare parasite would be introduced in the commodity, and find suitable intermediate hosts and paratenic hosts in New Zealand is considered to be negligible.

Oncicola pomatostomi is a common intestinal parasite parasite of feral cats in Australia (Adams 2003; Ryan 1976; Schmidt 1983). Birds are considered to be paratenic hosts (Schmidt 1983). The parasite presumably has a complex life cycle involving insects, birds and feral cats but no reports of the parasite occurring in domestic cats. It is likely that a diagnosis could be made by examination of faeces and that treatment with modern anthelmintics would be effective but no reports to confirm this were located.

32.1.5. Hazard identification conclusion

Information is presented for 36 species of exotic nematodes and three acanthocephalans. Most of the species discussed are of minor importance as parasites of cats or dogs. Humans are not true hosts of any of the parasites described except for *Dracunculus medinensis*, but may be accidentally infested by the larvae of some of the parasites. Humans are dead-end hosts for these parasites which mean they do not complete their life cycle if infested.

Angiostrongylus cantonensis is considered to be of no importance because the cat and dog are accidental hosts in which the larval development is not completed.

It is considered that the likelihood of *Dracunculus medinensis and Dracunculus insignis* being able to establish in New Zealand is negligible.

Gurlia paralysans and *Spirura rytipleurites* are considered to be rare curiosity parasites of minimal clinical significance.

Dioctophyma renale is unlikely to establish in New Zealand and is far more likely to be introduced in humans than in cats or dogs since millions more people enter New Zealand than cats or dogs.

Thelazia spp. are of minimal clinical importance and can be treated by anthelmintics and diagnosed by clinical examination.

Capillaria spp. can only be diagnosed by examination of a urine sample but are susceptible to anthelmintics and are of minor clinical importance.

The remaining relevant parasites can be diagnosed by examination of faeces samples. However, in order to detect eggs and larvae from all relevant parasite species, floatation, sedimentation and Baermann technique (examination for larvae) methods must be used when examining faeces samples.

All species considered can be treated effectively with anthelmintics except *Dioctophyma renale* and *Dracunculus* species.

Angiostrongylus cantonensis, Dracunculus medinensis, Dracunculus insignis, Gurlia paralysans, Spirura rytipleurites and *Dioctophyma renale* are not considered to be potential hazards. All other parasites mentioned in Section 32.1.1 are considered to be potential hazards.

Since nematodes and acanthocephalans in any of their life stages are not excreted in semen, they are not considered to be potential hazards in semen.

32.2. RISK ASSESSMENT

32.2.1. Entry assessment

The parasites considered to be potential hazards could be introduced from countries where they occur by animals carrying the parasites that show no signs of infestation. Therefore the likelihood of entry is considered to be non-negligible.

32.2.2. Exposure assessment

Since dogs and cats are likely to have contact with other dogs and cats after introduction and/or with relevant intermediate or paratenic hosts after introduction, the likelihood of transmission to other cats or dogs and establishment in New Zealand is considered to be non-negligible.

32.2.3. Consequence assessment

The parasites considered in this section are generally not important pathogens. They are likely to be less pathogenic than parasites already established in New Zealand. Therefore the consequences for cat and dog health are likely to be minimal.

Humans may be accidentally infested by the larvae of the following species. Migrating larvae of these species may cause sporadic cases of disease which may be serious when vital organs are affected. Species in which *larva migrans* has been described include *Ancylostoma brazilliense*, *Gnathostoma spinigerum*, *Lagochilascaris minor*, *Lagochilascaris major*, *Mammamonogamus ierei* and *Toxocara malaysiensis*. However, these cases are rare and the larvae do not develop to maturity.

The only wild carnivores that could be infected in New Zealand are feral cats. Therefore there could be no consequences for New Zealand native fauna.

New parasites could be introduced and become established in New Zealand. These parasites could cause rare cases of *larva migrans* in humans and mild signs of disease in cats and dogs. Therefore the consequences of introduction are considered to be non-negligible.

32.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, risk is considered to be non-negligible and nematodes and acanthocephalans are classified as hazards in the commodity. Therefore, risk management measures can be justified.

32.3. RISK MANAGEMENT

32.3.1. Options

The *Code* does not make any recommendations for the management of nematodes and acanthocephalans when importing cats and dogs.

The following factors were considered when drafting options for the effective management of the hazards in the commodity:

- The number of parasites considered is large and could be even greater since no hazard list will be exhaustive. Therefore specific options for each parasite are not practical and general measures designed to cover all parasites are necessary.
- All the relevant parasites except *Capillaria* and *Thelazia* spp. are diagnosed by examination of faeces. To cover all species, faeces should be examined by floatation, sedimentation and larval identification (Baermann method) techniques.

- *Capillaria* spp. can be diagnosed by examination of urine and *Thelazia* spp. can be diagnosed by physical examination of the eyes. However, both *Capillaria* and *Thelazia* are not important parasites.
- Effective anthelmintic treatments are available for all the relevant parasites.

The following options, given in ascending order of stringency are available for effective management of excluding parasites in the commodity:

Option 1.

Treatment with an anthelmintic as recommended by the manufacturer that is efficacious against nematodes (such as ivermectin, fenbendazole, levamizole or pyrantel pamoate) within 7 days of shipment.

Option 2.

Examination of faecal samples, followed by treatment with an efficacious anthelmintic and re-examination of faeces, 7-10 days after treatment, to confirm that parasites have been eliminated. If parasites have not been eliminated, the treatment could be repeated using a different anthelmintic and faeces could be re-examined. The procedure could be repeated as necessary until all parasites have been eliminated. Shipment could be within 7 days of a final negative faeces examination; *or*

Option 3.

As for Option 2 but an additional treatment with anthelmintic 3 days before shipment; *or*

Option 4.

As for options 2 or 3 but both faeces and urine could be tested for parasites and the eyes subjected to a physical examination for *Thelazia* spp.

Option 5.

Animals could be held in quarantine while carrying out the procedures in Options 2, 3 or 4.

33. Trematodes

33.1. HAZARD IDENTIFICATION

33.1.1. Aetiological agents

The following trematode parasites were identified in the preliminary hazard list:

Alaria canis
Alaria alata
Alaria marcianae
Alaria arisaemoides
Alaria nasuae
Ampimerus pseudofelineus (*Ophisthorchis guayaquilensis*)
Apophallus donicus (*venustus*)
Clinorchis sinensis
Echinostoma malayanum
Eurytrema (Concinnum) procyonis
Haplorchis yokogawai
Heterobilharzia americana
Heterophyes heterophyes
Heterophyopsis continua
Metagonimus yokogawai
Metagonimus tatahashi
Metorchis albidus
Metorchis conjunctus
Metorchis orientalis
Nanophyetus (Troglotrema) salmoincola
Ophisthorchis felineus (*tenuicollis*)
Ophisthorchis viverini
Paragonimus westermani
Paragonimus kellicotti
Paragonimus pulmonalis
Paragonimus miyazakii
Paragonimus heterotremus
Paragonimus ohirai
Paragonimus peruvianus
Paragonimus skrjabini
Paragonimus mexicanus
Pharyngostomum cordatum
Platynososum concinnum
Schistosoma japonicum
Troglotrema (Selacotyle) mustelae

33.1.2. OIE list

No species in Section 33.1.1 are listed.

33.1.3. New Zealand's status

All the species listed in 33.1.1 have been identified in the preliminary hazard list as species that do not occur in New Zealand.

33.1.4. Epidemiology

Brief, relevant information on each of the trematode species listed in Section 33.1.1 is given below.

Alaria spp.
Alaria canis
Alaria alata
Alaria marcianae
Alaria arisaemoides
Alaria nasuae

Parasites of the genus *Alaria* have a complex life cycle. Adult *Alaria canis* are found in the intestine of dogs and cats. Eggs are passed in the faeces. Larval development occurs in snails and frogs, rodents, snakes, pigs and several other species act as paratenic hosts (Foreyt 2001a; Milesevic et al 2004; Zajac & Conboy 2006f). Paratenic hosts are ingested by the primary host and the larvae penetrate the stomach wall and travel by various routes to the lungs where they are coughed up and swallowed and develop into adults (Foreyt 2001a; Zajac & Conboy 2006f). Pregnant bitches may pass larvae in their milk or through the placenta to their young (Shoop & Corkum 1987; Zajac & Conboy 2006f) and viable larvae may persist in the tissues of bitches for years (Shoop & Corkum 1987). Humans may be accidental hosts when ingesting poorly cooked frog meat (Fernandes et al 1976; Freeman et al 1976; Kramer et al 1996; Zajac & Conboy 2006f). In rare cases infestations in humans with *Alaria americanum* are fatal (Fernandes et al 1976; Freeman et al 1976).

Infestations are generally non-pathogenic for cats and dogs (Zajac & Conboy 2006f). Diagnosis can be made by faecal sedimentation or floatation techniques (Foreyt 2001a; Zajac & Conboy 2006f) and praziquantel or niclosamide can be used for treatment (Foreyt 2001a). Infestations are common in wild carnivores in many countries (Craig & Craig 2005; Dalimi et al 2006a; Henke et al 2002; Moks et al 2006; Pence et al 2003a; Saeed et al 2006; Segovia et al 2001; Wolfe et al 2001). *Alaria canis* and *Alaria marcianae* occur in America (Henke et al 2002; Pence et al 2003a; Shoop & Corkum 1987) and *Alaria alata* in Europe (Moks et al 2006; Sadighian 1969; Saeed et al 2006; Wolfe et al 2001).

Alaria nasuae was originally described from a coatimundi and has subsequently been described once from dogs in Mexico (Shoop et al 1989). It is therefore a rare parasite of dogs.

Alaria arisaemoides is a parasite of wild canids and occasionally dogs and occurs in northern USA and Canada (Dyer et al 1997; Smith 1978).

Clinorchis sinensis, the Chinese or oriental liver fluke is widely distributed in Asia. It has also been stated that it occurs in all parts of the world where there are Asian immigrants from endemic areas (Rim 2005). An estimated 35 million people are infested globally with about 15 million of these in China (Lun et al 2005). Prevalence rates in some areas may be over 30 % in humans (Yu et al 2003), 26 % in dogs (Wang et al 2006), 70 % in cats and 50 % in pigs (Lin et al 2005). The primary host reservoir animals are humans, dogs, cats, rats, pigs and other mammals. The first intermediate hosts are snails and the second intermediate hosts are

numerous species of fresh water fish (Rim 2005). Infestation in humans is often asymptomatic but it has also been associated with complications including cholangitis, cholecystitis, cholangiohepatitis and cholangiocarcinoma. Infestation in dogs and cats is usually asymptomatic. Diagnosis in dogs and cats can be made by examination of faeces and praziquantel is used for treatment. It is not known whether the parasite would find suitable first intermediate hosts in New Zealand. Even if suitable hosts are present the likelihood of establishment is not high as an unusual combination of circumstances and a minimal mass of organisms are likely to be necessary for the parasite to establish.

Echinostoma malayanum is an intestinal parasite of humans and possibly pigs but no reference could be found of it infesting dogs or cats. It has a life cycle involving snails and fish (Belizario et al 2007).

Apophallus donicus (*venustus*) is an intestinal trematode parasite with a typical life cycle involving cats, dogs, and many wild carnivores. Birds and humans as definitive hosts, snails as first intermediate host and fish as second intermediate host (Anonymous 2007a). Wild fish-eating carnivores in which the parasite occurs include otters, mink and polecats (Shimalov 2001; Shimalov et al 2000). Many species of fish carry the metacercariae and infestation may cause death in heavily infested young fish or deformities of cartilage (Kent & Watral 2004). Other species of *Apophallus* such as *brevis* and *muehling*i have a similar life cycle and can also infect dogs and cats. *Apophallus donicus* occurs in the North America, Europe and particularly Eastern Europe (Kent & Watral 2004; Vanparijs & Thienpont 1973). Parasite eggs can be found in faeces. No information was found concerning treatment but it can be assumed that praziquantel could be used since it is effective against a broad range of trematodes.

Ampimerus pseudofelineus (*Ophisthorchis guayaquilensis*) is a liver fluke with a typical life cycle involving snails and fish. Infestation of cats is rare. The parasite may also infest man and it has been described as a parasite of marsupials in Brazil (Correa-Gomes 1979). Most cases reported in cats involve damage to liver and pancreas but, in unreported cases, asymptomatic infestations are likely to be common. Cases have to be differentiated from other liver flukes of the family Opisthorchiidae. Liver flukes of cats can be treated with praziquantel (Rothuizen 2006). A report to DEFRA on exotic agents of cats and dogs describes the trematode as "rarely reported from dogs and cats anywhere and unlikely to establish in Great Britain because of its complex life cycle" (Bennett 2001).

Eurytrema procyonis is a trematode that has been found in the bile and pancreatic ducts of raccoon, fox, a coyote wolf hybrid and cats (Burrows & Lillis 1960; Wade et al 1989). Its life cycle is typical of trematodes and the land mollusc *Mesodon thyroidus* is the first intermediate host and grasshoppers are also involved in the life cycle (Carney et al 1970). A diagnosis can be made by identification of the eggs in faeces using a sedimentation method.

Haplorchis yokogawai is an intestinal fluke. Five other species of the genus occur in Asian countries in humans and have been found in mammals and birds. *Haplorchis yokogawai* has been reported to be transmitted to dogs and cats with metacercariae from mullet (Chai et al 2005). Although *Haplorchis yokogawai* occurs naturally in cats (Scholtz et al 2003), the parasite occurs at low prevalence in humans compared to other trematodes such as *Opisthorcis viverini, Haplorchis taichui* and *Haplorchis pumilo* (Chai et al 2007). The life cycle is typical with the first intermediate host a snail and the second intermediate host fish (Giboda et al 1991a). Praziquantel is an effective treatment for parasites of this genus (Belizario et al 2004; Giboda et al 1991b).

Heterobilharzia americana is a parasite of the mesenteric and hepatic portal veins of dogs and raccoons in north America. Other wildlife species and humans can be infested but patent infestations do not develop in several other species (Malek 1970b). Humans are an incidental host in which the parasite dies on penetration of the skin leaving an itchy lesion, the condition is known as swimmer's itch or cercarial dermatitis. Various snails of the genus *Lymnea* are intermediate hosts for the parasite, but the parasite does not occur in all areas where competent species of snails are present (Malek 1970a). At least four species of snails belonging to the genus *Lymnea* occur in New Zealand (Winterbourn 1973) but it is not known whether any are competent hosts for the parasite. A diagnosis can be made by examination of faecal samples either by sedimentation in saline or examination of faecal sediment in water which causes hatching of the cercariae which can then be identified (Goff & Ronald 1980). Treatment with praziquantel is effective (Flowers et al 2002).

Heterophyes heterophyes is a small intestinal parasite of humans and other mammals. It has a typical life cycle, with snails and fish as intermediate hosts (CDC 2007d). In most aspects including diagnosis and treatment it is similar to other trematodes of fish eating mammals (CDC 2007d).

Heterophyopsis continua is an intestinal parasite of humans (Guk et al 2006; Park et al 2007; Chai, 2002) and cats (Park et al 2007; Sohn & Chai 2005). It has been reported from Asia, particularly from Korea. The life cycle is similar to that of other trematodes. Humans and animals are infected from metacecariae in fish (Park et al 2007; Sohn et al 2005). Diagnosis and treatment is similar to that for other trematodes.

Metagonimus spp.

Metagonimus yokogawai
Metagonimus tatahashi

These parasites are small flukes found in the gut of humans and animals including dogs and cats. The parasites occur in the Far East, Siberia, Manchuria, the Balkan states, Israel, and Spain (CDC 2007a). Life cycle, diagnosis and treatment are similar to other trematodes (CDC 2007a).

Metorchis spp.

Metorchis albidus
Metorchis conjunctus
Metorchis orientalis

Meotorchis conjunctus is a parasite of the bile ducts and gall bladders of cats, dogs, foxes, mink and raccoons in North America. *Metorchis albidis* is a parasite of the gall bladder and bile ducts of dog, cat, fox and grey seal in Europe and North America (Soulsby 1969d). *Metorchis conjuctus* infects wolves with occasional involvement of the pancreas (Wobeser et al 1983). *Metorchis orientalis* is mainly a parasite of ducks (Zang 2007) but can complete its life cycle in chickens (Sohn et al 1992) and occurs in other animals. *Metorchis conjunctus* has been shown to be pathogenic in cats when sufficient numbers of parasites are involved (Axelson 1962; Watson & Croll 1981). Humans may be accidental hosts. Life cycles involve snails, fish and definitive hosts. Praziquantel was effective for treating ducks (Yang et al 2004) and is recommended for treatment of *Metorchis conjunctus* (American Society of Health System Pharmacists 2005).

Nanophyetus (Troglotrema) salmincola is an intestinal parasite of a wide range of fish eating mammals (Farrell et al 1974). In dogs it causes a relatively harmless infestation but can act as the vector of a fatal disease of dogs caused by *Neorickettsia helminthoeca* (Foreyt et al 1987). Humans are not susceptible to the rickettsial infection but may show symptoms resulting from infestation with the parasite (Fritsche et al 1989). The life cycle is typical with snails and fish, particularly salmonids, as first and second intermediate hosts. Diagnosis can be confirmed by sedimentation examination of faeces (Zajac & Conboy 2006g). Praziquantel can be used for treatment (Fritsche et al 1989).

Ophisthorchis spp.

Ophisthorchis felineus (*tenuicollis*)
Ophisthorchis viverini

Opisthorchis spp. are liver flukes. *Opisthorchis viverrini* is found mainly in Southeast Asia especially northeast Thailand, Laos, and Cambodia. *Opisthoruis felineus* is found in Europe, Asia, and the former Soviet Union. The definitive hosts are humans, cat, dog, civet cat and other fish-eating mammals (Rim 1982). Their life cycle, diagnosis and treatment are similar to those for other trematodes (CDC 2007b).

Paragonimus spp.

Paragonimus westermani
Paragonimus kellicotti
Paragonimus pulmonalis
Paragonimus miyazakii
Paragonimus heterotremus
Paragonimus ohirai
Paragonimus peruvianus
Paragonimus skrjabini
Paragonimus mexicanus

Paragonimus spp. are lung flukes of humans and animals including dogs and cats. Their life cycle is complex and typical of trematodes, the first intermediate host is a snail and the second intermediate hosts are crustaceans such as crabs, crayfish etc (Cambridge University Schistosome Research Group 1998; CDC 2007c). *Paragonimus westermanii* is the most important species in humans. *Paragonimus kellicotti* is found in cats in the USA. It may cause a subclinical infestation or an eosinophillic bronchitis and granulomatous pneumonia (Zajac & Conboy 2006g). Yokogawa listed 45 species of *Paragonimus* spp. of which at least 12 were known to infest humans and at least eight are known to infest cats and dogs (Yokogawa 1982). Most *Paragonimus* spp. are found in Asia but some species occur in America and Africa (Miyazaki 1982). It is possible that additional species have been found in humans and animals and the host parasite lists expanded since the writing of Yokogawa's review. The methods of diagnosis rely on faecal examination and infestations can be treated with praziquantel.

Pharyngostomum cordatum is an intestinal parasite of cats which are a definitive host. The first intermediate host is a snail, with frogs as the second intermediate host and frog-eating animals particularly grass snakes, as paratenic hosts (Chai et al 1990). The parasite has been described in cats and more rarely dogs in Asia and Russia (Cho & Lee 1981; Dubey 1970; Kajiyama et al 1980; Sohn & Chai 2005; Sudarikov et al 1991; Tanaka et al 1985). Rodents may be experimentally infested with metacecariae but they fail to develop in these hosts and migrate to extra-intestinal sites (Chai et al 1990; Shin et al 2001). Diagnosis can be made by

examination of faeces (To et al 1988; Zajac & Conboy 2006g) and praziquantel has been used for treatment (Fukase et al 1986).

Platynosonum concinnum is a liver fluke of cats. The intermediate host is a land snail *Subulina octona* that occurs only in tropical and subtropical climates (Haney et al 2006). Infestation with small numbers of parasites caused asymptomatic infestations and a greater infection dose caused mild signs of inappetance (Taylor & Perri 1977). Occasional cases of severe disease occur and when severe damage to the bile ducts has been caused treatment is likely to be unsuccessful (Haney et al 2006). Diagnosis can be made by sedimentation examination of faeces (Zajac & Conboy 2006h) and treatment is with praziquantel.

Schistosoma japonicum is a parasite that is found in the mesenteric blood vessels of its host. The life cycle involves a snail host from which the cercariae are released, the cercariae penetrate the skin of their host animal and migrate to the mesenteric vessels where male and female parasites mate and eggs penetrate the mesenteric vessels and are excreted in the faeces (CDC 2007e). The parasite is found in Asian countries and causes a serious disease in humans. Related parasites *Schistosoma mansoni* and *Schistosoma haematobium* occur in Africa, the Middle East and in the Carribbean. Dogs and cats and other animals are maintenance hosts for *Schistosoma japonicum* (Fernandez et al 2007). Infestation can be diagnosed by sedimentation examination of faeces but the number of eggs produced by the parasites declines as infestation progresses (Zajac & Conboy 2006i). Praziquantel is used for treatment (CDC 2007e).

Troglotrema (*Selacotyle*) *mustelae* is a minute intestinal parasite of mink and other mustelids that may occur in cats (Wallace 1935). References to it are rare, dated and difficult to locate which indicates that it is probably of minimal significance.

The list of trematodes given in the aetiological agents section is not comprehensive. Detailed study would probably reveal other parasites that can occur in cats and dogs. Indeed the list is so long that it would be impractical to consider them all individually. However, the following important factors should be noted:

i) All trematodes have similar complex life cycles. Most require at least one first intermediate host and one second intermediate host. Paratenic accumulator hosts are also important in the life cycles of many of the parasites. Fulfilling the requirements of these complex life cycles makes it unlikely that introducing single or small numbers of infested cats or dogs would lead to the establishment of the parasites.
ii) Diagnosis of infestations in live animals is always dependent on carrying out faecal examinations. For most parasites sedimentation techniques are used rather than floatation methods.
iii) Treatment with praziquantel is effective for virtually all trematodes.
iv) Many of the parasites considered above only cause mild or subclinical infestations except under abnormal conditions such as when overwhelming numbers of parasites infest the final hosts.
v) Humans are accidental or definitive hosts of many of the trematode parasites discussed above.

Therefore, it is possible to design efficient systems for diagnosis and treatment of all trematodes. Trematodes should be considered as a single group when devising strategies for preventing their introduction.

33.1.5. Hazard identification conclusion

Trematodes should be considered as a single group. Since at least some trematodes occur in all countries they are considered to be potential hazards in the commodity.

Trematodes are not excreted in semen during any part of their life cycle. Therefore they are not considered to be potential hazards in semen.

33.2. RISK ASSESSMENT

33.2.1. Entry assessment

Trematodes could be introduced from countries where they occur by dogs and cats that show no signs of infestation. Therefore the likelihood of entry is considered to be non-negligible.

33.2.2. Exposure assessment

Dogs and cats will not be directly contagious to other dogs and cats but could infest intermediate hosts. It is not known whether competent intermediate and paratenic hosts exist for exotic trematodes. The likelihood that imported dogs and cats could infest suitable intermediate hosts should be considered non-negligible.

33.2.3. Consequence assessment

The parasites considered in this section are generally not important pathogens. They are likely to be less pathogenic than parasites already established in New Zealand. An exception could be infestation of dogs with *Nanophytes salminicola*, which in itself would be relatively harmless but could act as a vector for the rickettsial disease caused by *Neorickettsia helminthoeca*. Feral cats and mustelids could be infested with some of the parasites. However, since these are likely to be of little clinical significance and mustelids are generally considered to be pests, the consequences are considered to be negligible.

Humans may be accidental or definitive hosts of some of the parasites and a few of these such as *Bilhazia japonicum*, cause significant health problems. Humans could be involved as accidental hosts in which migrating larvae could damage vital organs.

Since human health could be affected and there may be minor consequences for dogs, the consequences of introducing trematodes are considered to be non-negligible.

33.2.4. Risk estimation

Since entry, exposure and consequence assessments are all non-negligible, the risk is considered to be non-negligible. Therefore trematodes are classified as hazards in the commodity and risk management measures can be justified.

33.3. RISK MANAGEMENT

33.3.1. Options

The following points should be considered when drafting options to manage the risks associated with the introduction of trematodes in the commodity:

- Diagnosis of infestations in live animals is always dependent on carrying out faecal examinations. For most trematodes sedimentation techniques are used rather than floatation methods but to cover all cases both sedimentation and floatation methods should be used.
- Treatment with praziquantel is effective for virtually all trematodes, effective dosage regimens should be used.
- Many of the parasites considered above only cause mild or subclinical infestations in dogs and cats.
- The life cycles for many trematodes are complex and it is unlikely that the parasites would become established as a viable self-sustaining population when single or small numbers of cats or dogs are imported.

There is no *Code* chapter on trematodes of cats and dogs.

The following options, given in ascending order of stringency, are available to effectively manage the risk of introducing trematode parasites in the commodity:

Option 1.

Since the parasites are unlikely to establish or cause serious diseases, cats and dogs could be imported without restrictions.

Option 2.

Since praziquantel is an effective agent for the treatment of trematode infestations, all cats and dogs to be imported could be treated with an effective regime of praziquantel treatment.

Option 3.

i) cats and dogs could be treated with an effective dose of praziquantel, 3 weeks before shipment; and
ii) 1 week after treatment a faecal sample could be examined by both sedimentation and floatation methods by a laboratory approved by the veterinary authority of the exporting country, with negative results. Should trematode eggs be found, treatment could be repeated until a negative result is obtained.

Option 4.

Dogs for export could be held in quarantine while the recommendations of Option 3 are carried out.

34. Cestodes

34.1. HAZARD IDENTIFICATION

34.1.1. Aetiological agents

The following parasites are those identified in the preliminary hazard list:

Anoplotaenia dasyuri
Diphyllobothrium latum
Dipylidium spp.
Echinococcus spp.
- *Echinococcus granulosus*
- *Echinococcus vogeli*
- *Echinococcus oligarthus*

Joyeuxiella spp.
- *Joyeuxiella pasqualei*
- *Joyeuxiella echinorhynchoides*
- *Joyeuxiella fuhrmanni*

Mesocestoides lineatus (*variabilis*)
Spirometra spp.
- *Spirometra erinacei* (*Spirometra mansoni*)
- *Spirometra mansonoides*

Taenia spp.
- *Taenia crassiceps*
- *Taenia krabbei*

34.1.2. OIE list

Echinococcosis/hydatidosis is listed in the *Code*. No other parasite in Section 38.1.1 is listed.

34.1.3. New Zealand's status

None of the parasites listed in Section 38.1.1 are known to occur in New Zealand. *Echinococcus* spp. are notifiable organisms (Ministry of Agriculture & Forestry 2008). None of the other species listed in Section 38.1.1 are notifiable or unwanted organisms. *Dipylidium caninum* is an endemic species but other species in the genus may be exotic. *Spirometra erinacei* has recently been reported (Urgarte et al 2005) but other members of the genus are not known to occur in New Zealand.

34.1.4. Epidemiology

Anoplotaenia dasyuri is a tapeworm of Australian dasyurids (carnivorous marsupials), particularly Tasmanian devils and tiger cats (Beveridge & Jones 2002; Beveridge et al 1975; Gregory & Munday 1975). The intermediate hosts are marsupials. Natural infestations with metacestodes (tapeworm cysts) were found in pademelons (*Thylogale billiardieri*), potoroos (*Potorous apicalis*), Bennett's wallabies (*Macropus rufogriseus*) possums (*Trichosurus*

vulpecula) and kangaroos (*Macropus giganteus* and *Macropus fuliginosus*). Mice and guinea pigs were experimentally infested (Beveridge & Jones 2002; Beveridge et al 1975). Parasites were found in feral cats and rural dogs but metacestodes from wallabies failed to develop when fed to dogs but did develop in Tasmanian devils and tiger cats (Gregory & Munday 1975). No reports could be found that indicates a dog/marsupial or cat/marsupial cycle operates in Australia. The parasite has not been described outside of Australia, but because possums occur commonly in New Zealand the likelihood that it could establish in a dog/possum or cat/possum cycle, although unlikely, is non-negligible. It could presumably be diagnosed in dogs and cats by examination of faeces and treated with praziquantel.

Diphyllobothrium latum.
Information on this parasite has been obtained from three parasitology texts (Foreyt 1997a; Taylor et al 2007a; Zajac & Conboy 2006a). The tapeworm is known as the broad fish tapeworm. Final hosts are dog, cat, human and fish eating mammals. However, it is essentially a parasite of humans since it produces few fertile eggs in other species. Copepods act as first intermediate hosts and fish are second intermediate hosts. Since some species of copepods and fish occur in New Zealand establishment of the parasite may be possible. Infestation is contracted by a final host eating uncooked or unfrozen fish. It can be diagnosed by examination of faeces by visual inspection for tapeworm segments and by faecal floatation methods for eggs. *Diphyllobothrium latum* is a parasite of the Northern Hemisphere and South America. Praziquantel and nicosamide are effective for treatment of adult parasites.

Dipylidium spp.
The only important member of this genus is *Dipylidium caninum* and it is present in New Zealand. Other members of the genus are rarely mentioned in the scientific literature and are of no practical importance. Infestations can be diagnosed by faecal examination and effectively treated by praziquantel. Several named species may be synonyms for *Dipylidium caninum* (Soulsby 1969a).

Echinococcus spp.
Echinococcus granulosus
Echinococcus vogeli
Echinococcus oligarthus

Echinococcus granulosus occurs world-wide except Iceland and Eire (Taylor et al 2007c) and has been eradicated from New Zealand (Pharo 2002). The final hosts are dogs and related carnivores and the most important intermediate hosts are sheep. Cattle are less efficient intermediate hosts and kangaroos and other marsupials are also intermediate hosts in Australia (Jenkins & Morris 2003). Hydatid cysts are found in the liver and lungs of intermediate hosts. Humans can be infested with hydatid cysts as accidental, dead-end hosts, resulting in serious disease and sometimes death. Diagnosis in dogs is by faecal floatation to identify eggs or visual inspection of faeces to identify tapeworm segments (Taylor et al 2007c; Zajac & Conboy 2006b). However, diagnosis is difficult since tapeworm segments are small and only shed sparsely. Tests are available to identify the presence in faeces of *Echinococcus* antigen by ELISA and DNA by PCR (Craig 2004). Treatment with praziquantel is highly effective (Foreyt 1997b; Taylor et al 2007c).

Echinococcus multilocularis is primarily a parasite of foxes but also affects dogs. It occurs in Europe and many northern hemisphere countries (Hegglin et al 2003; Taylor et al 2007b). Its life cycle, diagnosis and treatment are similar to *Echinococcus granulosus*.

Echinococcus vogeli occurs in Central and South America where it infects bush dogs (*Speothus venaticus*) and more rarely dogs. The intermediate hosts are pacas (*Agouti paca*) or agoutis (*Dasyprocta aguti*) (Rodrigues-Silva et al 2002; Taylor et al 2007c). The geographic distribution is limited to Central and South America where suitable primary and secondary hosts are present.

Echinococcus oligarthus is a parasite of cougars, ocelots, jaguars and other wild felids. The intermediate hosts include the paca, agouti and spiny rat (D'Alessandro et al 1981). The geographic distribution is limited to Central and South America (D'Alessandro et al 1981). The likelihood that it would be introduced and establish in New Zealand is negligible.

Joyeuxiella

Joyeuxiella pasqualei
Joyeuxiella echinorhynchoides
Joyeuxiella fuhrmanni

There are three relevant species in the genus *Joyeuxiella* (Jones 1983). They infest dogs and cats. Their life cycle involves an unknown first intermediate host and a reptile as second intermediate host (Soulsby 1969a). Since skinks occur commonly in New Zealand and are frequently hunted by cats, the likelihood that the parasite could establish is non-negligible. *Joyeuxiella* spp. are widely distributed geographically and occur in Mediterranean countries such as Spain, Greece, Turkey (Calvete et al 1998; Haralabidis et al 1988; Millan & Casanova 2007; Papdopoulos et al 1997; Yaman et al 2006), Middle Eastern countries such as Jordan and Iran (Dalimi et al 2006b; El-Shehabi et al 1999; Mohammad et al 2007), South Africa (Minnaar & Krecek 2001) Australia (Biosecurity Australia 2001) and probably many other countries. Treatment with praziquantel is effective.

Mesocestoides lineatus (variabilis)

Mesocestoides lineatus is widely distributed in Europe, Africa and Asia (Taylor et al 2007d). Its primary hosts are dogs, cats, foxes, mink and wild carnivores. The first intermediate hosts are *Orabatid* mites and the second intermediate hosts may be amphibians, reptiles, murines, nonhuman primates, birds, and mammals such as rodents, dogs and cats (Toplu et al 2006) Potential intermediate hosts occur in New Zealand. Infestation of dogs and cats is usually subclinical. Humans are occasionally accidentally infested with intermediate stages of the parasite (Toplu et al 2006). Diagnosis in dogs and cats infested with adult parasites is by faeces examination for eggs, by faecal floatation, or visual examination for tapeworm segments (Foreyt 1997c; Zajac & Conboy 2006c). Praziquantel is used for treatment (Foreyt 1997c).

Spirometra spp.

Spirometra erinacei (*mansoni*, *erinaceieuropaei*)
Spirometra mansonoides

Spirometra spp. are parasites of cats, dogs and wild carnivores and occasionally humans (Taylor et al 2007e; Zajac & Conboy 2006d). *Spirometra erinacei/erinaceieuropaei* has been described in New Zealand in a feral cat (Urgarte et al 2005). Therefore, *Spirometra erinacei* is endemic but *Spirometra mansonoides* may be an exotic species. The first intermediate host is a copepod or crustacean and the second intermediate host may be a wide variety of species including frogs and snakes (Taylor et al 2007e; Zajac & Conboy 2006d). Potential intermediate hosts are present in New Zealand. Infestation of humans with the larval stages is rare but results in a condition known as sparganosis which may be fatal and requires surgical intervention (Sparganum was the old name for the plerocercoids of *Spirometra* spp.). Humans

can be infected by drinking water contaminated with infested copepods. The course of experimental infestations in humans has been described (Mueller & Coulston 1941). Diagnosis in cats and dogs is by identification of tapeworm segments or identification of eggs in faeces by sedimentation or floatation techniques (Zajac & Conboy 2006d). Praziquantel is suggested for treatment (Eom et al 1988; Foreyt 1997d), but it has been suggested that bunamidine is the drug of choice (Georgi 1987).

Taenia spp.
Taenia crassiceps
Taenia krabbei (*cervi*)

Taenia crassiceps is a parasite of dogs and foxes, with small rodents serving as intermediate hosts. *Taenia krabbei* is a parasite of dogs, with reindeer as the intermediate host. *Taenia cervi* is a parasite of dogs and foxes and other wild canids, with red deer and roe deer as intermediate hosts (Taylor et al 2007e). However, *Taenia krabbei* and *Taenia cervi* may be synonyms. The three parasites are widely distributed in the world, but have not been described in New Zealand (McKenna 1997). Potential intermediate hosts such as mice (*Taenia crassiceps*) and deer (*Taenia krabbei)* occur in New Zealand. Infestations with the cysts of *Taenia crassiceps* have been described in immunosuppressed humans (Heldwein et al 2006) and a cat (Wunschmann et al 2003). Diagnosis is by visual examination of faeces for tapeworm segments and for eggs (Foreyt 1997e; Foreyt 1997f; Taylor et al 2007e). Praziquantel and several other drugs can be used for treatment (Foreyt 1997e; Foreyt 1997f; Taylor et al 2007e).

34.1.5. Hazard identification conclusion

Since the parasites have not been recorded in New Zealand and several are considered to be health hazards to humans, dogs and cats, they are considered to be potential hazards in the commodity.

Since cestodes in any of their life stages are not excreted in semen, they are not considered to be potential hazards in semen.

34.2. RISK ASSESSMENT

34.2.1. Entry assessment

Since signs of infestation are seldom obvious, dogs coming from countries where any of the parasites occur could be carrying adult tapeworms and the risk of entry is non-negligible.

34.2.2. Exposure assessment

Known competent or potential intermediate hosts for all the parasites described in this section occur in New Zealand. Therefore, the likelihood of exposure of intermediate hosts and subsequent establishment of the parasite is non-negligible.

34.2.3. Consequence assessment

The consequences for cats and dogs are likely to be minor since infestation with adult tapeworms is generally of minimal clinical significance. However, since several of the parasites may accidentally infest humans, with sometimes serious or even fatal consequences (hydatidosis and sparganosis), the consequences are considered to be non-negligible.

34.2.4. Risk estimation

Since entry, exposure and consequence assessments are non-negligible, risk is considered to be non-negligible and cestodes are classified as hazards in the commodity. Therefore, risk management measures can be justified.

34.3. RISK MANAGEMENT

34.3.1. Options

The following factors were considered when drafting options for the efficient management of the hazards in the commodity:

- Cestodes all have similar complex life cycles.
- Praziquantel is effective for virtually all tapeworm infestations.
- A diagnosis of tapeworm infestation can be made by examination of faeces. For this purpose visual inspection of faeces for tapeworm segments and faecal sedimentation and floatation for eggs are necessary. Alternatively and with greater sensitivity, detection of antigen or parasite DNA may be used when tests are available for the specific parasite species.

The *Code* chapter for echinococcosis/hydatidosis makes the following recommendations for importation of dogs, cats and other domestic or wild carnivores:

Article 2.2.3.2.

Veterinary Authorities of *importing countries* should require:

for dogs, cats and other domestic or wild carnivores

the presentation of an *international veterinary certificate* attesting that the animals were treated against echinococcosis/hydatidosis prior to shipment, and that the treatment used is recognised as being effective.

There are no *Code* recommendations for any of the other cestodes covered in this risk analysis.

The following options, in ascending order of stringency are available for the effective management of cestodes in the commodity.

Option 1.

Treatment with an effective dose of praziquantel within the 7 days prior to shipment.

Option 2.

Examination of faeces within the 14 days prior to shipment by a laboratory approved by the veterinary authority of the exporting country using both sedimentation and floatation methods and examination of faeces for tapeworm segments, with negative results.

Option 3.

Cats and dogs could be treated with an effective dose of praziquantel 3 weeks before shipment; and

1 week after treatment a faecal sample could be examined by both sedimentation and floatation methods, and examination of faeces for tapeworm segments, by a laboratory approved by the veterinary authority of the exporting country, with negative results. Should

evidence of cestode infestation be found, treatment could be repeated until a negative test result is obtained. During the pre-export period of treatment and testing dogs should not be fed ruminant offal.

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VIRUS FAMILIES SECTION

35. Bornaviridae

35.1. HAZARD IDENTIFICATION

35.1.1. Aetiological agent

Borna disease virus is an RNA virus and is the sole member of the family Bornaviridae.

35.1.2. OIE List

Not listed.

35.1.3. New Zealand's status

Exotic and unwanted organism (Ministry of Agriculture & Forestry 2008).

35.1.4. Epidemiology

Classical Borna disease virus (BDV) encephalomyelitis, known as Borna disease (BD) in horses, cattle and sheep, is restricted to endemic regions in Germany, Switzerland and Austria (Staeheli 2000). A range of other animals from birds to primates, including cats, dogs and possibly humans can be infected. The definitive host for BDV has not been identified, but rodents and birds are suspected (Greene 2006).

Serological studies indicate that cats may have been subclinically infected in Britain and continental Europe, the Philippines, Indonesia, Iran, Turkey and Japan (Greene 2006; Reeves, 1999). Two dogs have been reported as having BD, one in Austria (Weissenbock 1998) and the other in Japan (Okamoto 2002).

Antibody to Borna disease virus has been found in humans suffering from psychiatric disorders. However, the significance of the virus in human infections and as a cause of psychiatric disorders remains controversial (Carbone 2001).

Virus is excreted in nasal secretions, saliva and conjunctiva of infected horses and sheep. Natural transmission is presumed to occur by direct contact with contaminated fomites, including food, which leads to inhalation and ingestion of the agent (Rott 2004). In recent studies however, all attempts to demonstrate infectivity in secretions of horses have failed (Staeheli 2000). There is no clear evidence that transmission from horse to horse occurs.

Infection does not appear to spread between cats either and there are no reports of vertical transmission occurring in any species (Staheli 2000). Susceptibility in cats appears extremely low as, experimentally, infection of cats is difficult and requires intracerebral inoculation (Lundgren 1997).

The virus is highly neurotropic, similar to rabies virus, and reaches the central nervous system (CNS) by intraaxonal transport. Injecting virus into the feet of neurectomized rats fails to lead to infection as virus is prevented from reaching the CNS (Carbone 1987). Intravenous injection of rats also failed to infect them, reinforcing the exclusiveness of the neural pathway. Experimentally the disease has been transmitted from infected rats and mice to naïve rats and mice through the olfactory route (Carbone 1987). This lends support to the theory that rodents may be the reservoir hosts of BDV and that the olfactory nerves carry the virus to the brain. However, overall the transmission route(s) of BDV remain largely unknown (Kamhieh 2006).

Despite the fact that the disease has been known for more than 250 years, there is controversy regarding diagnosis and relative significance of BDV in animals (Staeheli 2000).
In naturally occurring feline BD most infections appear to be subclinical since seropositive cats are usually clinically normal. However, in those cats that do develop nervous disease (ataxia, change in mental state, seizures) the usual outcome is death within 1 to 4 weeks. The few surviving cats are usually permanently affected with motor dysfunction, personality changes or both (Greene 2006). Borna disease reported in the two dogs has been characterized by a rapid onset of progressive CNS deficits similar to rabies and canine distemper (Weissenbock 1998).

Although antibodies have been detected in cats with a wide variety of clinical signs, the signs may not have been caused directly by BDV (Greene 2006). BDV can be pathogenic in cats, but the presence of BDV is not sufficient to confirm that clinical disease is a result of BDV infection as many animals may have subclinical infections. Histological examination of the brains of 180 cats with clinical signs suggestive of BD in Switzerland revealed changes typical of BD in all cases. However the presence of virus could be demonstrated by immunohistochemistry in a single case only (Staeheli 2000; Greene 2006). A paper which reviews the literature on ‘staggering disease’ in cats concludes that the virus is probably not the aetiological agent responsible for the clinical signs observed (Staeheli 2000). Therefore the aetiology of ‘staggering disease’ in cats is still unresolved.

The specificity of demonstrated antibody and the accuracy and reliability of the RT-PCR test to demonstrate the presence of viral RNA has been questioned (Staeheli 2000; Carbone 2001). Although viral RNA has been demonstrated in an increasing number of countries and animal species, the occurrence of the disease is still mainly confined to parts of Germany and surrounding countries. Since studies using RT-PCR have not generally been confirmed by viral isolation, it is not known whether closely related viruses occur and what role they might play in causing disease and stimulating antibody production.

Detection in the CNS of BDV antigen by immunohistochemistry, of BDV RNA by *in situ* hybridization, or both in combination with neurohistopathological alterations is considered the most reliable method of confirming active CNS classical Borna disease (Greene 2006). The sensitivity and specificity of serological assays varies considerably between Bornavirus laboratories. A reason for this is that titres are usually very low (1:5 to 1:320) as the immune response to viral antigens is weak and these antibodies may have been induced by infection

with an antigenetically related agent of unknown identity or exposure to some other related immunogen (Staeheli 2000).

No evidence was found that suggests that BDV is excreted in semen.

35.1.5. Hazard identification conclusion

The currently available diagnostic tests for BDV are not well suited to diagnosing *intra vitam* (during life) infections in animals or humans. The epidemiology of BDV remains unclear and several key questions, including whether it causes psychiatric disease in humans and the extent of its distribution worldwide are controversial.

Cats and dogs can be infected with BDV. Therefore it is concluded to be a potential hazard.

35.2. RISK ASSESSMENT

35.2.1. Entry assessment

BD is a rare disease primarily affecting horses and sheep in recognised endemic regions of Europe (Kolodziejek 2005). It is extremely rarely reported in cats and dogs and is difficult to diagnose, with a largely unknown epidemiology and distribution. No evidence was found to suggest that BDV is excreted in semen.

Single cases of BDV in cats have been reported from Belgium (Bosschere 2004), Switzerland (Staeheli 2000; Greene 2006) and Japan (Staeheli 2000). In Sweden a large wild cat (*Lynx lynx*) was diagnosed with classic BD (Degiorgis 2000). BDV in dogs is limited to two reports, one dog in Austria and the other in Japan (Weissenbock 1998; Okamoto 2002).

Serological evidence of BDV has been found in cats from Europe, Philippines, Indonesia, Iran, Turkey and Japan. Serology remains controversial since seropositivity does not necessarily mean the animal is carrying the virus. Although BDV infection has been reported in cats with ataxia and other neurological signs in the UK and Japan, a direct aetiologic role has not been established in these cases (Greene 2006).

Since BDV is extremely rarely reported in cats and dogs, and death results fairly quickly if affected, it is unlikely they are reservoir hosts. It is more likely that they are incidental hosts and are probably not important in the epidemiology of BD.

The likelihood of importing an infected cat or dog is remote therefore entry is assessed to be negligible.

35.2.2. Risk estimation

Since entry is assessed to be negligible, the risk of importing cats or dogs infected with BDV is estimated to be negligible. BDV is therefore not classified as a hazard in the commodity and risk management measures are not justified.

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36. Bunyaviridae

36.1. HAZARD IDENTIFICATION

36.1.1. Aetiological agent

The family Bunyaviridae includes five genera, of which three *Phlebovirus*, *Hantavirus*, and *Bunyavirus* contain species that infect cats and dogs.

36.1.2. OIE List

Rift Valley fever is included within the category of 'multiple species diseases'.

36.1.3. New Zealand's status

Bunyaviridae spp. are exotic and unwanted organisms (Ministry of Agriculture & Forestry 2008).

36.1.4. Epidemiology

Rift Valley fever (RVF)
Rift Valley fever virus (RVFV) is a mosquito-borne zoonotic virus within the genus *Phlebovirus*. Primarily it is a disease of sheep, cattle and humans (Walker 1970a).

In sheep, RVF causes abortion storms and deaths in neonatal lambs. In typical outbreaks in southern Africa mortality rates of 5-30 % and abortion rates of 40-90 % have been reported (Swanepoel 2004). In cattle, disease is less severe and goats are even more resistant again. RVF has been recognised in African and Middle Eastern countries. Epidemics occur in seasons associated with abnormally heavy rainfall and the expansion of the breeding sites of vector mosquitoes. Typically the disease is not seen in the years between epidemics. The virus has been isolated from 20 species of mosquito and 14 of them studied in the laboratory were capable of transmitting infection to domestic animals (Acha 1987). The virus is thought to be maintained through inter-epidemic periods by transovarial transmission in drought resistant eggs of certain mosquito species that can survive several years without hatching (Radostits 2007).

The incubation period varies from 12-36 hours (Swanepoel 2004). The disease usually follows an acute course in adult animals, with abortion in pregnant females and a peracute course in neonates. Very high titres of virus are found in the blood and viraemia persists for up to 7 days. Long term carriers of the virus have not been described.

Humans are very susceptible to RVFV and it is a major zoonotic agent (OIE 2007). Human infection occurs through mosquito bites or from contact with infected foetuses or other infected material. Infections result in high-titre viraemia that can persist more than a week. Humans probably participate in amplification of the virus during epidemics such as the one in

Egypt during 1977-79 where one million people were estimated to have been infected (Acha 1987). In less than 1 % of human infections, the haemorrhagic or encephalitic form of the disease may develop resulting in serious disease or death.

Information on RVFV in cats and dogs is based largely on experimentally infected animals. High mortality is seen in young pups and kittens less than 3 weeks of age with an incubation period of 24 hours (Walker 1970b). Transmission of infection from pup to pup and pup to mother was demonstrated. A similar situation has been shown in cats (Walker 1970b). In the experimentally infected adult dogs, 50 % developed a viraemia, but clinical signs were not observed. Viraemia in adult cats appears to be uncommon, with viraemia demonstrated in two cats of 14 infected experimentally. However the validity of the positive results is questionable. As with the puppies, no clinical signs were noticeable in infected cats older than 3 weeks of age (Walker 1970b).

Early work in the 1960s investigating susceptibility of animals to RVFV lists the dog as not susceptible (Walker 1970a). However, as shown by Walker, dogs and cats are susceptible through experimental inoculation. In a study which used a specific plaque reduction neutralisation test on sera collected from wild and domestic cats and dogs in endemic regions, only lions from areas of three prior outbreaks returned positive results (House 1996). These results suggest that natural infection in the dog, whether domestic or wild, is rare.

No evidence was found to suggest that RVFV is excreted in dogs' semen.

Other *Bunyaviridae* infections

Of the genus *Bunyavirus*, some species in the California encephalitis group (Jamestown Canyon virus, La Crosse virus, and Snowshoe hare virus) naturally infect cats and dogs. These viruses are maintained in a cycle of transmission between mosquito and normal mammalian hosts. The normal mammalian hosts (rodents, chipmunks, snowshoe hares etc.) show no clinical signs if infected. Periodically these viruses are transmitted by their mosquito hosts to animals such as cats, dogs and humans that are not their usual hosts, causing clinical illness (Greene 2006). Incidentally infected mammalian hosts are not contagious, nor do they develop viraemia that is sufficient to infect mosquitoes (Godsey 1988). Thus, people, cats and dogs are dead-end hosts that are unable to infect other vertebrate or invertebrate hosts (CDC 2007).

Experimentally, Tensaw virus inoculated into cats and dogs resulted in an asymptomatic viraemia whereby mosquito transmission from infected dogs was demonstrated (Greene 2006). However, there are no reports of natural infection in the cat or dog and, since infection is of no consequence, and the vector *Anopheles* spp. are not present in New Zealand (Holder 1999), therefore Tensaw virus is not considered a hazard.

In conclusion, viruses in the genus *Bunyavirus* are not considered potential hazards since natural infection in the cat and dog is rare, incidental and not contagious.

Hantaviruses in the genus *Hantavirus*, in contrast to all other viruses in the family *Bunyaviridae*, are not transmitted by arthropods, but are rodent-borne by aerosol exposure to excreta of infected rodents (Elliot 2000). Hantavirus infections in humans receive the most attention (pathogenic in humans), but the viruses have a wide host range among mammals including cats and dogs (Greene & Berg 2006). Diagnosis in cats and dogs is by serological testing as infection is asymptomatic. The very low seropositivity rates found in pets suggest that like humans, they acquire infection from infected rodents and their excreta (Greene &

Berg 2006). Cats and dogs are not considered important in viral maintenance and there is no evidence that infected cats or dogs can transmit infection to other animals or to humans (Nowotny 1994). Given that these viruses are rodent-borne and that infected cats and dogs have not been associated with transmission of Hantaviruses, they are not considered to be potential hazards.

36.1.5. Hazard identification conclusion

It is concluded that Rift Valley fever virus is a potential hazard in cats and dogs but not in semen. Other *Bunyaviridae* infections are not considered to be potential hazards in the commodity.

36.2. RISK ASSESSMENT

36.2.1. Entry assessment

There is no evidence that the cat or dog have carried RVFV to new areas of the world. Experimental studies demonstrate that cats and dogs less than 3 weeks of age are susceptible to RVFV with a short incubation period and high mortality rates occurring. Experimentally infected older animals remained asymptomatic with 50 % of infected dogs developing a viraemia that probably lasted several days (Walker 1970a). Adult cats appear not to develop a viraemia.

In a survey of domestic and wild dogs and cats within endemic regions, antibody to RVFV was found only in free-roaming lions (House 1996). These findings suggest that natural infection is probably very rare in cats and dogs.

RVF is primarily a tropical disease of ruminants and humans in sub-Saharan Africa. Since natural infection in cats and dogs appears to be very rare, and is of a short duration, the likelihood of importing a viraemic animal is assessed to be negligible.

36.2.2. Risk estimation

Since entry is assessed to be negligible, RVFV is not classified as a hazard in the commodity and risk management measures are therefore not justified.

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37. Flaviviridae

37.1. HAZARD IDENTIFICATION

37.1.1. Aetiological agent

The Family *Flaviviridae* is made up of three genera of which one, *Flavivirus*, contains species that infect cats and dogs (Thiel et al 2005). The preliminary hazard list (Appendix 1) includes nine viruses in this genus namely; Japanese encephalitis virus, yellow fever virus, louping ill virus, Murray Valley encephalitis virus, Powassan virus, St. Louis encephalitis virus, tick-borne encephalitis virus, Wesselsbron virus and West Nile virus.

37.1.2. OIE List

Japanese encephalitis and West Nile fever are listed within the category of "multiple species diseases" (OIE 2007).

37.1.3. New Zealand's status

Japanese encephalitis virus is a notifiable organism. Louping ill virus, Murray Valley encephalitis virus, and Wesselsbron virus are listed on the unwanted organisms register as 'other' exotic organisms. The remainder are not listed (Ministry of Agriculture & Forestry 2008).

37.1.4. Epidemiology

Most flaviviruses are arthropod-borne and are maintained in nature by transmission from haematophagous arthropod vectors to vertebrate hosts. Some viruses have a limited vertebrate host range (e.g. only primates) while others can infect and replicate in a wide variety of species (mammals, birds, etc.) (Thiel et al 2005).

Flaviviruses have a worldwide distribution but individual species are restricted to specific endemic or epidemic areas e.g. yellow fever virus in tropical and subtropical regions of Africa and South America, Japanese encephalitis virus in Southeast Asia and tick-borne encephalitis virus in Europe and Northern Asia (Thiel et al 2005).

More than 50 % of known flaviviruses have been associated with human disease, including the most important human pathogens: Yellow fever virus, Dengue virus, Japanese encephalitis virus, West Nile virus and Tick-borne encephalitis virus. Flavivirus-induced disease may be associated with clinical signs of the central nervous system (meningitis and encephalitis), fever, rash and haemorrhagic fever (Thiel et al 2005).

Several flaviviruses are pathogenic for domestic animals and cause economically important diseases.

In the following discussion the flaviviridae are divided into mosquito-borne and the tick-borne viruses.

Mosquito-borne flaviviruses

The mosquito-borne viruses include Japanese encephalitis virus, yellow fever virus, West Nile virus, Wesselsbron virus, Murray Valley encephalitis virus and St. Louis encephalitis virus.

West Nile virus (WNV) has been recognised in Europe and Africa for several decades but has recently emerged in North America. It causes neurological diseases in humans, horses and certain species of birds, and is primarily maintained in wild birds (Shaw 2005). Serological surveys indicate that dogs are naturally infected and seroconvert without clinical disease. In experimental infections, dogs developed viraemias of low magnitude and short duration without clinical signs. Cats developed higher viral titres than dogs and mild non-neurological clinical signs. However, the titres in cats were still low and transient compared with those found in birds (Austgen et al 2004; Lichtensteiger & Greene 2006). In general, mammals are considered dead-end hosts (Lichtensteiger & Greene 2006). There is no evidence that cats and dogs have played any role in transmission of West Nile virus to other vertebrates or mosquitoes naturally or experimentally. Since WNV infection causes low transient viraemia in cats and dogs, they are considered dead-end hosts. Therefore, WNV is not a potential hazard.

There is little information on St. Louis encephalitis virus (SLEV) infection in dogs and cats. This virus is very closely related to West Nile virus which has been better studied in the cat and dog. Disease due to SLEV is not known to occur in any species other than humans (Leighton 2000b). In humans, infection is usually inapparent but occasionally severe fatal encephalitis develops, particularly in the elderly (Luby 1994). SLEV is found only in the Americas and like WNV is maintained in certain species of birds that amplify the virus (Leighton 2000b). Cats were found to be refractory to experimental infection, and in human epidemics, dogs may be infected but are relatively resistant to infection (Greene & Baldwin 2006). It is likely SLEV behaves in a similar fashion to West Nile virus infection in dogs and cats, and it is concluded that infection is accidental and that they are dead-end hosts. Therefore SLEV is not a potential hazard.

Yellow fever is a tropical human disease transmitted by certain *Aedes* spp. of mosquito in endemic regions of Africa and Central and South America. Monkeys are the reservoir host, but all primates are susceptible, with humans also acting as amplifying hosts. No other animals are important in the life cycle (World Health Organization 2001). Wild rodents and birds are resistant to experimental infection (Meegan 1994). Puppies, even when splenectomised, could not be infected experimentally. A transient viraemia has been described in inoculated cats (Greene & Baldwin 2006). Natural infection has not been reported in cats or dogs. Yellow fever occurs only in countries with tropical climates where competent *Aedes* spp. mosquitoes and monkey reservoir hosts are present. As cats and dogs are not naturally susceptible to this virus, it is not a potential hazard.

Japanese encephalitis virus is endemic throughout much of Asia, particularly southeast Asia and Japan. Recently the virus has been detected in Torres Strait Islands and mainland Australia (Hanna et al 1999). The virus is transmitted by some mosquitoes in the genus *Culex*. No competent vectors occur in New Zealand. Infection of humans and horses may cause

severe and often fatal encephalitis. Water birds are maintenance hosts and pigs are amplifying hosts. No other animals carry the virus (CDC 2001). Infection in cats and dogs is subclinical, (Tipold & Vandevelde 2006) and, like humans and horses, they are considered dead-end hosts. Therefore, JEV is not a potential hazard.

Wesselsbron disease is an acute arthropod-borne infection of domestic ruminants found in warm, moist parts of southern Africa and Thailand. It is transmitted by species of *Aedes* mosquito that do not occur in New Zealand. Sheep, cattle and goats may play a role in the maintenance of the virus (Swanepoel & Coetzer 2004). Dogs may occasionally be infected, but there are no reports of transmission from the dog or cat to any other animal or arthropod. In addition, the required vectors are not present in New Zealand. This virus is therefore not a potential hazard.

Murray Valley encephalitis is a zoonotic viral disease in Australia. Most epidemics are limited to the Murray-Darling River basin. Most human infections are subclinical. Clinical disease in humans may be a mild febrile illness or result in encephalitis, in which case about 20% may be fatal. The virus is believed to be maintained in a cycle involving water birds and mosquitoes in northern Australia and New Guinea. The major vector is *Culex annulirostris* which does not occur in New Zealand. It has been suggested that following epidemics the virus disappears from southern Australia and is reintroduced from the north when bird-mosquito cycles build up in years of high rainfall in Queensland and the Northern Territory (Aaskov & Doherty 1994). Experimental studies indicate that Grey kangaroos and rabbits might be reservoir hosts. A wide range of mammals can be infected with MVEV including dogs (Kay et al 1985). However, there is no evidence that infection of cats or dogs is associated with disease, or that they can transmit infection to other animals or arthropods. In addition, the required vector is not present in New Zealand. MVEV is therefore not a potential hazard.

No evidence was found to suggest that any of the viruses are excreted in dogs' semen.

37.1.5. Hazard identification conclusion, mosquito-borne flaviviridae

The epidemiology of these viruses is complex and they normally cycle between birds or other animals and certain mosquito species in specific environments. Spillover into humans and other accidental hosts occurs only under specific conditions that are very unlikely to occur in New Zealand. The climate, maintenance hosts and vectors necessary for the establishment of the viruses do not occur in New Zealand. Therefore, no mosquito-borne flaviviruses are considered to be potential hazards in the commodities.

Tick-borne encephalitis viruses

The tick-borne encephalitis viruses listed include; louping ill virus, tick-borne encephalitis virus and Powassan virus.

Tick-borne encephalitis in central Europe, Norway, Italy and Greece tends to be endemic in focal areas. Disease has been described in humans, dogs, horses, monkeys and wild ruminants but not in cats (Tipold & Vandevelde 2006). The disease follows the distribution of the respective vectors, *Ixodes ricinus* or *Ixodes persulcatus*. Wild rodents (*Clethrionymys* and *Apodemys* species) are the reservoir hosts. In animals, transmission is entirely by ticks. In dogs virus is rapidly cleared if they are accidentally infected from exposure to infected ticks (Weissenbock et al 1998).

Louping-ill is an acute viral encephalomyelitis transmitted by the sheep tick *Ixodes ricinus* that occurs in the UK. Although louping-ill occurs most frequently in sheep, dogs, but not cats have been reported with meningioencephalitis caused by louping-ill virus. However, the ecology of louping-ill virus depends largely on a sheep-tick cycle, with little involvement of other animals (Tipold & Vandevelde 2006).

Powassan virus, a North American tick-borne flavivirus, is closely related to the Eastern hemisphere's tick-borne encephalitis viruses.

Powassan virus very rarely causes encephalitis in humans (27 cases in Canada and the northeastern United States reported between 1958 and 1998). Dogs and cats appear refractory to disease when experimentally infected (Leighton 2000a). Disease associated with infection with Powassan virus has not been reported in cats or dogs.

There is no evidence to suggest that any tick-borne flaviviruses are transmitted by dogs' semen.

37.1.6. Hazard identification conclusion, tick-borne encephalitis viruses

Tick-borne flaviviruses are exotic organisms that may cause significant disease in humans and domestic animals. Therefore, it is concluded that they are potential hazards in cats and dogs but not in dogs' semen.

37.2. RISK ASSESSMENT

37.2.1. Entry assessment

Tick-borne encephalitis viruses
Infected dogs and cats do not necessarily display clinical signs and infection can go unnoticed. They are unlikely to develop viraemias of sufficient quantity or duration to infect ticks and are not normally part of the life cycle of tick-borne encephalitis viruses. It is possible that animals that have been recently infected could be imported. However, periods of viraemia are short and no long term carrier state has been described.

The likelihood of entry is therefore assessed to be negligible for cats and dogs that are not infested with ectoparasites.

Since tick-borne encephalitis viruses are only carried by ticks it is important not to introduce ticks together with imported cats and dogs. Any ticks on an imported animal could harbour encephalitis viruses and act as a source of infection.

The virus could be introduced by importing dogs or cats infested with vector ticks. Therefore entry assessment is non-negligible for these animals.

37.2.2. Risk estimation

As the entry assessment is negligible for tick-borne encephalitis viruses, the risk is assessed as negligible for imported cats and dogs that are not externally parasitized.

The risk of introducing tick-borne encephalitis viruses through importation of dogs and cats is assessed as negligible. Therefore risk management measures are not justifiable.

However, measures to ensure that ticks are not introduced with cats and dogs entering New Zealand as described in Section 30.3 should be implemented.

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38. Herpesviridae

38.1. HAZARD IDENTIFICATION

38.1.1. Aetiological agent

Family Herpesviridae; suid herpesvirus1, Aujeszky's disease virus is exotic to New Zealand. Other canine and feline herpesviruses have worldwide distributions that include New Zealand.

38.1.2. OIE List

Aujeszky's disease is listed within the category of "multiple species diseases".

38.1.3. New Zealand's status

Aujeszky's disease virus is an exotic, unwanted organism
(Ministry of Agriculture & Forestry 2008).

38.1.4. Epidemiology

Aujeszky's disease (pseudorabies) is a disease of pigs that was eradicated from New Zealand in 1995 (OIE 2006). It occurs world-wide except Australia, Canada, Finland, Sweden, Denmark and the UK. Some countries are eradicating the disease (Van Oirschot 2004). Pigs are the principle reservoir host but the virus can be transmitted to sheep, goats, cattle, cats and dogs by close contact with infected pigs (Van Oirschot 2004; Vandevelde 2006). Humans are not susceptible. Dogs are infected by ingesting infected raw pork or biting infected pigs (Vandevelde 2006). In animals other than pigs the disease is characterized by acute pruritis, salivation, nervous signs and it is invariably fatal. Animals other than pigs are not known to carry the virus or to act as sources of infection (Van Oirschot 2004; Vandevelde 2006).

No specific treatment is available for cats and dogs and any supportive treatment such as anaesthetizing the animal is futile since infection is almost always fatal.

38.1.5. Hazard identification conclusion

Aujeszky's disease virus has been classified as an exotic, unwanted organism and is therefore concluded to be a potential hazard in the commodity.

38.2. RISK ASSESSMENT

38.2.1. Entry assessment

Aujeszky's disease in cats and dogs only occurs when they have been in close contact with infected pigs. Eradication of infection from pigs in many areas of the world means that disease in cats and dogs is becoming rare (Vandevelde 2006). When it occurs the clinical signs are severe and include neurological signs, excessive salivation and intense pruritis that manifests as self mutilation (Lake 1990). The outcome is almost always fatal and the course of disease until death is generally less than 48 hours (Vandevelde 2006).
Under these circumstances of rare infection in cats and dogs, combined with dramatic severe clinical signs (sudden death), the likelihood of entry is therefore assessed to be negligible for imported cats and dogs.

Likewise, the likelihood of an infected dog donating semen is negligible.

38.2.2. Risk estimation

Since the entry assessment is negligible, risk is estimated to be negligible; therefore risk management measures are not justified.

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39. Orthomyxoviridae

39.1. HAZARD IDENTIFICATION

39.1.1. Aetiological agent

The preliminary hazard list (Appendix 1) identifies three influenzaviruses in the family *Orthomyxoviridae* that infect either cats and/or dogs. These are influenzaviruses A (including highly pathogenic avian influenza virus) B and C.

39.1.2. OIE List

Highly pathogenic avian influenza (HPAI) is included within the category of avian diseases (OIE 2007).

39.1.3. New Zealand's status

Influenzavirus type A (exotic avian strains) are listed as unwanted, notifiable organisms (Ministry of Agriculture & Forestry 2008).

39.1.4. Epidemiology

Influenza viruses are RNA viruses that are inherently genetically unstable. New strains emerge continuously due to recombination and mutation events (antigenic shift and antigenic drift).

Type A viruses are divided into subtypes according to the antigenic nature of their surface glycoprotein haemagglutinins (H) and neuraminidases (N). There are currently 16 H types and 9 N types recognised (Olsen 2006). Virus isolates exhibiting many combinations of the H and N antigens have been found and due to the capacity of the influenza viruses to mutate and recombine, the types of virus circulating are constantly changing.

Some Type A viruses naturally infect humans and cause epidemics of acute respiratory disease. However, some influenzaviruses A also infect other mammalian species and a variety of avian species. Type A viruses are the only ones known to exhibit zoonotic potential under natural conditions (Slemons & Brun 1994). The evidence for this has been seen in the sporadic transmission of swine influenza to people and the recent transmission of an H5N1 strain from chickens to humans.

Influenza B virus strains appear to naturally infect humans and cause epidemics less frequently than the A viruses due to a slower antigenic drift. These viruses circulate continuously in humans causing respiratory disease commonly in childhood (Kawaoka 2005).

Type C viruses naturally infect humans and cause more limited outbreaks and may also infect pigs. Antigenic drift does not occur in these viruses as occurs in A and B viruses (Kawaoka 2005).

There have been many reports of serological evidence of infection of dogs and cats with influenza viruses. Experimental intranasal or intravenous infection of dogs and cats with influenza virus A and B strains and of dogs only with Type C strains have shown they are susceptible to these viruses (Greene 2006). Clinical signs are absent or mild, with inconsistent serological responses. In some instances cats or dogs have been infected by contact with animals that were infected. Natural infection has been associated with human populations that are experiencing epidemics of disease. There is no evidence to suggest that these viruses spread from infected pets back to humans (Greene 2006).

Waterfowl are commonly infected with influenza A strains and strains with all combinations of H and N antigens can be isolated from aquatic birds in various combinations; as such birds are regarded as the natural reservoir hosts (Daly 2006). Natural transmission of influenzaviruses is by aerosol (human and most non-aquatic hosts) or is water-borne (waterfowl) (Kawaoka 2005). Cats can become infected with H5N1 through close contact with infected birds (Leschnik et al 2007) and feeding on infected uncooked poultry meat or wild birds (Keawcharoen et al 2004).

In the avian influenza outbreak of 2004 caused by the Type A H5N1 strain that primarily occurs in birds, household cats and captive exotic cats in Thailand died in association with the death of chickens (Keawcharoen et al 2004). Numerous other mammalian species such as humans, seals, whales, mink and ferrets are also susceptible to avian influenza viruses (Greene 2006).

A cat found dead on the northern German island of Rugen, a highly infected area where wild birds with H5N1 infections have been found, tested positive for H5N1. No other cats in the area were found to be infected. It is thought the dead cat ingested large amounts of infected birds (Editorial team 2006). A dead infected cat that had ingested infected birds was reported from Austria.

Cats have been infected by experimental intratracheal inoculation and feeding of infected chickens. The incubation period is 2 days and virus is then shed in nasal secretions and faeces. Nasal secretions start 3 days after infection at relatively low titres and lasts 4 days or longer (Kuiken et al 2004). Clinical signs are fever, lethargy, dyspnoea and conjunctivitis. When clinical signs occur, the outcome of the disease is usually fatal within 1 week. Cats in close contact with infected dying cats for at least the first 7 days of infection can also be horizontally infected (Kuiken et al 2004).

There is no evidence that domestic cats play a role in the transmission cycle of H5N1 viruses (FAO Media Office 2007; Leschnik et al 2007) or are reservoirs of the virus. All available evidence indicates that cat infections are rarely documented and when they do occur, it is in association with H5N1 outbreaks in domestic or wild birds.

The sole case of H5N1 in a dog was reported in 2004 during an outbreak in Thailand. Fatal infection occurred 6 days after ingesting an infected duck (Songserm et al 2006).

Canine influenza is a newly emerging respiratory infection of dogs caused by influenza A subtype H3N8 virus which is of equine influenza origin (Daly 2006). Transmission between dogs is thought to be by close contact. Repeated introductions of the virus into kennels from

feeding dogs untreated meat, including lungs from infected horses, may also lead to infection (Daly 2005). In 23 states of the USA, greyhounds and a few pet dogs that mostly originated from shelters have been found to be seropositive. A serosurvey of rescue dogs and greyhounds did not find the infection to be endemic in the UK (Anonymous 2006). Experimental inoculation of four beagles resulted in mild fever, but none developed respiratory clinical signs. Only two dogs shed detectable amounts of virus; one dog for 2 days and the other for 4 days post-inoculation (Crawford 2005).

The failure to reproduce clinical signs in the experimentally infected dogs is not surprising since a large proportion of naturally infected greyhounds do not show clinical signs (Crawford et al 2006; Crawford et al 2005b). Clinical signs, if they occur, include a mild form and a more severe form that includes pneumonia. However, the majority of dogs with confirmed influenza infection recover without complications (Crawford et al 2006).

There is no evidence of natural transmission of canine influenza from dogs to other species such as humans, horses, cats or ferrets (Crawford et al 2006; Daly 2006).

39.1.5. Hazard identification conclusion

Exotic strains of avian influenzavirus type A infections have been reported in cats and dogs, therefore they are concluded to be potential hazards.

39.2. RISK ASSESSMENT

39.2.1. Entry assessment

Cat and dog infections occur through contact with H5N1 outbreaks in domestic or wild birds and by ingesting infected uncooked poultry meat or wild birds. When clinical signs occur, the outcome of the disease is usually fatal, within 1 week. Inapparent infection might occur for a limited period but persistent H5N1 infections have not been reported. Unlike domestic and wild birds, there is no evidence that domestic cats or dogs are reservoirs of the virus. There is no evidence that domestic cats and dogs play a role in the transmission cycle of H5N1 viruses and infection in domestic pets is rarely documented.

Since cats and dogs are rarely infected and are considered dead-end hosts in regards H5N1, risk of entry is assessed to be negligible.

Clinically affected domestic pets are rare and are excluded from travel. Subclinical infections are self-limiting.
Therefore the likelihood of entry of influenza viruses is assessed to be negligible.

39.2.2. Risk estimation

Since entry assessment is assessed to be negligible for H5N1, the risk from importing cats or dogs is estimated to be negligible.
The risk of introducing exotic avian influenza viruses in imported cats or dogs has been estimated as negligible. Therefore risk management measures are not justified.

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40. Paramyxoviridae

40.1. HAZARD IDENTIFICATION

40.1.1. Aetiological agent

The preliminary hazard list (Appendix 1) identifies viral species from three genera within the family Paramyxoviridae. These are Nipah virus and Hendra virus which are classified in the new genus *Henipavirus* (Eaton et al 2006). Also listed is Newcastle disease virus belonging to the genus *Avulavirus* and phocine distemper virus within the genus *Morbillivirus*. Canine distemper virus is endemic in New Zealand and is therefore not considered a potential hazard.

40.1.2. OIE list

Nipah virus encephalitis is listed under the category of 'swine diseases'.

40.1.3. New Zealand's status

Nipah, Hendra and Newcastle disease viruses are listed as unwanted, notifiable organisms (Ministry of Agriculture & Forestry 2008).

40.1.4. Epidemiology

Newcastle disease can infect a wide range of avian and nonavian species including cats that have been cerebrally inoculated or intranasally dosed with large amounts of virus (Alexander 2003; Greene & Lutz 2006). Chickens and other birds are the most important natural hosts and cats and dogs are not naturally infected (Alexander 2003). Therefore, Newcastle disease is not considered a hazard.

Phocine distemper virus (PDV) likewise does not naturally infect cats and dogs. The dog has been demonstrated experimentally to be susceptible to infection (Greene & Appel 2006). The natural reservoir host for this agent is the harp seal (Dierauf 2001). Since the likelihood of importing cats and dogs infected with PDV is considered negligible, PDV is not considered a hazard.

Nipah virus is a tropical disease that was first reported in Malaysia in 1998 and subsequently in Singapore, Bangladesh and India (Tan & Wong 2003; Katu 2004; Epstein et al 2006). This paramyxovirus that infects pigs, humans, horses, dogs and cats (Tan & Wong 2003) created a major public health crisis, with the death of 105 people attributed to Nipah virus infection when it first appeared (Katu 2004). Nipah virus attacks the central nervous system and respiratory systems. Encephalitis is the main cause of death in humans. Most human cases occurred in pig farmers.

The outbreak stopped once infected pigs in the area were destroyed. Over one million pigs were slaughtered to control and eradicate the outbreak in Malaysia (Katu 2004). The pigs

acquired infection from *Pteropid* species of fruit bat that have been identified as the natural reservoir host (Katu 2004).

Direct, close contact with pigs was the primary source of human infection. The virus multiplied but did not always cause clinical signs in pigs which were raised in high densities. Pigs excreted the virus in urine and respiratory droplets (Middleton et al 2002). The Malaysian outbreak led to the subsequent outbreak in Singapore. Infection in abattoir workers resulted from direct contact with infected pigs that had been imported from affected areas of Malaysia.

In Bangladesh from 2001 to 2005, five outbreaks were attributed to Nipah virus infection (Epstein et al 2006). These involved much smaller numbers of affected humans and no animal disease was evident, differing from the Malaysian epidemic. These outbreaks appear to have been due to spillover of virus directly from bats to humans (Epstein et al 2006). One outbreak was reported in 2001 in India, close to the Bangladesh border (Chadha et al 2006).

Only in the Malaysian outbreak were cats and dogs reported to be affected. There were two reports of dogs affected with Nipah virus; a dead village dog and a moribund dog that displayed clinical signs resembling that of distemper, consisting of respiratory distress with mucopurulent nasal and conjunctival discharges. Infection with Nipah virus was confirmed by histology and serology but not by virus isolation. There was only one field case of Nipah virus infection in a cat confirmed by necropsy and immunohistochemistry (Hooper et al 2001). Despite the lack of confirmed cases in the literature, farmers did notice large numbers of dogs dying on infected pig farms and cats were also reported by farmers as being affected (Kirkland 2006). Experimentally, cats could be infected with Nipah virus and developed clinical signs 6-9 days post-infection. Severe respiratory disease and systemic infection occurred (Middleton et al 2002). A serological survey showed that many dogs from infected pig farms had antibodies to Nipah virus (Hooper et al 2001).

There was, however, no evidence of lateral transmission and cats or dogs are not contagious although infection is associated with a high case fatality rate (Kirkland 2006).

It appears that natural infection might sometimes be subclinical but severe clinical signs leading to death are more likely.

Hendra virus is closely related to Nipah virus and occurs infrequently in Queensland, Australia. There have been six outbreaks reported since the virus was first identified in 1994 (Hanna et al 2006). It is a disease primarily of horses but in some outbreaks human infection also occurs. Transmission to humans, albeit rare, occurs through physical contact with nasal and oral secretions emanating from very ill, dying or dead horses (Hanna et al 2006). The reservoir host, *Pteropid* spp. of fruit eating bats, infects horses that may then transmit infection to humans in close contact with diseased horses.

There are no reports of natural infection with Hendra virus in cats or dogs. However, cats but not dogs are susceptible to experimental infection. Cats became clinically ill within 4-8 days post-infection and could then infect another cat that was in close contact (Westbury et al 1996). However, experimentally infected cats are not thought to be highly contagious and cats are not considered to be naturally susceptible (Westbury et al 1996).

There is no information on whether any of the viruses are present in semen of infected dogs.

40.1.5. Hazard identification conclusion

Newcastle disease virus and PDV are concluded not to be hazards.

Nipah virus and Hendra virus are listed as unwanted notifiable organisms. They are therefore concluded to be potential hazards in the commodity.

40.2. RISK ASSESSMENT

40.2.1. Entry assessment

Hendra virus: is a rare infection of horses and humans that occurs sporadically in a geographically restricted part of the world (Queensland, Australia). A limited survey of cats around metropolitan Brisbane did not find antibody to the virus (Westbury et al 1995). Since there are no reports of natural infection in cats or dogs, entry is assessed to be negligible for Hendra virus.

Nipah virus: the 1998 outbreak in Malaysia was primarily a disease of pigs and pig farmers. Since the Malaysian outbreak there have been six other reported outbreaks, five in Bangladesh (Epstein et al 2006) and one in India (Chadha et al 2006). These outbreaks involved human infections only and are considered to be a result of direct spillover of the virus from the natural reservoir (bats) to humans (Chadha et al 2006). Ongoing surveillance has been carried out in Malaysia where the disease is notifiable and no further cases have been reported since 1998.

Natural infection of cats and dogs with Nipah virus would appear to be very rare. Reported cases have been documented (Hooper et al 2001) only in the Malaysian outbreak. The incubation period for cats and dogs in natural infection is not known. The incubation period for cats is 6-9 days under experimental conditions. Since infection can be clinically obvious and fatal, it is highly unlikely that an animal displaying clinical signs would be imported from an endemic region. However, dogs may also be subclinically infected.

The low incidence of sporadic outbreaks and apparent eradication from Malaysia means the likelihood of importing infected cats and dogs is extremely low. However, since the virus presumably remains endemic in bats, further outbreaks may occur and the likelihood of entry is considered to be non-negligible for dogs and cats imported from countries where infected fruit bats occur. The likelihood of importing infected cats and dogs from non-affected countries is negligible.

40.2.2. Exposure assessment

Nipah virus has been isolated from the urine of experimentally infected cats, indicating that transmission via urine may be possible (Hooper et al 2001). However, there is no evidence that cats and dogs featured in the epidemiology of the Malaysian outbreak. Infections in cats and dogs are likely to be fatal or recovered animals would cease to excrete virus (Daniels 2007) and dogs and cats are not considered to be contagious (Kirkland 2006).

The disease is rare, appearing sporadically in tropical climates where the natural reservoir host *Pteropid* fruit bat species are found. The reservoir host does not occur in New Zealand, and cats and dogs are aberrant hosts that do not transmit infection to other animals.

Therefore the likelihood of transmission and establishment is negligible.

40.2.3. Risk estimation

Since the likelihood of entry is assessed to be negligible for Hendra virus, the risk from importing cats or dogs infected with this virus is estimated to be negligible.

Since the likelihood of exposure is assessed to be negligible for Nipah virus, risk is estimated to be negligible.

Since the risk estimate is negligible for Hendra and Nipah viruses, they are not classified as hazards in the commodities. Therefore, risk management measures are not justified.

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41. Reoviridae

41.1. HAZARD IDENTIFICATION

41.1.1. Aetiological agent

Two viruses in the genus *Orbivirus*, family *Reoviridae* infect dogs: African horse sickness virus (AHSV) and bluetongue virus (BTV) (Mertens et al 2005).

41.1.2. OIE List

African horse sickness and bluetongue are listed diseases.

41.1.3. New Zealand's status

African horse sickness virus and bluetongue virus are listed as unwanted notifiable organisms (Ministry of Agriculture & Forestry 2008).

41.1.4. Epidemiology

Bluetongue and African horse sickness viruses are non-contagious vector-borne diseases, with transmission requiring *Culicoides* spp. intermediate hosts (Coetzer & Guthrie 2004; Verwoerd & Erasmus 2004).

Bluetongue virus infects many ruminant species, in particular causing disease in sheep. It occurs in most tropical and sub-tropical countries. It is absent in countries south of 34^{o}, including New Zealand, and countries north of 50° (OIE 2006).

Natural infection in dogs is uncommon with low seropositivity reported from endemic regions (Greene & Baldwin 2006). There has been a recorded case of a BTV-contaminated vaccine used in pregnant bitches causing abortion and death (Wilbur et al 1994).

African horse sickness virus infects equine species and dogs. It is endemic in eastern, central and most parts of southern Africa. Dogs are occasionally infected with AHSV by ingesting infected uncooked horse meat (Coetzer& Guthrie 2004). The major clinical signs of infection are respiratory, leading to death (Van Rensberg & De Clerk 1981).

African horse sickness virus has been naturally and experimentally transmitted to dogs through ingestion of infected horse meat (OIE 2008), however, dogs are not thought to play any role in the spread of AHSV as infection is rapidly fatal, with the vector *Culicoides* spp. rarely feeding on dogs (Coetzer& Guthrie 2004). A study of 400 blood meals collected from *Culicoides* spp. from endemic areas failed to detect any canine blood (Braverman &Chizov-Ginzburg 1996).

There is no information on whether AHSV is present in the semen of infected dogs.

Cats are refractory to infection with AHSV (Coetzer & Guthrie 2004) and probably BTV since there have been no reports of infection in domestic cats.

41.1.5. Hazard identification conclusion

Since AHSV and BTV are unwanted notifiable organisms that cause severe disease in dogs, they are concluded to be potential hazards.

41.2. RISK ASSESSMENT

41.2.1. Entry assessment

The dog and cat are not the usual hosts for these viruses. There is a very low likelihood that recently infected dogs that show no noticeable clinical signs could be imported from endemic areas. The likelihood of entry is therefore assessed to be very low for dogs and negligible for cats since they are considered not to be susceptible to infection with AHSV or BTV.

41.2.2. Exposure assessment

AHSV and BTV are non-contagious diseases that are vector-borne requiring *Culicoides* spp. to transmit infection. A *Culicoides* surveillance programme has been operating in New Zealand since 1991 (Ryan et al 1991). About 15,000 insects collected from light traps are examined annually (Motha et al 1997) and sentinel cattle are monitored for seroconversion to viruses transmitted by *Culicoides* spp. To date, seroconversion to arboviruses has not been detected in sentinel cattle and no *Culicoides* have been trapped.

Since these viruses are non-contagious and require an intermediate host that is not present in New Zealand, the likelihood of exposure and establishment is negligible.

41.2.3. Risk estimation

Since the likelihood of exposure is negligible the risk is estimated to be negligible for AHSV and BTV and they are not classified as hazards in the commodity. Therefore risk management measures are not justified.

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42. Rhabdoviridae

42.1. HAZARD IDENTIFICATION

42.1.1. Aetiological agents

Order Mononegavirales, Family Rhabdoviridae, Genus *Lyssavirus*, rabies virus. Rabies virus is the representative member of the *Lyssavirus* genus. The genus is classified into four serotypes and seven genetic lineages based on molecular phylogenies (OIE 2004).

In addition, the viruses segregate into two phylogroups differing in biological properties such as antigenic cross-reactivity (WHO 2004).

Table 1: Classification of lyssaviruses. Reproduced from (WHO 2004).

Phylogroup	Genotype	Species	Abbreviation	Geographical origin	Principal host species
Isolates characterized					
I	1	Rabies virus	RABV	Worldwide (except several islands)	Carnivores (worldwide); bats (Americas)
I	4	Duvenhage virus	DUVV	Southern Africa	Insectivorous bats
I	5	European bat lyssavirus type 1	EBLV-1	Europe	Insectivorous bats (*Eptesicus serotinus*)
I	6	European bat lyssavirus type 2	EBLV-2	Europe	Insectivorous bats (*Myotis* Sp.)
I	7	Australian bat lyssavirus	ABLV	Australia	Frugivorous/insectivores bats (*Megachiroptera/Microchiroptera* Sp.)
II	2	Lagos bat virus	LBV	Sub-Saharan Africa	Frugivorous bats (*Megachiroptera* Sp.)
II	3	Mokola virus	MOKV	Sub-Saharan Africa	Unknown
Isolates to be characterized as new genotypes					
-	-	Aravan virus	ARAV	Central Asia	Insectovorous bats (Isolated from *Myotis blythi*)
-	-	Khujand virus	KHUV	Central Asia	Insectovorous bats (Isolated from *Myotis mystacinus*)
-	-	Irkut virus	IRKV	East Siberia	Insectovorous bats (Isolated from *Murina leucogaster*)
-	-	West Caucasian bat virus	WCBV	Caucasian region	Insectivorous bats (Isolated from *Miniopterus schreibersi*)

ICTV = International Committee on taxonomy of viruses

OIE List

Listed under “multiple species diseases”.

42.1.2. New Zealand’s status

Rabies is an unwanted, notifiable organism (Ministry of Agriculture & Forestry 2008).

42.1.3. Epidemiology

Bat lyssaviruses of phylogroup I are related to classical rabies viruses; they have only rarely been reported in humans or non-bat animal species (McCall et al 2005; Paweska et al 2006; Stantic-Pavlinic 2005). Commercial rabies virus vaccines are highly protective against these lyssaviruses, and they are recognised by commercial anti-rabies antibody preparations used for diagnostic tests (OIE 2004). The potential for infection of dogs and cats by these viruses is not fully understood. No natural infection has been reported but there have been experimental infections of dogs and cats with ABLV leading to mild clinical signs and seroconversion (McCall 2005; McColl 2007). Only classical rabies viruses of phylogroup I, genotype 1 are considered potential hazards in this analysis as these are the viruses associated with rabies in dogs and cats (Real et al 2005).

Rabies is a zoonosis that causes an acute, progressive, fatal encephalomyelitis. It is transmitted when virus is introduced into bite wounds, open cuts in skin, or mucous membranes from saliva or other potentially infectious material such as neural tissue (CDC 2006a). As rabies virus becomes sequestered in nervous tissue it is immunologically protected and therefore invokes a slow immune response in a natural infection. Virus spreads neuronally from the site of infection to the central nervous system and only spreads to other organs, most notably the salivary glands, in the terminal stages of the disease. Rabies virus is usually present in saliva from a few days before clinical signs appear (Fekadu 1991). The infective period in live domestic carnivores is considered to start 15 days before the onset of the first clinical signs and ends when the animal dies (OIE 2004). A measurable immune response is not usually present in domestic animals during incubation (Wandeler 2004).
Although the virus has a broad host range, it is considered to exist as distinct virus biotypes, each of which is adapted to one, or occasionally two, maintenance host(s) (Real et al 2005). One host cycle and its adapted biotype predominate in any geographical area, but more than one distinct transmission cycle can occur in a specific country. Once a cycle is established in a host species it can persist for decades, even centuries.
To persist, the virus needs to be excreted by the host and transmitted to another susceptible individual before death. Animal species vary in their susceptibility from very high (wolves, foxes, coyotes and jackals) to low (birds and marsupials). Domestic animals including the dog and cat are considered moderately susceptible, as are humans, raccoons, bats and skunks (Greene 2006). Herbivores and other non-biting animals, rodents and lagomorphs are dead-end hosts that play no role in the epidemiology of the disease.
Recovery from rabies has been recorded in experimental infections in up to 20 % of dogs. There are no definitive reports of recovery from natural infections (Fekadu 1991).
A level of natural immunity may exist, as shown by positive titres in unvaccinated dogs in endemic areas and reports of low virulence strains (East et al 2001). However, the lack of anamnestic response in dogs with pre-vaccination titres suggests that these may be cross reactions (Tepsumethanon et al 1991). As immunity from inapparent infections and recovery

would occur in very few animals, population immunity can be considered to be the result of vaccination alone.
The dog is the global reservoir, and species of the orders Carnivora and Chiroptera (bats) are recognised as the main wildlife reservoirs (WHO 2004). Important wild carnivores include foxes, raccoons, skunks and mongoose among others (Rupprecht et al 2004). Cats are not recognised as a reservoir species (Fogelman et al 1993).
Three main types of rabies transmission cycle occur: canine, wildlife (sylvatic) and bat related, with the virus adapted to the specific host type.

42.1.3.1. Canine cycles

Dogs are the main reservoir of rabies and the main transmitter to humans in the developing world. Such countries may also have poorly developed infrastructure, veterinary regimes and health status of the animal population. Half of the global human population lives in canine rabies endemic areas (WHO 2004).

Cats are less susceptible to canine biotypes than dogs. However, due to the high levels of challenge in these countries the likelihood of spillover is also high.

42.1.3.2. Wildlife cycles

As the incidence of dog rabies is reduced or eliminated, wildlife and bat rabies may become apparent (WHO 2004). This has occurred in the USA, Brazil and South Korea among others.

In areas with wildlife cycles, domestic carnivores are infected by spillover infection from wild animals (Cliquet & Picard-Meyer, 2004). Although rabies in dogs and cats is diagnosed less commonly than in wildlife in countries with wildlife cycles, dogs and cats may represent a higher risk to human beings because of the close association between pets and their owners (McQuiston et al 2001). Ninety percent or more of human infections are acquired from these domestic animals (Cliquet & Picard-Meyer 2004; McKay & Wallis 2005).

In their review of rabies in the USA in 1988 Eng and Fishbein (1990) noted that the cases were seasonal, coinciding with seasonal increases in rabid wild animals. In Maryland the disease in cats is seen to parallel the epidemic in raccoons (Fogelman et al 1993) and is consistent with the pattern of spillover to cats in Florida (from raccoons and foxes) and West Germany (from foxes).

42.1.3.3. Bat cycles

Infection of bats due to rabies virus is reported only in the United States and certain Latin American countries (Table 1). Chiropteran (bat) and carnivore viruses appear to be relatively compartmentalized and rabies of bat origin is not known to play an important role in terrestrial rabies endemics. However spillover occurs in domestic and wild terrestrial mammals (Rupprecht et al 2004). Occasional clusters have been reported of probable bat origin (Daost et al, 1996). The United States Centers for Disease Control and Prevention (CDC) report that most of the recent human rabies cases in the USA have been caused by rabies virus from bats (CDC 2006b). The presence of European bat lyssavirus and Australian bat lyssavirus is not considered to affect a country's rabies-free status, in a regulatory sense.

Although dogs and cats can be infected with rabies virus genotype 1, they do not appear to be commonly infected with bat associated rabies virus biotypes in the USA and are unlikely to be an important source of secondary transmission of these biotypes to humans (McQuiston et al 2001).

42.1.3.4. Disease in humans

Mainly in Asia and Africa approximately 55 000 people die each year from rabies. In more than 99 % of human cases the virus is acquired from dogs (WHO 2004).
Clinical rabies is incurable, but pre-exposure vaccination and post-exposure prophylaxis (PEP) are effective. PEP comprises wound care, passive treatment with human derived and heterologous rabies immunoglobulin and active vaccination. Ideally this is given within 2 hours of exposure (McKay & Wallis 2005). It may be 100 % effective in preventing death if given before clinical onset. Once clinically affected, treatment appears only to prolong the course of the disease (WHO 2004).

42.1.3.5. The disease in dogs and cats

Dogs and cats differ in their susceptibility to different rabies strains. Cats are generally more susceptible to wildlife isolates and less susceptible to canine isolates. In addition, cats are more susceptible to attenuated strains. In experimental studies, 18 month old cats have a decreased susceptibility to street virus when compared to kittens (Bunn 1991b). Rabies in cats may not be as readily recognised or reported as that in dogs (Fogelman et al 1993).

The incubation period is usually 2-8 weeks but can vary from 7 days to more than a year (Aubert 1992). It depends on the virulence of the virus in the infected species, the route and site of inoculation, the dose of virus and the immune status of the individual. The incubation period is considered to be 6 months for the purposes of international trade (OIE 2007).

The clinical course is 3-10 days. The disease in dogs and cats consists of a prodromal phase lasting from 1 to 3 days that may be missed by the owner. It is followed by either, or a combination of, the furious or paralytic form. These forms are not always clearly demarcated. The furious form is the most common syndrome in cats. The animal may wander aimlessly, bump into objects, display excitement, irritability, and bite or attempt to bite animals, people and inanimate objects (‘mad dog’ syndrome), have a depraved appetite, altered voice, muscle paralysis, salivation, convulsions, ataxia, paralysis and death. The paralytic or “dumb” form is most common in dogs. The animal is lethargic and hides, does not usually bite, has muscular tremors, perceived difficulty in swallowing, and terminal paralysis. An inapparent form has also been described in dogs and cats. Affected animals may seroconvert, survive and serve as a source of the virus for extended periods. This form is considered extremely rare (Swanepoel 2004). Chronic recrudescent rabies that can last more than 120 weeks has also been reported in cats (Perl et al 1977).

The different forms of the disease complicate clinical diagnosis. Rabies cannot easily be definitively diagnosed other than by post mortem examination of the brain.

42.1.3.6. Vaccination

Immunization with a licensed rabies vaccine produced from any fixed strain will protect an animal against infection with any virulent street rabies virus (Bunn 1991a). Vaccination will not reliably prevent the disease if given after exposure (OIE 2004). A delay in importation after vaccination is necessary to allow for development of clinical signs of rabies acquired prior to vaccination (Fooks et al 2000). This delay should be for the recognised period of incubation of the disease.

Cell culture produced, inactivated, adjuvanted vaccines have largely supplanted the modified live virus (MLV) vaccines because the latter have several important disadvantages, including reversion to virulence and the need for intramuscular administration (Precausta & Soulebot 1991). Inactivated cell culture vaccines can be used to provide stable, long-lasting immunity provided they are administered correctly (OIE 2004; WHO 2004).

There are limited data publicly available on long term duration of immunity as a result of vaccination with an inactivated, adjuvanted vaccine. Published reports demonstrate that all dogs survived challenge 444 days post vaccination under experimental conditions (Gerber et al 1985) and 3.5-4 years post vaccination under field conditions (Bahloul et al 2006). However, the number of animals used in these studies was small. A study has recently been published that demonstrates survival of 88 % of dogs vaccinated with a combined distemper, canine adenovirus type 1, canine parvovirus and rabies vaccine and challenged three years post vaccination (Lakshmanan et al 2006).

(MacDiarmid & Corrin 1998) assessed the likelihood of importing and releasing a rabies infected animal into New Zealand under a number of import policies based on vaccination and quarantine periods. The risk analysis concluded that vaccinated cats and dogs imported without prolonged quarantine pose no greater risk of introducing rabies than cats and dogs entering through 6 months quarantine. Before that assessment, cats and dogs were only imported into New Zealand from rabies free countries. As a result of the risk analysis MAF permitted the importation of dogs from countries in which rabies occurs in wildlife and long quarantine periods were not imposed.

42.1.3.7. Rabies case data

Incidence of rabies in vaccinated animals is hard to determine as results from scientifically designed field trials are not available. In Peru the incidence of rabies fell from an average of 1,233 cases to three cases following a vaccination campaign where 270 000 (65 % of the estimated dog population) were vaccinated (Chomel et al 1988).

In 1999, 308 dogs and cats diagnosed with rabies in the USA were evaluated. Only one was considered to be currently vaccinated, and this animal had had only one vaccination at the age of 2-3 months (vaccination protocols recommend at least three months of age at first vaccination), nine months prior to death. In addition it had fought with a wild animal one month prior to death (McQuiston et al 2001).

In their analysis of rabies reports from Texas, Clark & Wilson (1996) reported 25 laboratory confirmed cases of vaccination failure in dogs and cats between 1976 and 1990, eight of which had been vaccinated between one to two years previously. Between 1991 and early 1995, vaccine failure was reported in only seven animals (six dogs, one cat), six of which were immunised with inactivated, adjuvanted vaccines.

In these failures of cell culture derived, inactivated vaccines, no animals were reported to have been serologically tested post vaccination. In additional reports the vaccine type was not specified (Tepsumethanon et al 1991) or MLV vaccines (Eng & Fishbein 1990) were used.

42.1.3.8. Serological testing

To be protective a vaccine must be adequately potent and correctly stored and administered. Measurement of the cellular immunity after vaccination is not possible with tests currently available for certification of animals for export/import purposes. However, cell culture

derived, inactivated adjuvanted rabies vaccines elicit a highly protective response; evidence of an adequate serological response to a vaccine demonstrates effective vaccination.

There is a clear correlation between seroconversion before challenge and protection from challenge indicating that anti-rabies antibodies are important in protecting animals (Gerber et al 1985; Coyne et al 2001; Aubert 1992). Presence of antibody in dogs at the time of challenge is considered important, and has been shown to be associated with an increased survival 3 years post vaccination (Tepsumethanon et al 1991). Protection correlates with high initial titres. Antibody titres follow a typical response curve, peaking at about 2-4 weeks post vaccination (Mansfield et al 2004; Kallel et al 2006). After this time titres decrease and, as a result, the risk of test failure increases (Fooks et al 2002).

Seroconversion is likely to be influenced by factors such as vaccine potency, vaccine storage and operator factors (errors in administration). When these are adequate, the neutralising antibody response will be related only to the individual dog's immune response (Aubert 1992). A risk reduction of between 1.5 and 3.8 has been attributed to serological testing when the waiting time post-vaccination was 120 days (EFSA 2006).

Prescribed tests for international trade are the fluorescent antibody virus neutralization test (FAVN) and the rapid fluorescent focus inhibition test (RFFIT) (OIE 2004). These tests have been shown to give equivalent results (Briggs et al 1998; Cliquet et al 1998). An indirect ELISA is available, but it is not as sensitive as the FAVN or RFFIT (Cliquet et al 2004). A double-antigen sandwich ELISA that may have a future application has been described (Yang et al 2005).

The World Health Organization has designated serum neutralising titres of 0.5 IU/ml as a reliable indication of successful vaccination (WHO 1992). Almost all vaccinated animals can be expected to have titres above the threshold of 0.5 IU/ml, although the titre peak may be short-lived after the primary vaccination. High and durable antibody levels are usually achieved following booster vaccinations. In good quality serum samples neutralising activity is usually highly indicative of antibody; non-specific neutralising substances (e.g. haemoglobin caused by haemolysis) may cause virus neutralisation giving false titres. However, such non-specific titres are usually well below the 0.5 IU/ml cut-off value.

42.1.4. Hazard identification conclusion

Rabies is a zoonosis and can infect all mammals. It is a notifiable and unwanted organism that causes severe disease, therefore it is concluded to be a potential hazard in the commodity.

42.2. RISK ASSESSMENT

42.2.1. Entry assessment

Dogs and cats are susceptible to rabies. There is a long incubation period and clinical signs are variable. The movement of dogs with humans is associated with outbreaks of the disease, and historically with establishment of new cycles of infection (Bingham 2005). Since animals show no signs of infection during a long incubation period, the likelihood of introducing an infected animal is considered to be non-negligible.

Infection of semen has not been described in dogs. Viraemia does not occur in cases of rabies except in experimental infections of mice with large doses of virus (Swanepoel 2004). Infection of organs other than the nervous system does not occur except in the terminal stages of the disease when the salivary glands and some other organs may be infected (Swanepoel 2004). It is inconceivable that a dog in the terminal stages of rabies would be used as a semen donor. Therefore, the likelihood that semen would be infected with rabies when collected from dogs that remain healthy for 15 days after semen collection is considered to be negligible.

42.2.2. Exposure assessment

The incidence of rabies in a particular species is dependent on its susceptibility and the probability of potentially infectious encounters (WHO 2004). Spillover infection rarely leads to a transmission cycle in a novel host, and even if this occurs this cycle is likely to be short lived for a few generations of transmission. In Europe there is no evidence that fox rabies has been introduced into a rabies-free area through the movement of domestic animals, despite breaches in the control regulations (Aubert 1992). Canine cycles have not emerged in developed countries with wildlife cycles. In North America the epidemics in dogs and cats are related to the disease in the local wildlife vector, with no evidence of a canine cycle (MacDiarmid & Corrin 1998). Imports from countries with established cycles in stray dogs are likely to pose the highest risk of introducing a strain that could establish in indigenous dogs or cats.

There is little detailed information on dog and cat population densities, distributions and proportion of stray animals worldwide. However it appears that the New Zealand dog population is relatively sparse and subject to a relatively high level of control. The dog population is estimated to be 500,000 in a country of land area of 268,021 square kilometres, or 1.8 dogs per square kilometre over the whole country. Six percent are considered unowned (Department of Internal Affairs 2003). As a comparison, other published estimates describe population densities up to 2930 (stray dogs, Kathmandu) per square kilometre in canine rabies endemic areas (Kato et al 2003). For this reason the likelihood that a canine cycle could establish is believed to be low.

All mammals including humans could be infected with rabies if bitten by an infected dog or cat. Contact between imported animals and domestic cats and dogs are possible but traceback and control of contacts in such cases would probably be efficient. Stray dogs, feral cats and feral mustelids are potential reservoir hosts, but contact between these animals and imported dogs and cats is unlikely. Bats are rare and unlikely to contact imported dogs or cats. Contact between potential reservoir hosts and imported dogs and cats is unlikely but not impossible, therefore the likelihood of exposure is non-negligible.

42.2.3. Consequence Assessment

If an infected dog or cat were to be imported it would pose a risk to humans and other susceptible species during the clinical phase before it died. Such an infected animal is likely to be quickly identified as having rabies and contacts traced and eliminated. In the worst case scenario such an animal might not be identified as rabid, and could have had contact with multiple other animals. The severity of the consequences would depend on the speed of detection of the disease. If a case were not diagnosed the secondarily infected animals could be dispersed before they show clinical signs months later.

If rabies occurred in a single animal not in a quarantine facility, New Zealand would lose its rabies-free status for 6 months. The status would be lost for 2 years if an indigenously acquired case is confirmed (OIE 2006). The majority of our export certificates for live animals i.e. dogs, cats, cattle, sheep, horses etc and some germplasm export certificates have rabies freedom clauses. Losing this freedom would result in MAFBNZ needing to renegotiate rabies protocols with importing countries. A loss of rabies freedom would almost certainly result in most importing countries requiring rabies testing prior to export.

If rabies were introduced, all humans receiving dog or cat bites from potentially infected animals would require post exposure prophylaxis (PEP). PEP is the highest monetary cost associated with rabies in developed countries. In a recent case of rabies reported in France, in an imported puppy, 187 people received PEP, over 1200 suspect animals were investigated, and 57 animals were confirmed as contacts. All were found negative (EFSA 2006).

There would probably be significant public concern if even a single case of rabies occurred in an imported animal.

No references could be found relating to transmission of rabies venereally in dogs. Viral shedding may occur in the saliva just prior to a dog displaying clinical signs. Whether this may involve a generalised viraemia involving the reproductive organs is unlikely. It is considered that the likelihood of transmission from semen collected from clinically healthy dogs is extremely low.

The introduction of rabies virus is likely to cause significant direct and indirect negative consequences. The consequences are therefore assessed to be non-negligible.

42.2.4. Risk estimation

The likelihood of introduction of rabies virus in dog semen is considered to be negligible, provided the donor remains clinically healthy for 15 days after semen donation. Additional measures for semen are not warranted.

Since the likelihood of entry and exposure and the consequences of entry are assessed as non-negligible, the risk of introducing rabies virus when importing dogs and cats is considered to be non-negligible. Therefore, the implementation of measures to effectively control the importation of rabies virus in these commodities can be justified.

42.3. RISK MANAGEMENT

42.3.1. Options

The following should be considered when designing options to effectively control the importation of rabies virus in cats and dogs:

- Three main types of rabies transmission cycle occur in the world: canine, wildlife and bat related, with the virus adapted to the specific host type. The degree of risk is proportional to the type of cycle and degree of challenge in the country of origin. Imports from countries with established cycles in stray dogs are likely to pose the highest risk of introducing a rabies strain

that may establish in New Zealand. Rabies has a long incubation period, during which animals show no signs of infection and the disease cannot be definitively diagnosed in a living animal.
- In natural infection antibodies are not produced until clinical disease occurs. Testing for antibodies is therefore not an option.
- Vaccination is highly effective for protecting animals against rabies infection at a population level. In comparison to the number of animals receiving vaccination, an extremely small number of individual vaccine failures have been reported.
- The (MacDiarmid & Corrin 1998) risk analysis shows that vaccination is equivalent to 6 months quarantine.
- Serological tests are available to demonstrate that vaccination has been effective.
- The *Code* chapter on rabies defines a rabies free country and makes recommendations relating to the safe importation of dogs, cats and dogs' semen.

The *Code* chapter on rabies defines a rabies free country and makes recommendations relating to the safe importation of dogs, cats and dogs' semen. These are as follows:

Article 8.11.2.

Rabies free country

A country may be considered free from rabies when:

1. the disease is notifiable;
2. an effective system of *disease surveillance* is in operation;
3. all regulatory measures for the prevention and control of rabies have been implemented including effective importation procedures;
4. no *case* of indigenously acquired rabies infection has been confirmed in man or any animal species during the past 2 years; however, this status would not be affected by the isolation of an Australian or European Bat Lyssavirus;
5. no imported *case* in carnivores has been confirmed outside a *quarantine station* for the past 6 months.

Article 8.11.3.

Recommendations for importation from rabies free countries

for domestic mammals, and wild mammals reared under confined conditions

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the animals:

1. showed no clinical sign of rabies on the day of shipment;
2. were kept since birth or for the 6 months prior to shipment in a rabies free country or were imported in conformity with the regulations stipulated in Articles 8.11.5., 8.11.6. or 8.11.7.[F]

[F] The Articles 8.11.6 and 8.11.7 cover importations of domestic ruminants, horses, pigs, laboratory reared rodents and lagomorphs, and lagomorphs or wild mammals reared under confined conditions.

Article 8.11.5.

Recommendations for importation from countries considered infected with rabies

for dogs and cats

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the animals:

1. showed no clinical sign of rabies within 48 hours of shipment;

AND EITHER

2. were identified by a permanent mark (such as a microchip) and their identification number shall be stated in the *certificate*; and

3. were vaccinated against rabies:

    a. not less than 6 months and not more than one year prior to shipment in the case of a primary vaccination, which should have been carried out when the animals were at least 3 months old;

    b. not more than one year prior to shipment in the case of a booster vaccination;

    c. with an inactivated virus vaccine or with a recombinant vaccine expressing the rabies virus glycoprotein; and

4. were subjected not less than 3 months and not more than 24 months prior to shipment to an antibody test as prescribed in the *Terrestrial Manual* with a positive result equivalent to at least 0.5 IU/ml;

OR

5. have not been vaccinated against rabies or do not meet all the conditions set out in points 2, 3 and 4 above; in such cases, the *importing country* may require the placing of the animals in a *quarantine station* located on its territory, in conformity with the conditions stipulated in its animal health legislation.

Article 8.11.9.

Recommendations for importation from countries considered infected with rabies

for frozen semen of dogs

Veterinary Authorities should require the presentation of an *international veterinary certificate* attesting that the donor animals showed no clinical sign of rabies during the 15 days following collection of the semen.

Options for the effective management of rabies in cats and dogs are:

42.3.1.1. Cats and dogs

- Since identification is essential to confirm that vaccination has been effective the following should be combined with the option selected:
-
    1) animals are identified with a microchip and their identification number shall be stated in the *certificate*;
    2) the implanted microchip is be read at the time of vaccination and the number recorded;

3) the microchip is read at the time of serological testing and the number recorded on the laboratory forms;
4) the microchip is read at the time of export from the country of origin and on arrival in New Zealand to identify the animal and verify laboratory and vaccination certificates.

If an imported animal does not meet the conditions set out in the points above, and in the selected option; then the animal should not be eligible for biosecurity clearance and should be directed to a quarantine facility.

Option 1.

Animals from a rabies free country (as defined in the *Code*) could be imported without restrictions.

Option 2.

Animals from rabies infected countries could be imported provided that:
1) they are free from clinical signs of rabies; and
2) they have been vaccinated against rabies with an inactivated virus vaccine or with a recombinant vaccine expressing the rabies virus glycoprotein
   a. not less than 6 months and not more than one year prior to shipment in the case of a primary vaccination, which should have been carried out when the animals were at least 3 months old; or
   b. not more than one year prior to shipment in the case of a booster vaccination; and
3) were subjected not less than 3 months and not more than 24 months prior to shipment to an antibody test as prescribed in the *Terrestrial Manual* with a positive result equivalent to at least 0.5 IU/ml.

NB. This option reflects the *Code* recommendations for the safe trade in cats and dogs.

Option 3.

If a higher level of protection than that achieved by application of international standards is considered necessary, post-arrival quarantine may be considered appropriate. The period of PAQ could be as short as 1 month or up to 6 months.

42.3.1.2. Canine semen

Dog semen could be imported provided it is accompanied by appropriate veterinary certification that the donor remained clinically healthy for 15 days after semen donation.

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43. Togaviridae

43.1. HAZARD IDENTIFICATION

43.1.1. Aetiological agent

The family Togaviridae includes two genera of which one *Alphavirus* contains species that infect cats and dogs. There are currently about 30 known alphaviruses (Atasheva et al 2007). Members of the genus are antigenetically related to each other and grouped into complexes based on serologic cross-reactivity. The eastern, Venezuelan, and western equine encephalitis, Semliki Forest and Barmah Forest complexes contain species that infect cats and dogs (Weaver et al 2000). The preliminary hazard list identifies Sindbis, Ross River, Getah, Barmah Forest and the eastern, western and Venezuelan equine encephalitis viruses that infect cats and dogs.

43.1.2. OIE List

Equine encephalomyelitis (eastern and western) and Venezuelan equine encephalomyelitis are included within the category of 'equine diseases' (OIE 2007).

43.1.3. New Zealand's status

Equine encephalitic viruses (eastern, western and Venezuelan) are listed as unwanted notifiable organisms (Ministry of Agriculture & Forestry 2008).

43.1.4. Epidemiology

Alphaviruses are transmitted biologically between vertebrates by mosquitoes and other haematophagous arthropods. They have an almost worldwide distribution. Each virus usually has a preferred mosquito vector. Most alphaviruses can infect a wide range of vertebrates. Many have different species of birds as their primary reservoir host (Weaver et al 2000).

Eastern and western equine encephalomyelitis

Equine encephalomyelitis viruses are restricted to the Americas. WEEV occurs in the western states of the USA and the western provinces of Canada, as well as in Mexico, Central and South America (Ostlund 2004a). EEEV occurs in the eastern and southern states of the USA, Quebec and Ontario in Canada, Mexico, the Caribbean, and Central and South America (Ostlund 2004a).

The main endemic cycles of transmission of both EEE and WEE involve passerine birds (amplifying host) and specific mosquitoes. The primary vector of WEEV is *Culex tarsalis*, and the primary vector of EEEV is *Culiseta melanura* (Radostits 2007a).

During epidemics a wide range of domestic and wild birds, mammals and reptiles become accidentally infected. In humans, EEE causes severe disease with approximately 65 % mortality and a high level of permanent sequelae in patients who survive. WEE is usually

mild in adults but more severe in children. Mortality is approximately 3-14 % (Ostlund 2004a). Horses and humans are dead-end hosts since virus titres in their blood are usually insufficient to infect mosquitoes (Radostits 2007a). Vertebrate species that are accidentally infected are dead-end hosts (Gibbs 2004).

Dogs, and occasionally cats, are susceptible to subclinical infection, but naturally occurring clinical disease is very rare. There are only two reports of naturally infected pups exhibiting clinical signs of encephalitis with EEEV (Greene & Baldwin 2006). No reports of natural infection with WEEV causing clinical signs in dogs or cats could be found.

There is no evidence to suggest that the viruses are transmitted in dog semen.

Venezuelan equine encephalomyelitis

VEE viruses infect horses, humans, birds, rodents, dogs, bats, rabbits, marsupials and non-human primates (Gibbs 2004). In humans infection is often fatal (Ostlund 2004b). Endemic VEE viruses exist in the tropical and subtropical Americas, including the Florida everglades, Mexico, Central America and northern South America (Ostlund 2004b). The ecological niche for VEEV is tropical wet forest areas with high water tables or open swampy areas with sunlit streams. These are the areas of the tropical Americas where rainfall is distributed throughout the year or areas permanently supplied with water (Ostlund 2004b).

During epidemics VEE is transmitted by many species of mosquito, while endemic VEE tends to cycle between *Culex* spp. mosquitoes and vertebrates (Radostits 2007b). Mammals are accidental hosts. Viraemia and seroconversion occurs without clinical illness in dogs and only one report of encephalitis affecting a puppy has been documented (Greene & Baldwin, 2006). It is not clear whether accidentally infected dogs and cats are capable of amplifying the virus to levels that are infectious for mosquitoes (Gibbs 2004).

There is no evidence to suggest that VEE is transmitted in dog semen.

Other Alphaviruses

Sindbis viruses are probably ubiquitous and are capable of infecting mosquitoes and vertebrates. Whataroa virus, a sindbis-like virus occurs in New Zealand (Miles 1973). These viruses circulate between ornithophilic mosquitoes and various bird species which are amplifying hosts (Kurkela et al 2005). Other vertebrates may be accidentally infected but infection in the cat and dog has not been documented.

Ross River virus (RRV), Barmah Forest virus and Getah virus are closely related, with RRV considered a subtype of Getah virus (Weaver et al 2000). Serological evidence of natural infection has been found in a large number of vertebrate species (mammals, birds and reptiles).

RRV and Barmah Forest viruses (BFV) are endemic in Australia, with kangaroos and wallabies probably acting as the major mammalian reservoirs (Greene & Baldwin 2006; Hills 1996).

Getah virus causes disease in horses and possibly neonatal pigs and is widely distributed throughout Southeast Asia. The pig is considered an important amplifying host for Getah virus (Timoney 2004). Serological evidence of Getah virus infection has been found in a large

number of vertebrate species (mammals, birds, and reptiles). However, there is no evidence to indicate that Getah virus is a human pathogen (Timoney 2004).

Although dogs and cats might be exposed naturally to these viruses, and could become infected, they are unlikely to be important reservoirs (Greene & Baldwin 2006). Dogs and cats were relatively resistant to experimental transmission of RRV and BFV by mosquitoes. Only 10 % seroconverted, and none developed viraemia or clinical signs and they were unable to infect mosquitoes (Boyd & Kay 2002; Russell 2002).

Infection appears to be of no consequence in cats and dogs with Sindbis virus, RRV, Getah and Barmah Forest viruses. No evidence could be found that they are important reservoir hosts or that they are transmitted in dog semen. Therefore these viruses are not considered to be hazards in the commodities.

43.1.5. Hazard identification conclusion

The equine encephalomyelitis viruses are zoonotic unwanted notifiable organisms and are therefore concluded to be potential hazards. Other *Alphaviruses* are not considered to be potential hazards in the commodities. There is no evidence to suggest that the viruses are transmitted in dog semen.

43.2. RISK ASSESSMENT

43.2.1. Entry assessment

Cats and dogs infected with EEEV or WEEV are dead-end hosts. They are aberrant hosts that do not develop viraemias that are sufficient to infect mosquitoes. No mosquito species which are important in EEEV and WEEV epidemiology (e.g. *Culiseta melanura* and *Culex tarsalis*) are present in New Zealand.

Entry is assessed to be negligible, since cats and dogs are dead-end hosts for EEE and WEE.

Dogs, and occasionally cats, are susceptible to subclinical infection with VEEV, but naturally occurring clinical disease appears to be uncommon. Therefore it would be possible to import subclinically VEEV infected cats or dogs from endemic regions.

Since cats and dogs from endemic regions may be subclinically infected with VEEV entry is assessed to be non-negligible.

43.2.2. Exposure assessment

During epidemics of VEEV, horses are important amplifying hosts that may develop high titres of viraemia capable of infecting mosquitoes for up to 5 days (Gibbs 2004).
No reports were found to suggest that a VEEV carrier status occurs in cats or dogs. They are not amplifying hosts and imported cats and dogs are unlikely to have viral titres of a sufficient duration or magnitude to infect susceptible mosquitoes.

VEE viruses are restricted to tropical and subtropical regions of the Americas. These viruses have never spread beyond Central America and adjacent parts of North and South America. In these endemic areas VEEV occurs year round as a result of evenly distributed rainfall and a

complex bird/mosquito/environment cycle that is unique to these areas. It is unlikely that endemic VEEV cycles would establish here, as such cycles have never established outside of the Americas, or in temperate areas of the Americas.

Since cats and dogs are not amplifying hosts of VEEV and these viruses have complex life cycles dependent on tropical/subtropical environments, the likelihood that these viruses would establish in New Zealand is assessed to be negligible.

43.2.3. Risk estimate

WEEV and EEEV entry assessment is negligible; therefore the risk is estimated to be negligible.

Exposure is assessed to be negligible for VEEV; therefore the risk is estimated to be negligible.

Since the risk of introducing equine encephalomyelitis viruses in imported cats and dogs has been estimated as negligible, risk management measures are not justified.

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MISCELLANEOUS SECTION

44. Canine Transmissible Venereal Tumour

44.1. HAZARD IDENTIFICATION

44.1.1. Aetiological agent

Canine transmissible venereal tumour (CTVT) is a contagious venereal tumour that affects members of the canine family (Mukaratirwa & Gruys 2003). The tumour cells are clonal in origin and it has been claimed that they themselves (rather than an agent such as a virus) are the contagious agent (VonHoldt & Ostrander 2006).

44.1.2. OIE List

Not listed.

44.1.3. New Zealand's status

CTVT is not listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008). However it is not recognised as an endemic syndrome in the New Zealand dog population and is considered exotic.

44.1.4. Epidemiology

CTVT is a common canine tumour in tropical and warm temperate countries, particularly in cities of developing countries where large populations of free-roaming stray dogs exist. It is also seen where dogs are intensively bred and there are infected studs or breeding bitches (Moulton 1990).

It has not been reported from Sweden, Denmark, the UK or North America. It has been reported from other parts of Europe and from South America, Japan, China, some African countries, Indonesia, India and Papua New Guinea. The incidence varies in the USA (Nielsen & Kennedy 1990). CTVT has been reported from several states in Australia, where clusters tend to occur in remote indigenous northern communities which have little or no access to veterinary services (Gallimore 2007). There has been one case recorded in New Zealand that originated from a dog imported from Western Samoa (Richards & Williamson 2004).

Transmission is by direct contact with tumorous growths, generally during coitus. The growth generally appears 2-6 months after mating. Experimentally the tumour cannot be produced with cells that have been frozen, only by transplantation of viable tumour cells. There is no evidence of transmission by artificial insemination.

CTVT presents as a cauliflower-like, pedunculated growth on the genitals, anus or nose of affected dogs (Nelson & Couto 1992). In most adult dogs the tumours regress spontaneously and the dog becomes immune to subsequent exposure. Regression is associated with the development of IgG in the sera of dogs after a period (40 days) of tumour growth (Nielsen & Kennedy, 1990). There is a high incidence of spontaneous regression, but if the dog is immunocompromised, tumours can grow large (>15cm) and often become ulcerated, friable and bleed easily (Nelson & Couto 1992; Mukaratirwa & Gruys 2003). Metastasis occurs rarely in the immunocompetent animal. Infection in immunocompromised dogs and experimentally infected neonatal puppies leads to metastasis and death (Fenton & Yang 1988).

44.1.5. Hazard identification conclusion

CTVT is an exotic, infectious tumour that causes disease in naïve animals; therefore, it is concluded to be a potential hazard.

44.2. RISK ASSESSMENT

44.2.1. Entry assessment

CTVT is primarily a tumour of sexually active intact dogs. Entry is assessed to be non-negligible for dogs coming from countries where the disease occurs.

The likelihood of entry for desexed and unmated dogs that are healthy is assessed to be negligible.

44.2.2. Exposure assessment

Any dog with CTVT would be potentially infectious to naïve New Zealand dogs that might directly contact the tumour through mating or socialisation. The tumour is most common in tropical and warm climates where stray dog populations serve as the reservoir of infection. Although the disease is unlikely to establish in the general New Zealand dog population because of dog control laws, it could persist in breeding kennels or the greyhound industry as has occurred in more temperate climates such as Ireland. The likelihood of establishment and exposure is assessed to be non-negligible.

44.2.3. Consequence assessment

Establishment of CTVT is likely to have a negligible effect on the general dog population. However, the introduction into a naïve breeding population might initially cause reproductive losses for kennels. No effects on the environment would be noticeable since dogs are the only species affected.

Since the disease could establish in dog-related industries such as greyhound racing and breeding kennels causing economic losses, the consequences of infection are assessed to be non-negligible.

44.2.4. Risk estimation

Since the exposure assessment for healthy unmated and spayed female and male dogs is assessed to be negligible, risk from such animals is estimated to be negligible. Therefore in this class of commodity CTVT is not a hazard and risk management measures are not justified.

Entry, exposure and consequence assessments are assessed to be non-negligible in dogs that are sexually active. As a result the risk estimate for CTVT is non-negligible in these animals and it is classified as a hazard. Therefore risk management measures can be justified.

44.3. RISK MANAGEMENT

44.3.1. Options

Diagnosis of CTVT is strongly suspected on the basis of the physical appearance of the tumour which is almost always found on the external genitalia. It is confirmed by exfoliative cytology or fine needle aspirate.

Since infection is clinically obvious based on physical appearance of the tumour on the external genitalia, veterinary examination prior to travel could markedly decrease the likelihood that CTVT is introduced.

The available options for excluding CTVT, in ascending order of likely efficacy, are:

Option 1.

General veterinary examination of the dog pre-export could be performed by a veterinarian within one week of travel; with the dog certified to have no lesions suggestive of CTVT.

Option 2.

Specific veterinary examination of genitalia of bitches by vaginal speculum in females and examination of the penis and prepuce in males could be done to ensure dogs are free of CTVT within 3 days of travel. Dogs showing evidence of CTVT could be disqualified from travel.

Option 3.

Dogs to be imported could be desexed animals or entire adults that are certified as not having been mated in the 8 months prior to export or for their entire lives.

Option 4.

Dogs for import could be held in quarantine for 6 months immediately before shipment.

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45. Exotic Strain Variations of the Major Endemic Diseases

45.1. HAZARD IDENTIFICATION

45.1.1. Aetiological agent

The major infectious agents found world-wide that have core[G] vaccination protocols universally recommended for them are: canine and feline parvovirus, feline herpesvirus and calicivirus, canine distemper, infectious canine hepatitis and rabies (Greene et al 2006).

45.1.2. OIE List

Rabies is listed.

45.1.3. New Zealand's status

Apart from rabies, none are listed on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008). They are endemic and outbreaks may occur anywhere in New Zealand in susceptible animals. However, more pathogenic exotic strain variations are known to exist overseas, in particular for canine parvovirus.

45.1.4. Epidemiology

Vaccination is routinely practised to protect animal populations from the effects of these potentially fatal infections. There can be no certainty about what viral strains are present in a country because the strains may change because of mutations, antigenic drift or new introductions.

Canine parvoviral enteritis is one of the most common infectious diseases of dogs. It is highly contagious and often fatal caused by CPV-2, a DNA virus (McCaw & Hoskins 2006). Since its emergence in the late 1970s, a number of strains of CPV-2 have emerged. The original strain (now thought to be extinct) has been replaced by type 2a, type 2b and type 2c (McCaw & Hoskins 2006). The most recent strain to evolve (type 2c) was isolated in Italy in 2000 and is now widespread in Europe and the USA (May 2009). This strain has not been identified in the UK (Davies 2008) or New Zealand. The newer strains of CPV-2 cause a more rapid progression of enteritis with vomiting and haemorrhagic diarrhoea than the original strain which leads to dehydration and death as early as two days after the onset of clinical signs (McCaw & Hoskins 2006). Commercially prepared attenuated live and inactivated CPV-2 vaccines are available. Vaccines based on the original CPV-2 confer protection against the new strains (Davies 2008).

[G] Core vaccines are defined as those that are appropriate to provide protection in most animals against diseases that pose a risk of severe disease because the pathogens are virulent, highly contagious, and widely distributed. Core vaccines are considered to be highly efficacious, to have benefit-risk ratios high enough to warrant their general use.

Feline parvovirus infection is caused by a virus called feline panleukopenia virus. It is closely related to canine parvovirus. Vaccination has been credited as the most important factor in reducing the incidence of this disease in cats (Greene & Addie 2006). This is because the vaccine confers protection against all the strains of feline parvovirus which are serologically homogeneous.

Despite minor genetic variation due to antigenic drift, canine distemper virus (CDV) isolates are also serologically homogeneous. While there are strain differences in pathogenicity, prevention of disease from all strains is possible through vaccination (Greene & Appel 2006).

Infectious canine hepatitis is a disease of dogs caused by canine adenovirus (CAV-1) which is serologically homogeneous and is also serologically closely related to the canine respiratory virus CAV-2. Vaccination, most commonly using CAV-2, has effectively controlled and practically eliminated this potentially fatal disease from the domestic dog population worldwide (Greene 2006).

There is little strain variation in feline herpesviruses, and since all strains belong to one serogroup vaccination is considered to provide the same level of protection against all strains. There are no reports of exotic strains of greater pathogenicity than those present in this country.

In the case of feline calicivirus, while there are a large number of serologically different strains of the virus, which limits the level of cross-protection achieved by vaccination (Gaskell et al 2006), none of these strains have been reported to be significantly more pathogenic than strains present in this country.

45.1.5. Hazard Identification conclusion

There are no reports in other countries of significantly more pathogenic strains of feline herpesvirus, feline calicivirus or feline parvovirus.

Infectious canine hepatitis has been practically eliminated from the domestic dog population worldwide.

While strain variation in pathogenicity has been reported for canine parvovirus and canine distemper virus, in both cases vaccines that are effective against all strains are available and are widely used. Therefore it is unlikely that exotic strains of these viruses would be associated with the commodity.

Therefore exotic strains of these viruses are not considered to be potential hazards in the commodity.

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46. Fungal and Algal Infections

46.1. HAZARD IDENTIFICATION

46.1.1. Aetiological agents

The fungi listed in the hazard list are: *Aspergillus deflectus*, *Aspergillus flavipes*, *Aspergillus terreus*, *Blastomyces (Ajellomyces) dermatitidis*, *Coccidioides immitis*, *Histoplasma capsulatum*, *Pythium insidiosum*, *Rhinosporidium seeberi*, *Trichosporon beigelii* and *Trichosporon pullulans*.

46.1.2. OIE List

None are listed.

46.1.3. New Zealand's status

None of the fungal species listed are found on the Unwanted Organisms Register (Ministry of Agriculture & Forestry 2008).

46.1.4. Epidemiology

Over 100,000 fungal species have been identified, but only 150 are known to cause disease in animals and humans. Generally they are widely distributed in the world and occur where environmental conditions are suitable for a particular species. Many fungal species have been identified in New Zealand, but there are probably many endemic species that have not yet been reported. With the exception of the dermatophytes, fungi are not primary pathogens but are opportunistic or secondary invaders transmitted from environmental sources to animals. Infected animals are generally not contagious and infection from animal to animal does not occur or is rare (Acha & Szyfres 1991b; Picard & Vismer 2004; Various Authors 2006).

No mention of any fungal disease occurs in any of MAFBNZ's Import Health Standards (IHSs) or Overseas Market Access Requirements (OMARS), thus indicating that they are not considered by MAFBNZ or any of our trading partners to be important in the movement of animals from country to country. Ten species of fungi included in the hazard list are recorded as fungi that were not known to occur in New Zealand (Section 44.1.1). However, the Landcare Research checklist of fungi (Landcare Research 2007) lists *Aspergillus flavipes*, *Aspergillus terreus*, *Histoplasma capsulatum*, *Pythium insidiosum* and *Trichosporon pullulan* as present in New Zealand and therefore these are not potential hazards in the commodity.

Aspergillus deflectus is an opportunistic pathogen and causes rare, sporadic cases of disease in dogs (Jang et al 1986; Kahler et al 1990; Robinson et al 2000). It may be associated with special predisposing conditions in infected hosts. Dogs with nasal aspergillosis or

disseminated aspergillosis may have an underlying immunodeficiency (Day 2006; Mathews & Sharp 2006).

There are over 200 species of *Aspergillus* listed in the Landcare database (Landcare Research 2007) and many of these occur in New Zealand and the fact that *Aspergillus deflectus* is not listed does not necessarily mean that it is not present. Given the wide distribution of the *Aspergillus* spp. it is possible that it would be found if specifically looked for. Since aspergillosis caused by *Aspergillus deflectus* is rare, it is unlikely to be introduced in the commodity and because it causes a non-contagious disease, the organism would not be transmitted to susceptible animals in New Zealand. Therefore, the likelihood of introducing *Aspergillus deflectus* in the commodity is negligible.

Blastomyces dermatitidis causes blastomycosis, a systemic mycotic infection that is principally a disease occurring in south eastern areas of the USA. It occurs less commonly in Africa, India, Europe and Central America (Acha & Szyfres 1991a; Lengendre 2006). It most commonly infects dogs and humans and, more rarely, cats and other animals. It most commonly manifests as a respiratory infection.

The organism is dimorphic and grows as a spore forming mycelium in the environment and as yeast in tissues at body temperature. The disease is transmitted by inhalation of spores from the environment but the yeast form is not infectious. Therefore, infected animals are not contagious (Acha & Szyfres 1991a; Lengendre 2006). Since infected animals are not contagious the likelihood of the organism establishing is negligible.

Coccidioides immitis has a limited distribution. It occurs only in a specific soil type with suitable climatic conditions that are confined to southwestern areas of the USA, Mexico and some areas in South America (Acha & Szyfres 1991; Greene 2006). The growth of hyphae in the soil results in the production of arthroconidia which are released into the air and are the infectious form of the fungus. Arthroconidia are inhaled by host animals and may cause disease most often in humans and dogs but also other animals including cats. Respiratory infection results in mild or subclinical infections in most cases but sometimes causes more severe respiratory disease. The organism can develop into spherules which become filled with endospores that are disseminated to other parts of the body sometimes resulting in granulomatous lesions. Clinical signs vary depending on the organs infected. Infectious arthroconidia only develop in the soil and infected animals are not typically contagious (Acha & Szyfres 1991; Greene 2006). Since the organism is confined to certain areas of a typical soil type and climate; it would not establish in New Zealand and infected animals would not be contagious. Therefore the likelihood that the organism would be introduced and establish in New Zealand is negligible.

Rhinosporidium seeberi is not a classic fungus but a member of an aquatic protistan clade of organisms (Fredericks et al 2000; Herr et al 1999). The name Mesomycetozoa has been proposed for this group of organisms, indicating that they are between animals and fungi (Herr et al 1999).

Rhinosporidiosis has been described in several countries and occurs most commonly in the tropics especially in India and Sri Lanka. It is an uncommon disease of humans, and more rarely, animals. The exact mechanism of transmission is unknown but it is generally believed to be transmitted from the environment, possibly from water. Evidence of spread from human to human or animal to human has never been recorded (Arseculeratne 2002). Since the disease is not transmitted by infected animals the likelihood that it would establish is negligible.

Trichosporon beigelii. *Trichosporon* species are soil inhabitants and common colonisers of human skin and gastrointestinal tracts. It is therefore, highly likely that they are also colonisers of animal skin and gastrointestinal tracts. They are not primary pathogens but are opportunistic life-threatening pathogens in granulocytopenic and immunosuppressed hosts (Erer et al 2000; Greene & Chandler 2006; Sugita et al 1998). There is confusion about the nomenclature of the species. One worker concluded after analysing PCR-amplified fragments of 17 species and 5 varieties in the genus that the following 6 medically relevant species should be recognised: *Trichosporon asahii*, *Trichosporon inkin*, *Trichosporon asteroids*, *Trichosporon cutaneum*, *Trichosporon mucoides* and *Trichosporon ovoides* (Sugita et al 1999). *Trichosporon beigelii* would become a redundant species name. Six of the proposed species names are already listed in the Landcare database as occurring in New Zealand (Landcare-Research 2007). It is not clear which species are truly endemic or exotic and which species have been incorrectly classified in the past.

In addition, *Trichosporon* spp. are considered to be environmental organisms with a world-wide distribution, and are probably introduced daily on the skin and in the gastrointestinal tracts of animals and humans entering New Zealand. There is no evidence that infected animals are contagious to other animals or humans. Therefore there is no justification for classifying a single *Trichsporon* sp. as an exotic organism that should require sanitary measures to exclude them.

46.1.5. Hazard identification conclusion

All the fungal species discussed above are opportunistic pathogens that are generally widely distributed in the world. Animals infected with these fungi are not infectious and it is concluded that fungi are not potential hazards in the commodities.

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APPENDIX 1 THE PRELIMINARY HAZARD LIST

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
EXOTIC ORGANISMS						
List A and B diseases						
Viruses						
African horse sickness virus	African horse sickness (AHS)	Dog		No		(1)
	Avian Influenza	Cat				Added 2005
Bluetongue virus	Bluetongue (BT)	Dog		No		(2, 3)
Eastern equine encephalomyelitis virus	Eastern equine encephalomyelitis (EEE)	Dog		No		(4)
Japanese encephalitis virus	Japanese encephalitis	Dog	Yes	No		(4, 5)
Newcastle disease virus	Newcastle disease (Birds) CNS disease (Cats)	Cats/Dogs		Avirulent strains of APMV-1 have been identified in New Zealand. None of which fit the OIE definition of New castle disease(6).		(4, 7)
Pseudorabies virus (PRV)	Aujeszky's disease (Pseudorabies)	Cat/Dog		No, eradicated		(4, 8-11)
Rabies virus	Rabies	Cat/Dog	Yes	No		(4, 11)
Rift Valley fever virus	Rift Valley fever	Cat/Dog	Yes	No		(12)
Transmissible gastroenteritis virus	Transmissible gastroenteritis (TGE)	Cats/Dog		No		(13-15)
Venezuelan equine encephalomyelitis virus (Venezuelan equine encephalitis virus / Everglades virus)	Venezuelan equine encephalomyelitis(VEE) (Venezuelan equine encephalitis)	Dog		No		(4, 16)
Western equine encephalomyelitis virus	Western equine encephalomyelitis (WEE)	Dog		No		(4, 5)
Yellow fever virus		Cat				(4)
Bacteria						
Bacillus anthracis	Anthrax	Cat/Dog	Yes	1954(17). Exotic.		(4)
Brucella abortus *Brucella suis* *Brucella melitensis*	Bovine brucellosis Porcine brucellosis Sheep and goat brucellosis	Dog Dog	Yes	No		(4)
Cowdria ruminantium	Heartwater	Dog				(4)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Coxiella burnetii	Q Fever	Cat/Dog	Yes	No		(4, 18)
Francisella tularensis	Tularemia	Cat/Dog	Yes	No		(4)
Leishmania spp. • *Leishmania infantum* • *Leishmania donovani/ Leishmania chagasi* • *Leishmania braziliensis* • *Leishmania mexicana* • *Leishmania tropica*	Leishmaniasis	Cat/Dog	Yes	No		(4, 19-21)
Leptospira spp. • *Leptospira kirschneri serovar grippotyphosa* • *Leptospira interrogans serovar canicola* • *Leptospira interrogans serovar icterohaemorrhagiae* • *Leptospira interrogans serovar bataviae* • *Leptospira bratslava*	Canine leptospirosis	Cat/Dog	Yes	No		(4, 11, 22)
Pseudomonas mallei	Glanders	Cat/Dog	Yes	No		(4)
Cestodes						
Echinococcus multilocularis		Cat/Dog	Yes	No		(19)
Protozoan						
Trypanosoma evansi	Surra	Dog (rare in cats)		No		(19, 23)
Trypanosoma spp. • *Trypanosoma congolense* • *Trypanosoma brucei* • *Trypanosoma gambiense* • *Trypanosoma rangeli*	Trypanosomiosis	Cat/Dog Cat/Dog Cat Cat	Yes(Primarily)	No		(4, 19, 20)
Flies						
Cochliomyia hominivorax	New World Screwworm	Dog(Rare in Cats)	Yes	No		(19, 23)
Chrysomyia bezziana	Old World Screwworm	Cat/Dog		No		(19, 23) (24)
Unlisted diseases						
Viruses						
	Canine acidophil cell hepatitis	Dog		Has not been reported.		(4)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
				Exotic.		
Borna disease virus	Borna disease	Cat		No		(4, 25)
Cowpox virus	Feline cowpox infection	Cat	Yes	No		(4)
Hantavirus	Hantavirus infection	Cat	Yes	No		(4, 26)
Hendra virus (HeV) (Equine morbillivirus (EMV)	Hendra virus infection	Cat	Yes	No		(27, 28)
Influenza A, B and C	The flu virus Common cold	Dog	Yes	Influenza A, B and C present in New Zealand(29, 30) Strain differences recognised.		(4)
California serogroup: LaCrosse virus Showshore hare virus (SSH) Jamestown Canyon virus (JCV)	LaCrosse virus encephalitis	Dog Cat Cat/Dog		No		(31) (4, 32)
Louping-ill virus	Louping-ill	Dog		No		(4, 33, 34)
Murry Valley encephalitis virus Sindbis virus Getah virus Ross River virus		Dog Dog Dog Cat/Dog		No		(5, 35) (35)
Nipah virus	Nipah virus infection	Cat	Yes	No		(36)
Phocine distemper virus	Phocine distemper	Dog		No		(37)
Powassan virus	Powassan encephalitis	Cat/Dog	Yes	No		(4, 32)
St Louis encephalitis virus	St Louis encephalitis	Cat/dog		No		(4, 5)
Tenshaw virus	Tenshaw	Cat/Dog		No		(4)
Tickborne encephalitis virus	Tickborne encephalitis (TBE)	Dog	Yes			(38)
Wesselsbron virus	Wesselsbron disease (WSL)	Dog		No		(4, 39)
West nile virus	West Nile virus infection	Dog	Yes	No		(40)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Ehrlichia canis *Ehrlichia ewingii* *Ehrlichia chaffeensis* *Ehrlichia phagocytophila* *Ehrlichia microti (?Human granulocytic ehrlichiosis agent)*	Ehrlichiosis	Dog Dog Dog (experimentally) Dog(experimentally) Dog				(4)
Anaerobiospirillum spp.		Cat/Dog (part of normal flora)	Yes	Need to determine if already present in NZ		(41)
Anaplasma phagocytophila (New name for *Ehrlichia equi)*		Dog				(4)
Bartonella vinsonii subsp berkhoffi	Bartonellosis	Dog		No		(4, 42, 43)
Borrelia spp. • *Borrelia burgdorferi* • *Borrelia afzelii* • *Borrelia japonica* • *Borrelia garinii*	Lyme disease (Lyme borreliosis)	Dog Dog				(4, 11, 44)
Brucella canis	Canine brucellosis	Dog	Yes			(4)
Burkholderia pseudomallei	Melioidosis	Cat/Dog	Yes	No		(4)
Ehrlichia platys	Infectious cyclicthrombocytopenia	Dog				(4, 45)
Ehrlichia risticii (? Now *Neorickettsia risticii*)	Potomac horse fever	Dog				(4)
Exotic *Salmonella spp.*		Cat/Dog	Yes			
Neorickettsia helminthoeca	Neorickettsiosis (Salmon posioning complex/disease) Elokomin fluke fever	Dog				(4)
Rickettsia conorii	Boutonneuse/Mediterranean spotted fever	Dog				(4, 23)
Rickettsia rickettsii *Rickettsia akari* Others of RMSF group	Rocky mountain spotted fever	Dog	Yes			(4, 46)
Rickettsia typhi	Murine typhus	Cat				(4)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Rickettsia prowazekii *Rickettsia felis*						
Yersinia pestis	Plague	Cat/Dog	Yes			(4)
Fungi						
Aspergillus deflectus *Aspergillus flavipes* *Aspergillus terreus*	Aspergillosis	Dog				(4)
Blastomyces (Ajellomyces) *dermatitidis*	Blastomycosis	Cat/Dog	Yes			(4)
Coccidioides immitis	Coccidioidomycosis	Cat/Dog	Yes			(4)
Histoplasma capsulatum	Histoplasmosis	Cat/Dog				(4)
Pythium insidiosum	Pythiosis	Cat/Dog				(4)
Rhinosporidium seeberi	Rhinosporidiosis	Dog	Yes			(4)
Trichosporon beigelii *Trichosporon pullulans*	Trichosporonosis	Cat	Yes	Maybe present in New Zealand, need to confirm.		(4)
Mycoplasma						
Mycoplasma spp. • *Mycoplasma cynos* • *Mycoplasma spumans* • *Mycoplasma gatae*	Mycoplasmosis	Cat/Dog				(4)
Nematodes						
Ancylostoma spp. • *Ancylostoma braziliense* • *Ancylostoma ceylanicum*		Cat/Dog Cat/Dog	Yes Yes	No No		(19) (47)
• *Angiostrongylus* (Parastrongylus) *cantonensis* • *Angiostrongylus vasorum* (French heartworm)	Angiostrongylosis	Dog Dog	Yes			(48, 49)
• *Brugia malayi* • *Brugia beaveri* • *Brugia pahangi*	Brugia filariasis	Cat Cat (experimental only)	Yes			(19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
• *Brugia patei*		Cat/Dog Cat/Dog				
Capillaria spp. • *Capillaria plica* (*Pearsonema plica*) • *Capillaria feliscati* (*Pearsonema felicati*)		Cat/Dog Cat				(50)
Crenosoma vulpis	Lungworm	Dog				(48, 51)
Cyathospirura seurati (*Cyathospirura dasyurids*)		Cat				(19)
Cylicospirura sp. • *Cylicospirura heydoni* • *Cylicospirura felineus* • *Cylicospirura subaequalis*		Cat Cat/Dog Cat				(19) (52)
Dioctophyma renale	Kidney worm	Dog				(53)
• *Dirofilaria repens* • *Dirofilaria immitis* (Heartworm)	Dirofilariasis	Cat/Dog Cat/Dog	yes yes			(19, 23, 54) (48, 53)
Dracunculus spp. • *Dracunculus medinensis* • *Dracunculus insignis*		Cat Dog				(19, 20, 53, 55)
• *Filaroides* (*Andersonstrongylus*) *milksi* • *Filaroides hirthi*		Dog Dog				(48, 56-58)
Gnathostoma spinigerum	Gnathostomiasis	Cat	yes			(19, 50, 52)
Gurltia paralysans		Cat				(19)
Lagochilascaris spp. • *Lagochilascaris minor* • *Lagochilascaris major*		Cat (experimental only) Cat	yes			(19)
Mammomonogamus spp. • *Mammomonogamus ierei* • *Mammomonoganus auris*		Cat Cat	yes			(19)
Physaloptera spp. • *Physaloptera praeputialis* • *Physaloptera rara* (*Physaloptera felidis*) • *Physaloptera canis*		Cat Cat Dog	yes			(19, 47, 53, 59, 60) (52)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Spirocera lupi	Canine spirocercosis	Dog				(47, 52, 53)
Spirura rytipleurites		Cat				(19)
Strongyloides spp. • *Strongyloides planiceps* (*Strongyloides cati*) • *Strongyloides felis* • *Strongyloides stercoralis* • *Strongyloides tumefaciens*		Cat/Dog Cat Dog (cat-experimental only) Cat	 Yes			(19, 50, 53)
Thelazia spp. • *Thelazia callipaeda* • *Thelazia californiensis*		 Cat/Dog Cat/Dog	 Yes Yes			(19, 53, 61)
Toxocara spp. • *Toxocara malaysiensis* • *Toxocara mystax*	Toxocariasis	 Cat Cat	 Yes			(56, 62)
Trichuris felis (*Trichuirs serrata, Trichuris campanula*)		Cat				(19)
Acanthocephalans						
Macracanthorhynchus ingens		Dogs				(63)
• *Oncicola canis* • *Oncicola pomatostomi*		Dog Cat				(19, 64)
Trematodes						
Alaria spp. • *Alaria canis* • *Alaria alata* • *Alaria marcianae* • *Alaria arisaemoides* • *Alaria nasuae*		 Dog Dog Cat Cat/Dog Dog	 yes			(19, 53, 65-68)
Amphimerus pseudofelineus (*Opisthorchis guayaquilensis*)		Cat/Dog	yes			(19)
Apophallus donicus(m) (*Apophallus venustus*)		Cat (rarely dogs)				(19)
Clonorchis sinensis	Clonorchiasis	Cat/Dog	yes			(19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Echinostoma malayanum (*Artyfectinostomum sufrartyfex*)		Cat	yes			(19)
Eurytrema spp. • *Eurytrema* (Concinnum) *procyonis*		 Cat				(19)
Haplorchis yokogawai		Cat/Dog	yes			(19)
Heterobilharzia americana	North American canine schistosomiasis	Dog	Cercarial dermatitis			(69-72)
46.1.5.1. Heterophyes heterophyes		Cat	yes			(19)
Heterophyopsis continua		Cat/Dog	yes			(19, 73) (may not be disease causing)
Metagonimus spp. • *Metagonimus yokogawai* • *Metagonimus tatahashii*		 Cat/Dog Cat/Dog	 yes yes			(19)
Metorchis spp. • *Metorchis albidus* • *Metorchis conjunctus* • *Metorchis orientalis*		 Cat/Dog Cat/Dog Cat/Dog	 yes			(19)
Nanophyetus (*Troglotrema*) *salmincola*		Cat/Dog	yes			(19, 53)
Opisthorchis spp. • *Opisthorchis felineus* (*Opisthorchis tenuicollis*) • *Opisthorchis viverrini*	Opisthorchiasis	 Cat/Dog Cat	 Yes Yes			(19) (74)
Paragonimus spp. • *Paragonimus westermani* • *Paragonimus kellicotti* • *Paragonimus pulmonalis* • *Paragonimus miyazakii* • *Paragonimus heterotremus* • *Paragonimus ohirai* • *Paragonimus peruvianus* • *Paragonimus skrjabini* • *Paragonimus mexicanus*	Paragonimiasis	 Cat/Dog Cat/Dog Cat/Dog Cat/Dog Cat/Dog Cat Cat/Dog Cat Cat/Dog	 Yes Yes Yes Yes Yes Yes Yes Yes Yes			(19, 53, 75)
Pharyngostomum cordatum		Cat				(19, 73)
Platynososum spp.		Cat				(19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Platynososum concinnum						
Schistosoma japonicum		Cat/Dog	Yes			(19, 20)
Troglotrema mustelae		Cat				(19)
Cestodes						
Anoplotaemia dasyuri		Cat/Dog				(76)
Diphyllobothrium latum	Diphyllobothriasis	Cat/Dog	yes			(19, 50, 53)
Diplopylidium spp.		Cat				(19)
Echinococcus spp. • *Echinococcus granulosus* • *Echinococcus vogeli* • *Echinococcus oligarthus*	Echinococcosis/hydatidosis	Dog Bush dog* Felids* * Need to access is appropriate to keep included	Yes	Provisional freedom declared for *Echinococcus granulosus* (77)		(11, 47, 53, 78)
Joyeuxiella spp. • *Joyeuxiella pasqualei* • *Joyeuxiella echinorhynchoides* • *Joyeuxiella fuhrmanni*		Cat/Dog Cat Cat				(19)
Mesocestoides lineatus(*Mesocestoides variabilis*)		Cat/Dog	Yes			(19, 66)
Spirometra spp. • *Spirometra erinacei* (*Spirometra mansoni*) • *Spirometra mansonoides*		Cat Cat/Dog	Yes Yes			(19, 50, 53)
Taenia spp. • *Taenia crassiceps* • *Taenia krabbei*		Dog Dog				(53, 56, 79)
Fleas						
Archaeopsylla erinacei		Cat/Dog				(80, 81)
Chaetopsylla globiceps		Cat/Dog				(81)
Echinophaga gallinacea		Cat				(19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Hystrichopsylla talpae		Cat/Dog				(81)
Nosopsyllus fasciatus		Cat/Dog				(81)
Paraceras melis		Dog				(80, 81)
Spilopsyllus cuniculi		Cat				(80, 81)
Tunga penetrans		Dog				(20)
Xenopsylla cheopis		Dog				(82)
Other flea spp.						
Lice						
Heterodoxus spiniger		Dog				(83)
Mites						
Lynxacarus radovskyi	Fur mite	Cat				(19, 53)
Pneumonyssus caninum	Nasal mite	Dog				(84)
Trombicoulid spp. • *Trombicula autumnalis* (*Neotrombicula autumnalis*)		Cat	Yes			(19)
Protozoan						
Babesia spp. • *Babesia herpailuri* • *Babesia gibsoni (Asian, Californian, and Spanish strains)* • *Babesia felis* • *Babesia canis canis* • *Babesia cati* • *Babesia canis vogeli* • *Babesia canis rossi* • *Babesia pantherae*	Babesiosis	Cat Dog Cat Dog Cat Dog Dog Cat				(4, 11, 19, 85, 86)
Besnoitia darlingi		Cat				(19)
Caryospora bigenetica		Dog				(87)
Cytauxzoon felis	Feline cytauxzoonosis	Cat				(4, 19)
Hammondia hammondi		Cat				(4, 19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Hammondia pardalis		Cat				
Hepatozoon canis *Hepatozoon americanum*	Hepatozoonosis American canine hepatozoonosis	Cat/Dog Dog				(4, 19, 88)
Isospora spp. • *Isospora burrowsi* • *Isospora neorivolta*		Dog Cat				(4, 11)
Pentatrichomonas hominis		Cat/Dog	Yes		No	(4, 11, 19)Currently no evidence is pathogenic
Sarcocystis spp. • *Sarcocystis buffalonis* • *Sarcocystis cameli* • *Sarcocystis cymruensis* • *Sarcocystis equicanis* • *Sarcocystis leporum* • *Sarcocystis levinei* • *Sarcocystis miescheriana* • *Sarcocystis suicanis* • *Sarcocystis bertrami* • *Sarcocystis odoi* • *Sarcocystis porceifelis* • *Sarcocystis moule*		Cat Dog Cat Dog Cat Dog Dog Dog Dog Cat Cat Cat				(19, 89-94)
Tetratrichomonas felistomae		Cat				(19)
Trypanosoma cruzi	Chagas' disease (American trypanosomiasis)	Cat/Dog	Yes			(4, 11, 19)
Ticks						
Amblyomma spp. • *Amblyomma americanum* • *Amblyomma maculatum* • *Amblyomma cajennense* • *Amblyomma ovale* • *Amblyomma triguttatum spp.*		Dog Dog Dog Dog	Yes Yes Yes Yes			(19, 95) (96) (97)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Boophilus microplus		Dog				(96)
Dermacentor spp. • *Dermacentor andersoni* • *Dermacentor reticulatus* • *Dermacentor variabilis* • *Dermacentor occidentalis*		 Dog Dog Cat/Dog Dog	 Yes			(4, 11, 95, 97, 98)
Haemaphysalis spp. • *Haemaphysalis punctata* • *Haemaphysalis leachi* • *Haemaphysalis bancrofti*		 Dog Dog/Cat				(83, 98-100)
Ixodes spp. • *Ixodes scapularis* (*dammini*) • *Ixodes pacificus* • *Ixodes ricinus* • *Ixodes holocyclus* • *Ixodes persulcatus* • *Ixodes cornuatus* • *Ixodes hexagonus* • *Ixodes canisuga*		 Dog Dog Cat/Dog Cat/Dog Cat/Dog Cat/Dog	 Yes Yes Yes Yes			(19, 23, 50, 97-100)
Ornithodoros spp. • *Ornithodoros talaje* • *Ornithodoros puertoriciensis*		 Cat/Dog	 yes			(19)
Otobius spp. *Otobius megnini*		 Cat/Dog				(19)
Rhipicephalus spp. • *Rhipicephalus sanguineus* • *Rhipicephalus longus*		 Cat/Dog Dog				(53, 83, 96, 98, 99)
Leech						
Myxobdella annandalei		Dog				(23)
Pentastomid						
Armillifer armillatus		Cat				(19)
Insects e.g.flies						
Dermatobia hominis	Tropical warble fly	Cat/Dog	Yes			(19)
Lucilia caesar (Blow fly)		Cat	Yes			(19)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Cordylobia anthropophaga	African thumbu fly	Cat/Dog	Yes			(19, 23, 83)
Cuterebra sp.	Rodent bot fly	Cat/Dog	Yes			(19) (53)
Wohlfahrtia *spp.* • *Wohlfahrtia vigil*	Flesh flies Sacrophagid flies	Cat/Dog	Yes			(19)
Amebas						
Acanthamoeba culbertsoni	Acanthamoeba	Dog				(4)
Prion diseases						
	Feline spongiform encephalopathy (FSE)	Cat				(101)
Other						
	Transmissible venereal tumour (TVT)	Dog				Added 2005
ENDEMIC						
Viruses						
	Parapoxvirus in cats	Cat		Yes(102, 103)		
Canine adenovirus type 1	Infectious canine hepatitis	Dog		Yes(104-106)	No	(4, 11)
Canine adenovirus type 2 (Infectious laryngotracheitis virus)	Infectious canine tracheobronchitis	Dog		Yes(107)	No	(4, 11)
Canine coronavirus	Canine viral enteritis	Dog		Yes	No	(11)
Canine distemper virus	Canine distemper	Dog		Yes(108-110)	No	(4, 11)
Canine herpes virus		Dog		Yes(111)	No	(4, 11)
Canine parvovirus type 2	Parvoviral enteritis Canine viral enteritis	Dog		Yes(108, 112, 113)	No	(4, 11)
Canine Parvovirus type1	Minute virus of canines (MVC)	Dog		Has not been isolated, but is considered to be present in New Zealand.	No	(4)
Canine type 2 parainfluenza virus	Infectious canine tracheobronchitis	Dog		Yes(108)	No	(4, 11)
Enterovirus (human)	Enterovirus infection			Yes(29, 114)	No	(4)
Feline astroviruses		Cat		Yes(115, 116)	No	(4, 11)
Feline calicivirus	Feline respiratory infection	Cat		Yes(116-118)	No	(4, 11)
Feline coronavirus		Cat		Yes(116, 119, 120)	No	(4, 11)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
• Feline enteric coronavirus • Feline infectious peritonitis virus	Corona virus enteritis Feline infectious peritonitis (FIP)					
Feline herpes virus-1 (Feline rhinotracheitis virus)	Feline rhinotracheitis Feline respiratory disease	Cat		Yes(116, 118)	No	(11)
Feline immunodeficiency virus (FIV)	FIV "Feline AIDS"	Cat		Yes(116, 117, 121)	No	(4, 11)
Feline leukaemia virus (FeLV) (Individuals may develop Feline sarcoma from FeLV infection)	Feline leukaemia Viral fibrosarcoma	Cat		Yes(116, 122, 123)	No	(4, 11)
Feline panleukopaenia virus	Feline viral enteritis	Cat		Yes(116)	No	(4, 11)
Feline spumavirus/ Feline foamy virus/ Feline syncytium forming virus		Cat		Yes(124)		(125)
Mumps virus	Mumps	Dog (inconclusive evidence)		Yes(126)	No	(4)
Papillomavirus	Canine viral papillomatosis Feline viral papilllomatosis	Dog Cat		Papillomas occur in dogs in New Zealand(127). Viruses are considered to be present in New Zealand, although not isolated(128).	No	(4)
Rotavirus	Canine viral enteritis Feline viral enteritis	Dog/Cat		Yes(129)	No	(4, 11)
Bacteria						
Bartonella henselae *Bartonella carridgeiae*	Cat scratch disease	Cat	Yes	Yes(130) Bartonella carridgeiae has not been Reported, but are considered to be present. Cat scratch disease does occur in New Zealand.	No	(42)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Bordetella bronchiseptica	Canine infectious bronchitis (Kennel cough) Feline respiratory disease	Cat/Dog		Yes(131)	No	(11)
Campylobacter spp. • *Campylobacter coli* • *Campylobacter jejuni* • *Campylobacter upsaliensis*	Campylobacteriosis	Cat/Dog		Yes(106, 132, 133)	No	(4, 11)
Chlamydia psittaci (*Chlamydophila felis*)	Feline chlamydiosis (Feline Pneumonitis)	Cat		Yes(117)	No	(4)
Clostridium botulinum	Botulism	Dog		Yes(134)	No	(11)
• *Clostridium perfringens* (*welchii*) • *Clostridium sordellii* • *Clostridium chauvoei* • *Clostridium difficile* • *Clostridium septicum*	Enteritis/Gas gangrene	Cat/Dog		Yes(135-138) (137, 139)	No	(4, 135, 140, 141)
Clostridium piliforme (*Bacillius piliformis*)	Tyzzer's disease	Cat/Dog		Yes(142)	No	(4, 11)
Clostridium tetani	tetanus	Cat/Dog		Yes(143)	No	(4, 11)
Dermatophilus congolensis	Dermatophilosis	Cat/Dog		Yes(137)	No	(4)
Helicobacter spp. Helicobacter pylori Helicobacter felis Helicobacter heilmanni/bizzozeronii, Helicobacter canis	Helicobacter gastritis Hepatitis	Cat Dog	Yes	46.1.5.1.2. Yes(144) *46.1.5.1.3.* Helicobacter heilmanni/bizzozeronii, Helicobacter canis *are present in New Zealand.*	No	(4)
Leptospirosis spp. • *Leptospira borgpeterseni* serovar *hardjo* • *Leptospira interrogans* serovar *copenhageni* • *Leptospira interrogans* serovar *pomona* • *Leptospira interrogans* serovar *tarassovi* • *Leptospira ballum* • *Leptospira balanica*	Leptospirosis	Cat/Dog Cat/Dog Cat/Dog		Yes(145-147)	No	(146, 147)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
		Dog Cat Cat	Yes			
Listeria monocytogenes	Listeriosis (circling disease)	Cat/Dog	Yes	Yes(137, 148)	No	(4)
Mycobacterium bovis	Bovine tuberculosis	Cat/Dog	Yes(149)	Yes (150, 151) (152) (149)(Under an eradication program)	Yes	(150, 153)
Mycobacterium lepraemurium	Cat leprosy	Cat		Yes(116, 149, 154)	No	(4)
Mycobacterium spp. • *Mycobacterium avium* • *Mycobacterium tuberculosis*		Cat/dog	Yes Yes	Yes(116, 149, 155)	No	(11)
Nocardia asteroides	Nocardiosis	Cat/Dog		Yes(116, 156)	No	(4)
Pasteurella multocida		Cat/Dog		Yes(157)		(4)
Rhodococcus equi (Corynebacterium equi)	Pyogranulomatosis lesions	Cat/Dog		Yes(139, 158)	No	(4)
Salmonella spp. • *S.anatum* • *S.arizonae* • *S.typhimurium* • *S.enteriditis* • *S.Choleraesuis* • *S.saintpaul* • *S.bovimorbificans* • *S.newington* • *S.Hindmarsh* • *S.Mbandaka* • *S.Brandenburg* • *S.Havana* • *S.enteritis* • *S.enterican subspecies Houtenae* • *S.Adelaide* • *S.Ohio* • *S.Tennessee*		Cat/Dog		Yes(159) (116, 148, 160-163)	No	(4, 11, 161, 164)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
• *S.Typhimurium DT04* • *S.singapore*						
Shigellosis spp. • *Shigellosis dysenteriae* • *Shigellosis flexneri* • *Shigellosis boydii* • *Shigellosis sonnei*	Shigellosis	Dog	Primarily a human pathogen	Yes (165-167) (168, 169)	No	(4)
Streptobacillus moniliformis	Rat bite fever	Cat	Yes	Yes(170)		(170)
Streptococcus *spp.* Streptococcus canis (Group G)	Canine streptoccocal toxic shock syndrome	Dog		Yes(171-173)		(174, 175)No recognised virulent strain of Streptococcus canis(176).
Yersinia *spp.* • *Yersinia enterocolitica* • *Yersinia pseudotuberculosis*	Enterocolitis/enteritis	Cat/Dog		Yes(177)	No	(4)
Mycoplasmas						
Mycoplasma spp. • *Mycoplasma felis* • *Mycoplasma canis*		Cat/Dog	Yes	Yes(178-180)		(4)
Fungi						
• *Aspergillus fumigatus* • *Aspergillus nidulans* • *Aspergillus flavus* • *Aspergillus niger*	Aspergillosis	Dog		Yes(181-183)		(4)
Candida albicans	Candidiasis	Cat/Dog		Yes(181, 184)		(4)
Cryptococcus neoformans		Cat/Dog	Yes	Yes(116, 185)	No	(4)
Microsporum spp. • *Microsporum canis*	Dermatophytosis (Ringworm)	Cat/Dog		Yes(116, 186)	No	(4, 186)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
• *Microsporum cookei* • *Microsporum distortum* • *Microsporum gypseum*						
Sporothrix (*Sporotrichum*) *schenckii*	Sporotrichosis	Cat/Dog	Yes	Yes(116)	No	(4)
Trichophyton *spp.* • *Trichophyton mentagrophytes* var. *mentagrophytes* • *Trichophyton terrestre* • *Trichophyton equinum* var. *autotrophicum* • *Trichophyton mentagrophytes* var. *erinacei* • *Trichophyton verrucosum*		Cat/Dog		Yes(116, 186)	No	(4, 186, 187)
Algae						
Prototheca wickerhamii *Prototheca zopfii*	Protothecosis	Cat/Dog		Yes(188-190)		(4)
Parasites						
Nematode						
Aelurostrongylus abstrusus		Cat		Yes(191)	No	(191)
Ancylostoma caninum (*Ancylostoma tubaeforme*)		Dog	Yes	Yes(108, 192)	No	(47)
Anisakis spp.		Dog/Cat		Yes(193)	No	(73, 194)
Capillaria aerophila (*Eucoleus areophilus*)		Cat		Yes(191)	No	(191)
Capillaria erinacei (*Capillaria putorii, Aonchotheca erinacei*)		Cat/Dog		Yes(195)	No	(195)
Capillaria hepatica (*Hepaticola hepatica, Calodium hepaticum*)		Dog		Yes(196)	No	(196)
Trichinella spiralis	Trichinellosis	Cat	Yes	Yes		
Cestodes						
Cylicospirura advena		Cat		Yes(197)	No	(197)
Dipetalonema reconditum		Dog		Yes(198)	No	(191)
Dipylidium caninum		Cat/Dog	Yes	Yes(195, 199)	No	(195)
Filaroides (*Oslerus*) *osleri*		Dog		Yes(108, 200)	No	(200)
Ollulanus tricupis		Cat		Yes(195, 201, 202)	No	(202)
Taenia hydatigena		Dog		Yes(195)	No	(195)
Taenia multiceps		Dog		Yes(192)	No	(47)
Taenia ovis		Dog		Yes(199, 203)	No	(199)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Taenia pisiformis/serrata		Dog		Yes(203)	No	(203)
Taenia serialis (*Multiceps serialis*)		Dog		Yes(203)	No	(203)
Taenia taeniaeformis		Cat		Yes(195)	No	(195)
Toxacaris leonina		Dog/Cat		Yes(108, 192, 199)	No	(116, 199)
Toxocara canis		Dog	Yes	Yes(195, 199)	No	(195)
Toxocara cati		Cat		Yes(195)	No	(195)
Trichostrongylus axei		Cat		Yes(202)	No	(202)
Trichuris vulpis		Dog		Yes(195, 199)	No	(195)
Uncinaria stenocephala		Cat/Dog		Yes(192, 195, 199)	No	(195, 204)
Protozoan						
Balantidium coli		Dog	Yes	Yes(205)	No	(11)
Besnoitia wallacei		Cat		Yes	No	(206)
Cryptosporidium *spp.*		Cat/Dog	Yes	Yes(192, 207, 208)	No	(19)
Encephalitozoon cuniculi	Encephalitozoonosis	Cat/Dog	Yes	Yes(209)	No	(210)
Entamoeba histolytica	Amebiasis	Cat/Dog	Primarily a human parasite	Yes(211)	No	(11)
Giardia intestinalis (also termed lamblia or duodenalis)	Giardiasis	Cat/Dog	Yes	Yes(192, 212, 213)	No	(11)
Haemobartonella canis	Haemobartonellosis	Dog		Yes(163)	No	(4)
Haemobartonella felis	Feline hemobartonellosis	Cat		Yes(214)	No	(214)
Hammondia heydorni		Dog		Yes(215)	No	(4, 11)
Isospora spp. • *Isospora canis* • *Isospora felis* • *Isospora ohioensis* • *Isospora rivolta*		 Dog Cat Dog Cat		Yes(215, 216)	No	(56, 215, 216)
Neospora caninum	Neosporosis	Dog		Yes(109)	No	(4, 11)

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Pneumocystis carinii		Cat/Dog		Yes(217)	No	(4, 11)
Sarcocystis *spp.* • *Sarcocystis muris* • *Sarcocystis arieticanis* • *Sarcocystis cruzi* (*Sarcocysts bovicanis*) • *Sarcocystis gigantea* • *Sarcocystis hirsuta* • *Sarcocystis medusiformis* • *Sarcocystis capracanis* • *Sarcocystis tenella/ovicanis*		 Cat Dog Dog Cat Cat Cat Dog Dog		 Yes(192, 216, 218-222)	 No	 (19, 218, 219, 221, 222)
Toxoplasma gondii	Toxoplasmosis	Cat/Dog	Yes	Yes(109, 216)	No	(4, 216)
Fleas						
Ceratophyllus gallinae		Cat	Yes	Yes(223)	No	(81)
Ctenocephalides canis		Cat/Dog	Yes	Yes(223, 224)	No	(224)
Ctenocephalides felis		Cat/Dog	Yes	Yes(223, 224)	No	(224)
Nosopsyllus fasciatus		Cat/Dog	Yes	Yes(192, 223)	No	
Pulex irritans		Dog	Yes	Yes(223, 224)	No	(224)
Lice						
Felicola subrostratus		Cat		Yes(223)	No	
Linognathus setosus		Dog		Yes(223)	No	
Trichodectes canis		Dog		Yes(223)	No	
Mites						
Cheyletiella blakei		Cat		Yes(225)	No	
Cheyletiella parasitivorax		Cat	Yes	Yes(226)	No	
Cheyletiella yasguri		Dog	Yes	Yes(201)	No	
Demodex canis	Mange	Dog		Yes(223)	No	
Demodex cati	Mange	Cat		Yes(223)	No	
Dermanyssus gallinae		Dog	Yes	Yes(227)	No	(227)
Notoedres cati		Cat		Yes(223)	No	
Ornithonyssus bursa		Cat		Yes(228)	No	(228)
Otodectes cynotis		Cat/Dog		Yes(223)	No	(223)
Sarcoptes scabiei	Mange	Dog	Yes	Yes(223)	No	
Flies						
Oestrus ovis		Cat/Dog	Yes	Yes(223)	No	(192, 229)
Stomoxys calcitrans		Dog	Yes	Yes(223)	No	

Disease agent	Disease name	Susceptible species (Cat/Dog)	Zoonotic	Present in New Zealand	Potential hazard (Yes/No)	Reference that occurs in cats/dogs
Ticks						
Haemaphysalis longicornis		Cat/Dog	Yes	Yes(223)	No	
Pentastomida						
Linguatula serrata	Tongue worm	Cat/Dog		Yes(223)	No	
Other hazards						
Soil and seeds carried in coats		Cat/Dog				Included at the request of The Department of Conservation.

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