Import risk analysis:
Zoo primates from Australia, Canada, the European Union, USA and Singapore

FINAL

Photo: Fraans de Waal (public domain)

April 2011

This page is intentionally blank

MAF Biosecurity New Zealand
Pastoral House
25 The Terrace
PO Box 2526
Wellington 6011
New Zealand

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

Policy and Risk
MAF Biosecurity New Zealand

Import risk analysis:
Primates from Australia, Canada, the EU, the USA, and Singapore

FINAL

April 2011

Approved for general release

Christine Reed
Manager, Risk Analysis
MAF Biosecurity New Zealand

This page is intentionally blank

Contents

Contributors to this risk analysis

Primary author

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

Co-author

S C MacDiarmid	Principal International Adviser, Risk Analysis	MAF Biosecurity New Zealand

Internal peer review

R Somervell	Senior Adviser Risk Analysis (Animals)	MAF Biosecurity New Zealand
C Dyer	Senior Adviser Animal Imports and Exports	MAF Biosecurity New Zealand
D Lush	Principal Adviser Human Health	MAF Biosecurity New Zealand
H Pharo	Team Manager Risk Analysis (Animals)	MAF Biosecurity New Zealand

External peer review

N Masters	International Zoo Veterinary Group	Twycross Zoo United Kingdom

Executive Summary

The commodity definition for this import risk analysis is confined to19 species of nonhuman primates from zoos in Australia, Canada, the EU, the USA and Singapore. It specifies that primates to be imported must be clinically healthy and originate from premises under veterinary supervision and not from what the OIE's *Terrestrial Animal Health Code* calls 'an uncontrolled environment'. The specification that the premises of origin must be under veterinary supervision is important in that it minimises the risk associated with a number of diseases potentially carried by the primates. The commodity definition explicitly excludes primates which have been caught in the wild.

Fifty nine disease pathogens or groups of pathogens are considered to be of potential concern and could be introduced in imported primates. This list is essentially the same as that in a draft risk analysis produced by Biosecurity Australia. Each pathogen is subjected to an initial examination of the likelihood of its introduction in the commodity. Pathogens meeting the following criteria are eliminated from the list;

- The pathogen does not occur in any of the countries to which this risk analysis applies.
- The pathogen does not occur in any of the 19 species covered by this risk analysis.

The reduced list of pathogens, termed the 'preliminary hazard list', contains ten viruses, three bacteria, internal metazoan parasites (trematodes, cestodes, nematodes and acanthocephalans), external arthropod pests (fleas, lice, insect agents of myiasis, ticks and mites). Each of these is subjected to a full risk assessment. Options for the effective management of the risks are given for the following which are assessed to be a risk in the commodity;

- Hepatitis B virus
- Rabies virus
- Tuberculosis (*Mycobacterium tuberculosis* and *M. bovis*)
- Enteric bacteria
- Helminth parasites
- Lice
- Ticks
- Mites
- Weed seeds

1. Introduction

This risk analysis has been conducted in response to a request from the Australasian Regional Association of Zoological Parks and Aquaria (ARAZPA) for an Import Health Standard (IHS) for nonhuman primates (referred to throughout the text simply as 'primates') from specified countries. Completion of a risk analysis is required before an IHS can be written.

2. Scope

The risk analysis is limited to disease-causing organisms as defined in the Biosecurity Act. It covers viral, bacterial, fungal, protozoal, arthropod and helminth pathogens that can infect the primate species listed in the commodity definition below. Genetic diseases and other risk factors that may be of commercial or captive management importance to importers are not considered.

The risk analysis is qualitative. The *Terrestrial Animal Health Code* (referred to throughout as 'the *Code*') of the World Organisation for Animal Health (OIE)[1] makes recommendations on precautionary measures to be followed by staff exposed to nonhuman primates or to their body fluids, faeces and tissues (Article 6.12.7. It is assumed that importers of primates will follow these guidelines.

3. Commodity definition

The term 'primate' in this analysis refers to nonhuman mammals of the taxonomic order Primates. The risk analysis considers the primate species nominated in the ARAZPA Regional Census and Plan, as supplied to MAF Biosecurity New Zealand (MAF) by the Wellington Zoo Trust. The source countries considered are Australia, the United States of America (USA), Canada, the European Union (EU)[2] and Singapore.

The 'commodity' covered by this risk analysis is defined as the following 19 species of primates sourced from the countries listed above;

A. Of the Madagascan lemurs belonging to the Infraorder Lemuriformes, relevant species are;

Ring-tailed lemur – *Lemur catta*
Black and white ruffed lemur – *Varecia variegata variegata*

---

[1] All references to the *Terrestrial Animal Health Code* refers to the current version. http://www.oie.int/eng/normes/mcode/a_summry.htm

[2] The EU comprises Austria, Belgium, Bulgaria, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Netherlands, Poland, Portugal, Romania, Slovakia, Slovenia, Spain, Sweden, and United Kingdom.

These species from Madagascar have evolved in isolation from other primates.

B. Of the New World monkeys of the Pavorder Platyrrini, relevant species are;

Pygmy marmosets – *Callithrix pygmaea*
Golden lion tamarin – *Leontopithecus rosalia*
Emperor tamarin – *Saguinus imperator*
Cotton-top tamarin – *Saguinus Oedipus*
Spider monkey – *Ateles geoffroyi*
Black cap Capuchin – *Cebus paella*

C. Of the Old World monkeys of the Superfamily Cercopithecoidea, relevant species are;

Mandrill – *Mandrillus sphinx*
Hamadryas baboon – *Papio hamadryas*
Black and white colobus – *Colobus quereza*
Francois's leaf monkey (langur) – *Trachypithecus francoisi*
White-cheeked gibbon – *Hylobates leucogenys*

D. Of the apes belonging to the Superfamily Hominoidea, relevant species are;

Siamang – *Hylobates syndactylus*
Gorilla – *Gorilla gorilla*
Chimpanzee – *Pan troglodytes*
Bornean orangutan – *Pongo pygmaeus*

E. Of the bush babies belonging to the family Galagidae, or African prosimians, relevant species are;

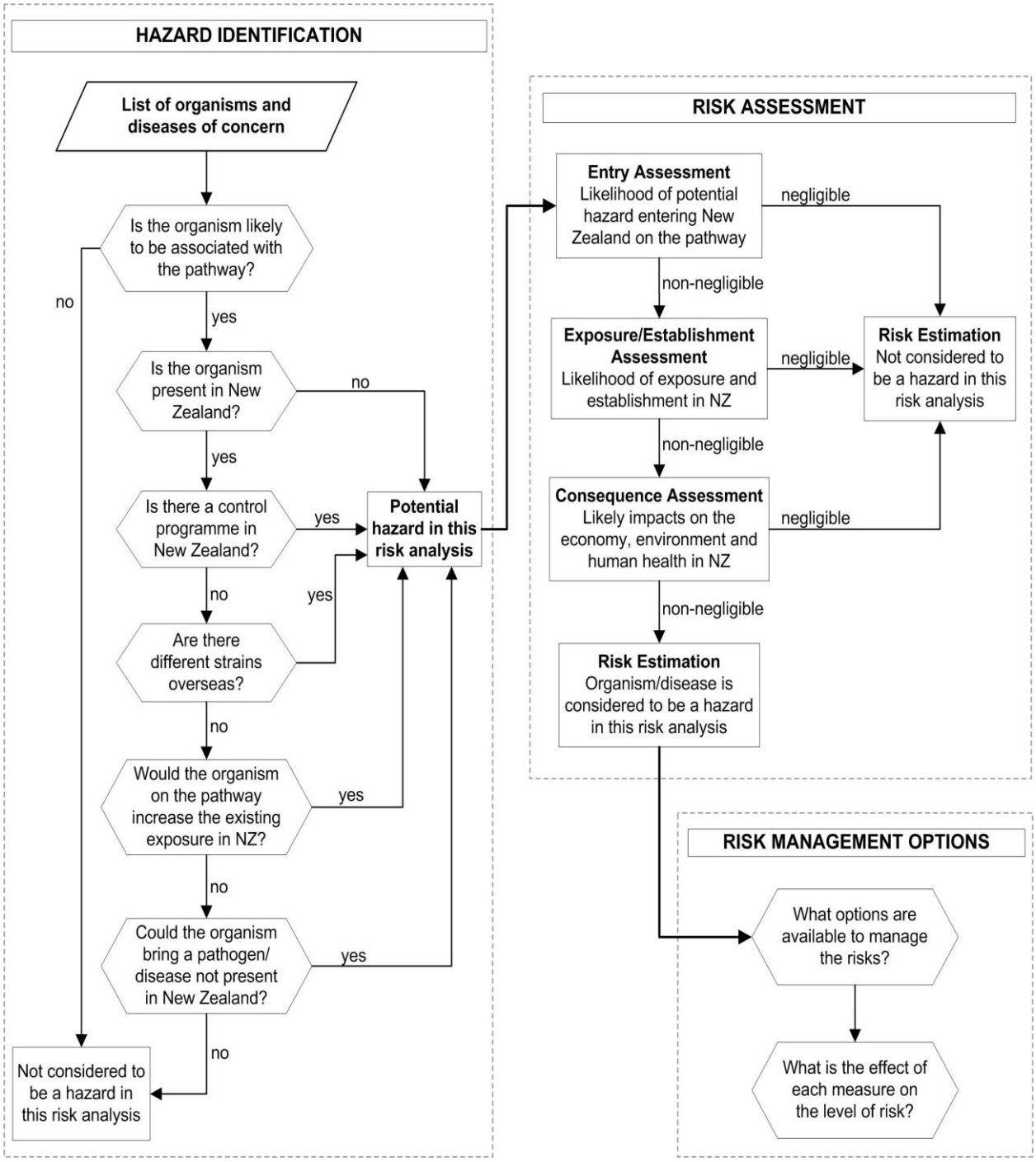
- Lesser bush babies – *Galago moholi* and *Galago senegalensis*
  Greater bush babies –*Otolemur crassicaudatus* and *Otolemur garnettii*

Primates must originate from zoos or animal holding facilities under permanent veterinary supervision and registered by the official veterinary authority of the exporting country to keep primates. For the purposes of this risk analysis, such a premises is referred to as 'a controlled environment'. The primates to be imported must be of a species that is approved for importation by New Zealand's Environmental Risk Management Authority (ERMA). After importation the primates must be permanently housed in zoos or other registered containment facilities. Primates captured from the wild may not be imported. Prior to export, the primates must be certified to be healthy by a veterinarian specialising in zoo medicine. They must be individually identified and have lived for a minimum of two years or since birth in a controlled environment under veterinary supervision

4. Risk analysis methodology

The methodology used in this risk analysis is based on Chapter 2.1 of the *Code* and the MAF guidelines (MAF 2006). This process is summarised in Figure1.

Figure 1. The risk analysis process.



4.1. COLLATION OF A LIST OF ORGANISMS OF POTENTIAL CONCERN

The *organisms of potential concern* that could be associated with primates are those that could be transmitted from imported primates to domestic, feral or wild animals or humans.

Biosecurity Australia has carried out extensive work on a *Draft Generic Import Risk Analysis of Nonhuman Primates* and has made this draft available to MAF. The list of disease agents in that Australian document was supplemented with additional ones identified from various electronic databases, scientific journals and books or suggested by experts consulted.

4.1.1. Organisms of potential concern

Viruses (those listed under the official names given by the International Committee on Taxonomy of Viruses are italicized)

Adenoviridae
- *Adenovirus species*

Arenaviridae
- *Lymphocytic choriomeningitis virus*

Arteriviridae
- *Simian haemorrhogic fever virus*

Bunyaviruidae
- *Oropouche virus*

Caliciviridae
- *Primate calicivirus*

Coronaviridae
- *Severe acute respiratory syndrome coronavirus*

Filoviridae
- *Ebola virus*
- *Marburg virus*

Flaviviridae
- *Dengue virus*
- *Japanese encephalitis virus*
- *Kyasanur Forest disease virus*
- *West Nile virus*
- *Yellow fever virus*

Hepadnoviridae
- *Hepatitis B virus*

Herpesviridae
- *Cerccopithecine herpesvirus 1 (*B virus*)*
- *Cerccopithecine herpesvirus 2 (*Simian agent 8*)*
- *Cerccopithecine herpesvirus 9 (*Herpesvirus tamarinus*)*
- *Saimirine herpesvirus1 (*Herpesvirus tamarinus*)*
- *Saimirine herpesvirus 2*
- *Ateline herpesvirus 2*

Morbilliviridae
- *Measles virus*

Papilloviridae
    Papilloma viruses
Picornaviridae
    Hepatitis A virus
    Poliovirus
    Simian enterovirus A
Polyomaviridae
    Simian virus 40
Pox viridae
    Monkeypox virus
    Tanapox virus
    Yabapox virus
    Marmosetpox virus
Reoviridae
    *Rotavirus A (*Simian rotovirus*)*
    Baboon orthoreovirus
Retroviridae
    Simian retroviruses
Rhabdoviridae
    Rabies virus
Togaviridae
    Chikungunya virus
    Mayaro virus

Transmissible spongiform encephalopathy agents (prions)

Bacteria

Borrelia burgdorferi
Brucella species
Enteric bacteria
Erhlichia chaffeensis
Francisella tularensis
Haemophilus influenzae
Mycobacterium tuberculosis and *M. bovis*
Mycobacterium leprae
Rickettsia conorii
Yersinia pestis

Fungi

Protozoal parasites
Babesia spp. and *Entopolypoides* spp.
Cyclospora spp.

Enteric protozoal parasites
Giardia spp.
Entamoeba histolytica
Cryptosporidia spp.
Balantidium coli
Haemogregarina cynomolgi
Hepatocystis simia and *Hepatocystis kochi*
Leishmania spp.
Plasmodium spp.
Tritrichomonas mobilensis
Trypanosoma cruzi and related trypanosomes
Trypanosoma brucei

Internal helminth parasites

Trematodes and cestodes
Nematodes and acanthocephalans

Arthropod parasites

Other metazoan parasites

Pentastomids
Annelids

4.2. PRELIMINARY HAZARD LIST

Organisms that meet any of the following criteria were eliminated from the list of organisms of potential concern and not considered further;

- Organisms that are not known to infect primates of the 19 species in the commodity definition.
- Organisms that infect primates but are not known to infect humans or other animals. These organisms may be of importance to importers since they may cause disease in their captive primate populations. However, since there are no wild or feral primates in New Zealand, these organisms are not considered to be biosecurity hazards.
- Organisms that are not known to occur in the exporting countries.

However, in order to allay possible public concern, a small number of high profile disease agents that could have been eliminated using the above criteria were retained in the preliminary hazard list.

Each organism remaining in the preliminary hazard list was submitted to *hazard identification.*

4.3. HAZARD IDENTIFICATION

Hazard identification involves a more detailed consideration of the epidemiology and key factors related to transmission of each agent classified as *preliminary* hazards, in order to decide whether there is sufficient evidence for it to be subjected to an individual *risk assessment*.

4.4. RISK ASSESSMENT

According to the OIE methodology, the following assessments are carried out for each potential hazard:

a) Entry assessment - the likelihood of the organism being imported in the commodity.

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 the organism.

d) Risk estimation - a conclusion on the risk posed by the organism based on the entry, exposure and consequence assessments. If the risk estimate is non-negligible, then the organism is classified as a *risk*.

Not all of the above steps may be necessary in all risk assessments. The OIE methodology makes it clear that if the likelihood of entry is negligible for a particular hazard, then the risk estimate is automatically negligible and the remaining steps of the risk assessment need not be carried out. Similarly, if the likelihood of entry is non-negligible but the likelihood that a susceptible animal in the importing country will be exposed is assessed to be negligible, or when both entry and exposure are non-negligible but the consequences of introduction are assessed to be negligible.

Organisms classified as *risks* are subjected to the *risk management* process.

4.5. RISK MANAGEMENT

Risk management is a consideration of the options available for managing the risk posed by each hazard. Where the *Code* gives recommendations for the management of a risk, these are described along with options of similar, lesser or greater stringency, where available. Recommendations as to which sanitary measures should be applied to achieve the effective management of risks are not made in this document. Sanitary measures will be determined during the process of developing an IHS.

As obliged under Article 3.1 of the WTO *Agreement on the Application of Sanitary and Phytosanitary Measures* (the so-called *SPS Agreement*), the measures adopted in an IHS should be based on international standards, guidelines and recommendations, where such exist, except as otherwise provided for under Article 3.3 (where measures providing a higher level of protection than international standards may be applied when there is scientific justification, or when there is a level of protection that the member country considers more appropriate following a risk assessment).

4.6. SPECIAL CONSIDERATIONS

Some special considerations are applicable to the *risk assessment* and *risk management* processes.

The source of the primates: The *Code* gives recommendations (Chapter 6.12.) for importation of primates in two situations;

- from what it calls "an uncontrolled environment", which is one in which the animals are captured from the wild or come from sources not subject to permanent veterinary supervision, or;

- from a facility under permanent veterinary supervision. In this analysis such a facility will be called "a controlled environment."

This risk analysis deals solely with primates sourced from a controlled environment.

Further, with the exception of Singapore, all the countries from which primates may be sourced in this analysis are outside the natural range of nonhuman primates. This fact essentially eliminates the likelihood that primates in a zoo collection could be infected through contact with wild primates.

Importation is into a containment facility: Primates imported into New Zealand are all destined to spend their lives in a containment facility. Being kept in such a controlled environment eliminates the likelihood of their coming into contact with other animals and with humans other than their handlers. Zoo staff working with primates are all suitably trained in their handling.

Quarantine: A period of quarantine may be considered appropriate to manage the risk posed by a particular hazard. In such situations, the incubation period and the time for which an animal may remain infectious are critical for determining the duration of quarantine. In some diseases the infectious period may be extended, sometimes for the life of the animal. In such cases quarantine is not a useful option for excluding introduction of the particular hazard. In the case of diseases for which long-term carriers do not occur, the infectious period is generally confined to the period during which the animal is viraemic or bacteraemic. An animal could have been infected with a disease on the day it enters quarantine. After the incubation period, it will be viraemic or bacteraemic for a period that differs with each disease.

As a rule, animals should be quarantined for the maximum known incubation period plus the maximum period for which viraemia or bacteraemia can last.

Should a period of quarantine be deemed necessary fore the safe importation of primates, quarantine conditions must be at least equivalent to those recommended in Chapter 5.9. of the *Code*. During this period of quarantine, the primates should be subjected to tests and treatments as specified in the appropriate IHS which will be developed on the basis of this risk analysis. All diagnostic tests should be carried out in a laboratory approved by the veterinary authority of the exporting country. Collection and shipment of diagnostic specimens should be carried out in accordance with the recommendations of Chapter 1.1.1. of the OIE's *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* and Article 4.15.1. of the *Code*.

Many diseases are shared with humans: Since many diseases of primates also affect humans, special attention should be paid to the zoonotic potential of each disease agent. However, primates imported into the controlled environment of a zoo pose a different risk than animals imported into an uncontrolled environment. They are generally held in confined conditions that exclude direct contact with humans other than their keepers and other trained professionals who may occasionally handle them. Such staff are trained in the handling of primates and the hygienic measures necessary for occupational safety and health. Imported primates, therefore, are more likely to pose a risk to other primates, in the captive populations into which they are introduced, than to humans.

Since there are no free-living primates in New Zealand, biosecurity risks posed by imported primates are largely limited to the possible introduction of human diseases, although there may be some risk to other animals. Some of the zoonotic diseases of primates are already endemic in humans in this country and, therefore, introduction of a small number of infected animals into facilities in which they will have little direct contact with humans does not pose a significant increase in biosecurity risk. However, in this risk analysis each case is considered individually and a risk averse approach is taken in formulating risk management options.

Proportionality: Nevertheless, in considering whether or not measures should be applied to eliminate risks from primates infected with human diseases, it is important to consider the issue of proportionality. In situations where there is a non-negligible likelihood of an imported primate carrying a disease that is equally likely to be carried by a human, it would be unjustifiable to impose onerous measures on the importation of a few primates, all entering a permanently controlled environment, when no meaningful measures are, or can be, applied to the hundreds of thousands of human who enter the country each year. Not only would the imposition of measures to the primate importation do nothing to significantly reduce the biosecurity risk to New Zealand, it would also be contrary to the obligations of the *SPS Agreement* which requires proportionality.

Anaesthetics are stressful: When selecting which sanitary measures should be incorporated into an IHS, it must be kept in mind that for many procedures it may be necessary to anaesthetise primates. This procedure is not without risk to the animals and is certainly stressful to them. Procedures which require primates to be anaesthetised should be required only when absolutely necessary to protect New Zealand's biosecurity.

5. Development of a preliminary hazard list from the list of organisms of potential concern

5.1. VIRUSES

5.1.1. Adenoviruses

At least 27 serotypes of adenovirus occur in primates (Mansfield and King 1998). Serotypes of the virus are generally specific to a particular species. Strains vary in organ tropism and signs of infection vary according to the tropism of the infecting strain. Many infections are subclinical and no evidence was found that adenoviruses from primates are transmitted to humans or other animals. Therefore adenoviruses are not considered to be a hazard in the commodity.

5.1.2. Lymphocyte choriomeningitis virus (Callitrichid hepatitis virus)

Lymphocytic choriomeningitis virus is carried by mice. As humans and primates may be infected as accidental hosts (Peters et al 1996), lymphocytic choriomeningitis virus is considered to be a preliminary hazard in the commodity.

5.1.3. Simian haemorrhagic fever virus

Simian haemorrhagic fever virus is an *Arterivirus* that causes an acute infection with high mortality in rhesus macaques (London 1997; Myers et al 1972; Snijder et al 2005). Patas monkeys are apparently subclinically infected carriers of this virus (London 1997). Since the virus is not known to infect humans or other animals (Snijder et al 2005) it is not considered to be a hazard in the commodity.

5.1.4. Oropouche virus

Oropouche fever is a disease of humans that is transmitted by *Culicoides paraensis* (Pinheiro et al 1982). The virus appears to be maintained in an urban cycle in humans but there is also an undefined sylvatic cycle (Gonzalez-Scarano and Nathanson 1996). The disease is confined to Central and South America and has not been described in primates. However, antibody has been found in cebus and howler monkeys (Anderson et al 1961) and virus has been isolated from three-toed sloths. The sylvatic cycle could involve sloths, monkeys and jungle mosquitoes (Gonzalez-Scarano and Nathanson 1996). In humans the incubation and viraemic periods are short. The disease agent is not considered to be a hazard in the commodity because:

- It is confined to Central and South America.
- It is typically a disease of humans.
- The virus has not been isolated from primates except for humans.
- The disease has not been described in captive primate populations.

- It is carried by *Culicoides paraensis* and, possibly, by jungle mosquitoes, neither of which occur in New Zealand.

5.1.5. Primate calicivirus

Primate calicivirus is the only calicivirus of primates described by the International Committee on Taxonomy of Viruses (Koopmans et al 2005). Primate calicivirus is considered to be a synonym for *vesicular exanthema virus of swine*. No evidence could be found that it causes disease in other animals or humans. It is not known to be transmitted from primates to pigs. Therefore, it is not considered to be a hazard in the commodity.

5.1.6. Severe acute respiratory syndrome coronavirus (SARS CoV)

There is no evidence to suggest that primates are reservoir hosts for SARS virus. Therefore it is not considered to be a hazard in the commodity.

5.1.7. Filoviruses (Marburg and Ebola viruses)

Both Marburg and Ebola viruses can infect a variety of primates, including humans. The viruses are extremely virulent and cause a high mortality rate in humans. These viruses are considered to be high profile disease agents and, for this reason, are considered a preliminary hazard in the commodity.

5.1.8. Dengue fever virus

Dengue fever is caused by a *Flavivirus* transmitted by insects. It infects humans and several species of Old World monkeys in Asia and Africa. It is the cause of a serious emerging disease of humans and its distribution is expanding. Therefore it is considered to be a preliminary hazard in the commodity

5.1.9. Japanese encephalitis virus

Japanese encephalitis virus is a mosquito-borne *Flavivirus* that causes a serious disease of humans. Maintenance hosts are birds, particularly egrets and herons, while pigs act as multiplying hosts (CDC 2006; World Health Organization 2006). However, although antibodies have been found in a few species of primates they have not been incriminated as maintenance or multiplying hosts. Therefore the virus is not considered to be a hazard in the commodity.

5.1.10. Kyasanur Forest disease virus

Kyansur Forest disease occurs only in Karnataka State, India. The virus is transmitted by at least ten species of tick, with the main vector being *Haemaphysalis spinigera* (Monath and Heinz 1996). Humans are susceptible, as are langurs, bonnet monkeys and a range of other animals including rodents and bats (Mansfield and King 1998). The virus is maintained in a tick-wild vertebrate cycle involving rodents and insectivores. The incubation period following a tick bite is 3-8 days in humans and the viraemic period last up to 13 days. The

disease may be biphasic, with a second febrile attack occurring 1-2 weeks after the first (Monath and Heinz 1996).

The virus is not considered to be a hazard in the commodity because;

- The disease is restricted to a part of India.
- It is carried by tick species that do not occur in New Zealand and imported primates will be required to be tick free.
- The disease has not been described in captive primates.

5.1.11. West Nile virus

West Nile fever is caused by an insect-borne virus which has recently caused a major epidemic in North America. The virus also occurs in Africa and Europe and causes a serious disease in humans. The virus is maintained in various species of birds (Bunning et al 2004). There is no evidence to suggest that primates play any role as either maintenance or multiplying hosts. Therefore, the virus is not considered to be a hazard in the commodity.

5.1.12. Yellow fever virus

Yellow fever virus infects humans and Old and New World primates. Yellow fever is considered to be a high profile disease and is considered to be a preliminary hazard in the commodity.

5.1.13. Hepatitis B virus

Since testing of primates for hepatitis B virus is recommended in the *Code*, it is considered to be a preliminary hazard in the commodity.

5.1.14. Cercopithecine herpesvirus 1 (*Herpesvirus simiae*, Herpes B virus)

The virus occurs naturally only in macaques (Holmes et al 1995). No other Old or New World monkeys are known to carry it (Whitley 1996). Latent infections occur in macaques (Fenner 1996; Whitley 1996). The geographic distribution of *Herpesvirus simiae* is Asia and North Africa, where macaques are found. Humans are susceptible and the mortality rate in infected humans is high (Fenner 1996; Whitley 1996). The incubation period of *Herpesvirus simiae* infection is less than 3 weeks. Primates may be clinically infected, infection can be fatal, and latent infections may be established (Fenner 1996). However, since the commodity definition does not include macaques, the virus is not a hazard in the commodity.

5.1.15. Cercopithecine herpesvirus 2 (simian agent 8, *Herpesvirus papionis*, African monkey herpesvirus).

Herpesvirus papionis has been isolated from vervet monkeys and baboons (Martino et al 1998). Only baboons become clinically affected. Small vesicles, pustules and ulcers develop in the oral cavity, penis, prepuce, vulva and perineum. Secondary bacterial infections frequently lead to sterility. Some infected baboons remain asymptomatic. The disease is generally transmitted venereally (Martino et al 1998). Transmission to humans or other animals has not been recorded. Since disease occurs only in baboons, *Herpesvirus papionis* is considered to be a population management issue for importers but not a biosecurity hazard in the commodity.

5.1.16. Cercopithecine herpesvirus 9 (CeHV-9) simian varicelloviruses.

Simian varicella virus (SVV), Liverpool vervet herpesvirus, patas monkey herpesvirus, delta and Medical Lake macaque herpesvirus are considered by the ICTV to be synonyms of cercopithocine herpesvirus 9 (Davison et al 2005). These viruses may represent strains of the same virus with slightly different characteristics. The disease is characterised by vesicular exanthema ending fatally in a few days (Mansfield and King 1998). The viruses in this group are not known to infect humans or other animals and are, therefore, not considered to be a hazard in the commodity.

5.1.17. Saimirine herpesvirus 1 (*Herpesvirus tamarinus*)

The reservoir hosts of *Herpesvirus tamarinus* are subclinically infected squirrel monkeys (*Saimiri* spp) (King et al 1967). The virus causes fatal disease in owl monkeys (*Aotus* spp.), tamarins (*Saguinus* spp.), cotton-topped marmosets (*Saguinus oedipus*) and marmosets (*Callithrix* spp.) (Mansfield and King 1998). *Lagothrix* spp., *Cebus* spp., *Callithrix* spp. and *Ateles* spp. have been found to be serologically positive without showing clinical signs. In susceptible species, infections may cause oral, labial and dermal lesion and epizootics with high mortality rates (Mansfield and King 1998). Since the virus is associated with New World monkeys and has not been described in humans or other animals, it is considered to be of population management concern to importers of marmosets and tamarins but not a biosecurity hazard in the commodity.

5.1.18. Saimirine herpesvirus 2 (*Herpesvirus saimiri*) and ateline herpesvirus 3 (*Herpes ateles*)

Herpesvirus saimiri and *Herpesvirus ateles* have been isolated from squirrel and spider monkeys respectively (King 2001). These viruses produce in subclinical infections only in their natural hosts (squirrel and spider monkeys) but are oncogenic and cause leukaemia and lymphoma when inoculated into other monkey species. There are no records of infection in humans or other animals and therefore the viruses are not considered to be a hazard in the commodity.

5.1.19. Measles virus

Measles virus can infect primates and is the cause of a serious disease of humans. The incubation period is 10-14 days and the infectious period lasts only until antibody is

produced (Griffin and Bellini 1996). Immunity in humans and primates is life-long (Griffin and Bellini 1996). Measles is endemic in New Zealand and possible limited exposure of trained zoo staff to a small number of imported primates imported from a controlled environment would create no significant biosecurity risk . Therefore measles virus is not considered to be a hazard in the commodity.

5.1.20. Papilloma viruses

The ICTV lists 14 genera in the family *Papillomaviridae*. These genera include 26 species which infect humans, one which infects rhesus monkey species, one species (*Human papillomavirus* 6) that infects both humans and chimpanzees and 16 species which infect other species of animals (de Villiers et al 2005). However, the ICTV states that "putative new papillomaviruses of a variety of different species have been identified by partial sequences. More than 300 such sequences are presently available in the databanks". It is also stated that "the demarcation of species is extremely difficult to apply to papillomaviruses" (de Villiers et al 2005). Papilloma viruses cause tumours and warts in humans and other animals, but are generally species specific. Species in the genus *Alphapapillomavirus* preferentially infect the buccal or anogenital mucosa. In this genus *Chimpanzee papillomovirus 1* has been described in pygmy chimpanzees (de Villiers et al 2005; Sundberg et al 1992). *Rhesus monkey papilloma virus 1* is associated with genital tumors and warts in rhesus macaques (a species that will not be imported) and no reference could be found to it occurring in other species. Papillomaviruses have been isolated from several subclincally infected primate species, but no evidence was found for interspecies transmission (Antonsson and Hansson 2002). Most papilloma viruses are harmless or only mildly pathogenic. They are transmitted by close contact, on fomites or venereally and therefore the likelihood of spread from zoo primates to humans is remote. In addition, papilloma viruses of humans are universally distributed and most species are probably already endemic in humans in New Zealand. Therefore, papilloma viruses are not considered to be a hazard in the commodity.

5.1.21. Hepatitis A virus

Hepatitis A virus occurs in primates and humans but the nucleotide sequence of the primate strain differs significantly from that of the strain in humans. At least 4 variants of primate viruses occur and these differ from human isolates. It has been suggested that the primate strains have evolved separately from human strains of the virus (Balayan 1992). It has been shown that *Macaca rhesus* are susceptible to infection with simian strains but not with human strains (Zamiatina et al 1990). However, transmission of hepatitis A virus from chimpanzees to humans has been described (Dienstag et al 1976). It is unclear, therefore, whether some human strains can infect primates, particularly chimpanzees and whether primate strains can, in turn, infect humans.

Viraemia may last for up to 391 days in humans and chimpanzees (Bower et al 2000). Hepatitis caused by type A virus occurs sporadically in humans in New Zealand. Ninety one cases were notified in the year to December 2008 (ESR 2009). Since importation of infected primates could cause outbreaks of a disease that occurs only sporadically in humans, the virus is considered to be a preliminary hazard in the commodity.

5.1.22. Polio virus

Polio virus can infect a number of primates giving rise to signs similar to those seen in the human disease. However, polio has been eradicated from all countries of relevance to this risk analysis (Anonymous 2009) and so the virus is not a hazard in the commodity.

5.1.23. Simian enteroviruses

A single episode of a fatal disease in rhesus monkeys associated with an enterovirus has been described (Kaufmann et al 1973) . Since no references to other episodes of disease were found, it is considered to have been a unique event and the virus is not considered to be a hazard in the commodity.

5.1.24. Simian virus 40

Simian virus 40 occurs commonly as a latent infection in subclinically infected monkeys. The virus contaminated millions of doses of polio vaccine which were used in humans. More than 98 million Americans received at least one dose of the injectable form of the vaccine (CDC 2007). Despite various investigations involving hundreds of thousands of subjects, there is no evidence to suggest that simian virus 40 is oncogenic or otherwise harmful to humans (CDC 2007). Therefore, although many monkeys may carry the virus it is neither of importance to importers nor a hazard in the commodity.

5.1.25. Monkeypox virus.

Monkey pox virus infects primates, humans, rodents and lagomorphs. The disease is restricted to central and western Africa and Sudan (Heymann et al 1998). The maintenance hosts are rodents. A serological survey failed to detect antibodies against monkeypox virus in wild Asian macaques but African green monkeys have a high prevalence of antibodies (Mansfield and King 1998). A major outbreak of the disease occurred in the USA following the importation of rodents from Ghana (CDC 2003). In that outbreak, the virus was isolated from imported giant pouched rats (*Cricetomys* spp.), rope squirrels (*Funisciuris* spp.) and dormice (*Graphiuris* spp.).The disease also involved prairie dogs (kept as pets) and humans (Hutson et al 2007).

Transmission from monkeys to humans and between humans is inefficient (Hutin et al 2001, Heymann et al 1998). Healthcare workers in contact with infected persons were not infected in the US outbreak (Fleischauer et al 2005) and no case of human to human transmission was confirmed (Siegel et al 2007). However, in one outbreak of the disease in the Congo up to six sequential transmissions of monkeypox virus from person to person were hypothesized to have occurred (Learned et al 2005). Transmission requires close contact between humans, or monkeys and humans, and is often associated with preparing and eating primate ("bush") meat. The median incubation period was 12 days (range: 1-31 days) and infected monkeys remain viraemic for about a week (CDC 2003). A carrier state has not been described.

The virus is not considered to be a hazard in the commodity because;

- The disease is restricted to central and western Africa.

- Primates are not considered to be the primary reservoirs of the virus.
- Transmission from human to human, primate to human, and primate to primate is inefficient.

5.1.26. Tanapox virus

The disease has occurred in humans in the Tana river area of Kenya (Manson-Bahr and Downie 1973), the Congo (Knight et al 1989) and in humans handling macaques held in captivity (Downie et al 1971) . Pox-like lesions develop on skin and may ulcerate before resolving (Fenner 1996; Mansfield and King 1998). In monkeys the disease occurs most commonly in macaques but several species of African primates have been found to be serologically positive without showing signs of disease. Antibody has not been found in Asian monkeys, indicating a lack of exposure (Fenner 1996). Outbreaks of disease in primate colonies in the USA have been associated with contact between susceptible Asian monkeys and imported African monkeys (Downie et al 1971).

In humans, the disease is usually mild. Spread from human to human has not been described. There is circumstantial evidence that the tanapox virus is transmitted by insects in endemic areas of Africa (Stich et al 2002), but transmission by close contact is also possible since it has apparently been transmitted from laboratory animals to humans (Downie et al 1971; Hall and McNulty 1967). The incubation period is 4-6 days and lesions resolve within 3-4 weeks (Mansfield and King 1998). No reference was found to suggest that long-term carriers occur. The likelihood of introducing the virus in healthy monkeys originating from the controlled environment of a registered zoo in which the disease has not been seen is considered to be negligible and so tanapox virus is not considered to be a hazard in the commodity.

5.1.27. Yaba monkey tumor virus.

Yaba monkey tumour virus belongs to the genus Yatapox (Buller et al 2005). The virus is not antigenically related to monkey pox but some cross reactivity occurs with tanapox virus which is also a member of the Yatapox genus. The virus most commonly infects macaques but has also been found in other Old World monkeys , apes and baboons (Mansfield and King 1998). Tumours develop in the skin of monkeys and humans after subcutaneous or intradermal injection of Yaba monkey tumour virus (Fenner 1996). Naturally-acquired infections have also been described in humans but are rare. The virus causes subcutaneous tumours, especially on the plantar surfaces of the hands and feet, but the disease resolves within several weeks (Mansfield and King 1998). It has been suggested that Yaba monkey tumour virus is transmitted by insects but the method of transmission is uncertain. Experimental transmission by aerosols resulted in tumour development in the lungs (Wolfe et al 1968). The virus causes a non-fatal disease of monkeys and baboons and is rarely transmitted to humans in which it is a self-limiting infection. No references were found describing human to human transmission. While Yaba monkey tumour virus might be considered to be of commercial importance to importers, it causes a mild disease only in humans and is not contagious from person to person and so is considered not to be a hazard in the commodity.

5.1.28. Marmosetpox virus

A single outbreak only of this disease, which was confined to marmosets, has been described. Twenty nine of 80 common marmosets were infected and eight died, but deaths were no necessarily attributable to the virus (Gough et al 1982). A pox virus was seen by electron microscopy but could not be isolated. Since the disease has been described only once it must be considered to be a rare curiosity. It has not been described in humans or other animals. Therefore, it is not considered to be a hazard in the commodity.

5.1.29. Rotavirus A (Simian rotavirus)

Rotaviruses occur universally and are common in humans and other species in New Zealand. *Rotavirus A* (simian rotavirus*)* is distinct from *Rotavirus C* (human rotavirus) (Ramig et al 2005). Transmission of animal rotaviruses to humans is rare (Kapikian and Chanock 1996) and they are not considered to be a hazard in the commodity.

5.1.30. Baboon orthoreovirus

A single case of a disease of juvenile baboons infected with an orthoreovirus has been described. The disease was characterised by a non-suppurative meningoencephalomyelitis. The orthoreovirus isolated from the cases did not cause any clinical signs when inoculated into young baboons but typical lesions were demonstrated and orthoreovirus was isolated from their brains (Leland et al 2000). There is no evidence that baboon orthoreovirus infects humans. Since the disease has been described only once it is regarded as a rare infection of little significance and is not considered to be a hazard in the commodity.

5.1.31. Simian retroviruses

The retroviruses of primates recognised by ICTV (Linial et al 2005) are;

Genus: *Betaretrovirus*
Langur virus
Mason-Pfizer monkey virus (including *Simian retrovirus* 1 and 2 (SRV 1 and 2)

Genus: *Gammaretrovirus*
Gibbon ape leukaemia virus
Woolly monkey sarcoma virus

Genus: *Deltaretrovirus*
Primate T lymphocytic virus 1
Primate T lymphocytic virus 2
Primate T lymphocytic virus 3

Genus: *Lentivirus*
Simian immunodeficiency virus (SIV)
Genus: Spumavirus
Simian foamy virus (SFV) group
African green monkey foamy virus
Macaque simian foamy virus

Simian foamy virus

Mason-Pfizer (SRV 1 and 2) and SIV are associated with simian acquired immunodeficiency syndrome.

SFV is not associated with disease in humans or primates but may be transmitted to humans butchering and eating wild primates (Wolfe et al 2004) or who have close contact while handling captive primates (Heneine et al 1998; Khan et al 1999; Switzer et al 2004). Further human to human transmission of SFV, even between married couples, has not been described (Heneine et al 1998; Switzer et al 2004).

Other simian retroviruses are associated with various tumours or malignancies in primates.There is no evidence for the transmission of any of these viruses to humans.

While the simian retroviruses may be of interest because they are seen as models for studying human retroviral diseases, none has been associated with human disease and, therefore, they are not considered to be a hazard in the commodity.

5.1.32. Rabies virus

Rabies virus can infect most animal species and, although rare in primates, it can probably infect all species. Since rabies is invariably fatal in humans who do not receive early prophylactic treatment, it is considered to be a preliminary hazard in the commodity

5.1.33. Chikungunya virus

Chikungunya virus is an insect-borne alphavirus that infects a wide range of primates including humans. It is considered to be a preliminary hazard in the commodity

5.1.34. Mayaro fever virus

Mayaro fever is a non-fatal mosquito-borne disease that occurs in South and Central America (Johnston and Peters 1996a). The disease in humans is characterised by fever and influenza-like symptoms. Many cases are complicated by arthritis (Acha and Szyfres 1994; Johnston and Peters 1996b; Tesh 1982). The incubation period is short, followed by a febrile period which lasts for 3-6 days. Viraemia persists for 4-5 days after the onset of symptoms (Torres et al 2004). The main vectors are *Haemagogus* mosquitoes although other mosquitoes may also be involved (Acha and Szyfres 1994; Johnston and Peters 1996b). Marmosets and other New World primates have been shown to develop viraemia of short duration and it has been suggested that they may be amplifying hosts, with the maintenance hosts being birds or rodents (Acha and Szyfres 1994). Long-term carriage of the virus has not been reported. The disease has not been described in primate laboratory colonies or zoo populations.

Mayaro virus is transmitted by mosquito species which are not present in New Zealand. Because the virus is confined to South and Central America, the incubation and viraemic periods are short and a carrier state has not been reported, Mayaro virus is not considered to be a hazard in the commodity.

5.2. TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHY AGENTS (PRIONS)

Transmissible spongiform encephalopathy (TSE) agents include;

- Bovine spongiform encephalopathy (BSE) in cattle and a few other species
- Scrapie in sheep and goats
- Chronic wasting disease (CWD) in deer
- Transmissible mink encephalopathy (TME)
- Kuru in humans
- Creutzfeldt-Jakob disease in humans
- Variant Creutzfeldt-Jakob disease in humans infected with BSE agent.

Primates are not known to be affected with TSEs except for a few isolated cases that have been infected through eating rations containing meat meal produced from tissues contaminated with the BSE agent, or by experimental inoculation (Bons et al 1999; Herzog et al 2005; Sigurdson and Miller 2003). There is no evidence that primates play any part in maintenance or transmission of TSEs and they are not considered to be a hazard in the commodity.

5.3. BACTERIA

5.3.1. *Rickettsia conorii*

Antibodies to *Rickettsia conorii* have been found in wild vervet monkeys in South Africa (Kaschula et al 1978). The organism is transmitted by ticks, in which it is transmitted transovarially. Rodents are commonly infected (Acha and Szyfres 1994). There is no evidence that infection causes disease in monkeys or that primates are involved in the epidemiology of the infection. *Rickettsia conorii* is not considered to be a hazard in the commodity.

5.3.2. *Ehrlichia chaffeensis*

An outbreak of *Ehrlichia chaffeensis* infection in lemurs has been described (Williams et al 2002). The lemurs were apparently infected by *Amblyomma americanum* ticks which are the natural vectors. Infected lemurs showed no clinical signs (Yabsley et al 2004). The natural hosts of the organism are deer (Williams et al 2002) and primates are not known to play any role in maintenance of the agent. No reports of infection in primates other than lemurs were found. The tick vector, *A. americanum*, is not found in New Zealand.

Infection with *E. chaffeensis* is rare in lemurs and primates are not known to act as maintenance hosts. *Ehrlichia chaffeensis* is therefore not considered to be a hazard in the commodity.

5.3.3. *Borrelia burgdorferi*

Borrelia burgdorferi typically causes disease in humans and a variety of other animals. It is transmitted by ticks, particularly *Ixodes* spp., and the maintenance hosts are rodents and deer (Margaletic 2003; Matuschka et al 1992; Pawelczyk et al 2004). There is no evidence that primates play any role in transmission of the organism or act as maintenance hosts. Therefore, *B. burgdorferi* is not considered to be a hazard in the commodity.

5.3.4. *Brucella* species

The primary hosts of *Brucella abortus, B. melitensis* and *B. suis* are cattle, sheep and goats, and pigs respectively. Humans and other primates are dead end hosts and of no importance in the maintenance or spread of these organisms and *Brucella* spp. are not a hazard in the commodity.

5.3.5. *Enteric bacteria*

The *Code* recommends that nonhuman primates for export should be tested for enteric bacteria including *Salmonella* spp., S*higella* spp., *Yersinia* spp., pathogenic and toxigenic *Escherichia coli* and *Campylobacter* spp. Therefore, enteric organisms are regarded as a preliminary hazard in the commodity.

5.3.6. *Yersinia pestis*

Yersinia pestis is the aetiological agent of plague and has been responsible for massive epidemics in humans and is responsible for over 2,000 cases of disease annually (Cornelis 2000). Maintenance hosts are rodents and transmission is generally by fleas, although transmission by the respiratory route and by scratch and bite wounds also occurs (Davis et al 1975). However, primates are not considered maintenance hosts and healthy primates sourced from a controlled environment and free from ectoparasites will not transmit the infection. Therefore, *Y. pestis* is not considered to be a hazard in the commodity.

5.3.7. *Haemophilus influenzae*

No reports were found to suggest that primates can be infected with *Haemophilus influenzae* or act as maintenance hosts. In addition, *H. influenzae* is endemic in the New Zealand human population (Saravani et al 1992). Therefore, it is not considered to be a hazard in the commodity.

5.3.8. *Francisella tularensis*

Francisella tularensis is the aetiological agent of tularaemia, a serious disease of humans. Outbreaks of disease have been described in primates. Therefore, it is considered to be a preliminary hazard in the commodity.

5.3.9. *Mycobacterium tuberculosis* group

The *Code* recommends that nonhuman primates for export should be tested for infection with *Mycobacterium tuberculosis* or *M. bovis*. Other closely related species of bacteria include *M. caprae*, *M. canetti*, *M. africanum*, *M. microti* and *M. pinnipedii*. The group is referred to as the *M. tuberculosis* complex . These organisms are considered to be a preliminary hazard in the commodity.

5.3.10. *Mycobacterium leprae*

Mycobacterium leprae is the aetiological agent of leprosy. Naturally acquired and experimentally transmitted leprosy has been described in several species of primate (Meyers et al 1991; Wolf et al 1985). Leprosy is rare in primates and would not go undiagnosed in a captive population in a controlled environment. Transmission requires close contact. In the countries to which this risk analysis applies leprosy is extremely rare and the likelihood of a human becoming infected by an imported animal is considered to be negligible. Therefore, it is not considered to be a hazard in the commodity.

5.4. FUNGI

Over 100,000 species of fungus have been identified, but only 150 are known to cause disease in animals or 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 almost certainly many endemic species that have not yet been identified. With the exception of the dermatopytes, fungi are not primary pathogens but are opportunistic or secondary invaders, acquired from the environment. Infected animals are generally not contagious and infection from animal to animal either does not occur or is rare (Acha and Szyfres 1994; Picard and Vismer 2004; Various Authors 2006). No mention of any fungal disease, other than epizootic lymphangitis of horses, occurs in any of MAF's Import Health Standards or Overseas Market Access Requirements (OMARS), thus indicating that they are not considered by MAF or our trading partners to be important in the movement of animals from country to country. The only fungal disease in the *Code* is epizootic lymphangitis of horses.

The fungal agents listed in the Australian *Draft Generic Import Risk Analysis Report of Nonhuman Primates* are *Histoplasma capsulatum*, *H. duboisii*, *Coccidioides* spp. *Paracoccidioides brasiliensis* and three members of the order Entomorphthorales. However, these are all organisms that occur in soil and the environment and only infect animals and humans opportunistically. Further, infected animals are not contagious. Therefore, fungal disease agents are not considered to be a hazard in the commodity.

5.5. PROTOZOAL PARASITES

5.5.1. *Tritrichomonas mobilensis*

Tritrichomonas mobilensis is related to, but distinct from *Trichomonas foetus* and to *Tritrichomonas suis* (Felleisen 1998). It is commonly found in subclinically infected

squirrel monkeys (Culberson et al 1986, Scimeca et al 1989). It has also been associated with gastritis in rhesus macaques infected with SIV (Kondova et al 2005). *T. mobilensis* is a secondary pathogen of immunocompromised primates, in which it causes mild disease. There is no evidence to suggest that it is pathogenic to humans and it is, therefore, not considered to be a hazard in the commodity.

5.5.2. *Trypanosoma cruzi* and related trypanosomes

Trypanosoma cruzi is the cause of Chagas disease. *T. cruzi* and the related trypanosomes *T. minasense*, *T. devei* and *T. rangeli* are parasites of over 100 species of animals and humans (Bradley et al 2000). New World monkeys are commonly infected. Infected animals are not infectious, the parasites being transmitted by kissing bugs or triatomids. Kissing bugs are not carried on animals but periodically emerge from their hiding places to take blood meals on animals and humans (Krinsky 2002; Taylor et al 2007). Triatomids are not confined to the Americas; some species are also found in India, Southeast Asia and Africa (Krinsky 2002; Taylor et al 2007). However, despite innumerable movements of humans and animals, Chagas disease has remained confined to the southern United States, Central America and northern parts of South America. The likelihood that the triatomid vectors would either be introduced on primates or from the countries to which this risk analysis applies is negligible. Therefore, *T. cruzi*, *T. minasense*, *T. devei* and *T. rangeli* are not considered to be a hazard in the commodity.

5.5.3. *Trypanosoma brucei*

Trypanosoma brucei var *gambiens*e and *T. brucei* var *rhodesiense* are the two species of Afican trypanosomes that can infect humans (Connor and Van den Bossche 2004). All other species are either unable to infect humans or are non-pathogenic. Antibodies against *T. brucei* (Jeneby et al 2002) and RNA demonstrated by PCR (Njiokou et al 2004) have been found in primates but there is no evidence that these organisms cause disease in primates. Monkeys have been infected experimentally with *T. brucei* var *gambiens*e and *T. brucei* var *rhodesiense* (Ouwe-MissiI-Oukem-Boyer et al 2006) but these two species are transmitted naturally only by tsetse flies (*Glossina* spp.) and infected humans and primates are not contagious. These trypanosomes and their vectors are confined to parts of Africa and are not found in any country relevant to this risk analysis. Since tsetse flies could not be introduced with imported primates and could not establish in New Zealand even if they were, *T. brucei* var *gambiens*e and *T. brucei* var *rhodesiense* are not considered to be a hazard in the commodity.

5.5.4. *Leishmania* spp.

A number of *Leishmania* spp. can infect animals and humans. Some species have been transmitted experimentally to monkeys and baboons (Hailu et al 1995; Lawyer et al 1990). Infection is transmitted naturally by sand flies of the genera *Lutzomyia* and *Phlebotomus* (Van der Lugt and Stewart 2004) and infected animals are not contagious. Because the disease is not contagious and suitable insect vectors are not found in New Zealand, introduction of infected primates would not result in establishment of the disease. For these reasons *Leishmania* spp.are not considered to be a hazard in the commodity.

5.5.5. *Cyclospora* spp.

Cyclospora cayetanensis causes diarrhoea in humans and is often found in travellers. *Cyclospora* spp. found in primates include *C. cercopitheci* from African green (vervet) monkeys (*Chlorocebus aethiops*), *C. colobi* from black and white colobus monkeys (*Cercopithecus guereza*) and *C. papionis* from olive baboons (*Papio anubis*). However, *Cyclospora* spp. are strictly host specific (Eberhard et al 1999; Eberhard et al 2001; Lopez et al 1999; Smith et al 1996) and therefore primate *Cyclospora* spp. will not infect humans and are not considered to be a hazard in the commodity.

5.5.6. *Haemogregarina cynomolgi*

Haemogregarina is a genus of hemoprotozoans, parasitic mainly in cold-blooded vertebrates (Wikipedia 2010). *Haemogregarina cynomolgi* has been observed in blood of some species of Old World primates, such as macaques and baboons (Hegner 1928; Toft and Eberhard 1998), but no illness has been associated with infection and it is regarded as non-pathogenic. There is no evidence to suggest that this parasite is able to infect humans. Therefore it is not considered to be a hazard in the commodity.

5.5.7. *Plasmodium* spp.

Plasmodium spp. cause malaria in many species of animals. *P. falciparum, P. malariae, P. ovale, P. vivax* infect humans, with *P. falcifarum* being the most important (CDC 2004; World Health Organization 2009). At least 14 other species are known to infect primates (Escalante et al 1998). Primate parasites that are able to infect humans, but which do not cause serious disease are *P. inui, P. cynomolgi, P. simiovale, P. brasilianum, P. schwetz, P. eylesi* and *P. simium* (Acha and Szyfres 1994). *P. knowlesi*, also a simian parasite, may infect humans and cause serious disease (Cox-Singh et al 2008). The relationships between the various parasites and their preferred hosts, and their ability to also infect other hosts, is complex. However, all of the *Plasmodium* parasites of primates are transmitted by *Anopheles* spp.mosquitoes. Of the 430 species of *Anopheles*, between 30 and 50 are vectors of malaria (CDC 2004). Since *Anopheles* spp. are not found in New Zealand, and because infected primates are not contagious, *Plasmodium* spp. are not considered to be a hazard in the commodity.

5.5.8. *Babesia* spp. and *Entopolypoides* spp.

Several *Babesia* spp. and *Entopolypoides* spp. occur in primates (Bronsdon et al 1999; Jeneby et al 2008; Voorberg-vd Wel et al 2008). The *Babesia* and *Entopolypoides* spp. associated with primates are either apathogenic or pathogenic only in immunosuppressed or splenctomised individuals (Bronsdon et al 1999). Infections with these species in humans are rare. Infections with *B.microti* have been reported in immunocompromised, often splenectomised, humans. *Babesia divergens* and *B. bovis* have been found to infect non-splenectomised humans (Acha and Szyfres 1994; Zintl et al 2003). All *Babesia* and *Entopolypoides* spp. are transmitted by ticks, and infected animals are not contagious. *Haemaphysalis longicornis* is the only mammalian tick parasite in New Zealand and is capable of acting as a vector for several *Babesia* spp.(Heath 2002). The *Babesia* and *Entopolypoides* spp.of primates are basically apathogenic for humans and other primates. It is unlikely that imported primates would become parasitized by *H. longicornis* in New

Zealand zoos. Therefore, *Babesia* and *Entopolypoides* spp. are not considered to be a hazard in the commodity.

5.5.9. *Hepatocystis simia* and *Hepatocystis kochi*

Hepatocystis simia and *H. kochi* are parasites causing subclinical infections in Old and New World primates and they do not infect humans (Phillips-Conroy et al 1988; Seethamchai et al 2008; Takenaka et al 1990; Zeiss and Shomer 2001). These parasites are transmitted by *Culicoides* midges. Because they do not infect humans, and because there are no species of *Culicoides* present in New Zealand, *H. simia* and *H. kochi* are not considered to be a hazard in the commodity.

5.5.10. Enteric protozoal parasites

Giardia spp., *Entamoeba histolytica, Cryptosporidia* spp. *Balantidium coli* are all endemic in New Zealand and are therefore not considered to be a hazard in the commodity.

5.6. INTERNAL HELMINTH PARASITES

5.6.1. Trematodes and cestodes

The Australian *Draft Generic Import Risk Analysis of Nonhuman Primates* lists 11 species of cestodes or trematodes. However, there are many more species of cestodes and trematodes that may infect humans, either as primary or accidental hosts, and could conceivably infect primates. It is not practical or useful to consider each of these species individually. Most are broadly similar in that they require an intermediate host to complete their life cycle, are diagnosed by similar methods of faecal examination, and are treated with the same anthelminthics. Therefore, it is possible to devise general methods for preventing the introduction and establishment of these parasites. For this reason trematodes, considered as a consolidated group, are considered to be a hazard in the commodity. Similarly, cestodes are considered as a separate group and are considered to be a preliminary hazard in the commodity.

5.6.2. Nematodes and acanthocephalans

The Australian draft risk analysis lists 32 nematodes and acanthocephalans. As with the trematodes and cestodes, this list is likely to be incomplete and it is more practical to deal with all nematodes and acanthocephalans as a single group and they are considered to be a preliminary hazard in the commodity.

5.7. ARTHROPOD PARASITES

A large number of arthropod parasites may infest primates. These can be considered in two groups: namely insects, including fleas and lice, and arachnids, including ticks and mites. Since many arthropod parasites can cause disease or act as vectors for diseases, these are considered to be a preliminary hazard in the commodity

5.8. OTHER METAZOAN PARASITES

5.8.1. Pentastomids

Pentastomids have been described in a number of prosimians, New and Old World monkeys and great apes (Toft and Eberhard 1998). They occur rarely in humans and infestations are usually asymptomatic. Species belonging to three genera of the pentastomidae are found in snakes. The fourth genus is *Linguatula*, and *L. serrata* may be found in canids, domestic animals and humans. Adult parasites live in the nose of the primary host (dogs and, rarely, humans). They shed eggs in the nasal discharge. The eggs are taken up by intermediate hosts in which they develop through several larval stages to infectious nymphs. Infectious nymphs are typically found in lymph nodes or other organs of intermediate hosts such as rabbits and sheep. On ingestion by a final host (canids or, rarely, humans) the larvae migrate to the sites where they develop into adults (Acha and Szyfres 1994). Primates act as intermediate hosts (Toft and Eberhard 1998), however since primates in zoos are not eaten by humans or canids, pentastomids that might be introduced in imported primates would be unable to complete their life cycle and are not considered to be a hazard in the commodity.

5.8.2. Annelids

The leech *Dinobdella ferox* is found in southern Asia and occurs commonly in the nasal cavity of wild macaques. The leech has a direct life cycle, adults are hermaphrodites and eggs are shed from the nose of the host and attach to vegetation or other objects in water. After hatching immature leeches stay on the surface of the water and infestation of the nasal passages occurs when a host is drinking. The immature leeches remain in the nose of the host from a few days to several weeks (Toft and Eberhard 1998). The parasite has remained confined to southern Asia. It is known to infest humans. The parasites are unlikely to be present in the controlled environment from which the primates considered in this analysis will be imported. Therefore, *D. ferox* is not considered to be a hazard in the commodity.

5.9. PRELIMINARY HAZARD LIST

All agents considered to be preliminary hazards are consolidated into the following list:

Viruses

Chikungunya virus
Dengue virus
Filoviruses
Marburg virus
Ebola virus
Hepatitis A virus
Hepatitis B virus
Lymphocytic choriomeningitiis virus
Rabies virus

Yellow fever virus

Bacteria

Francisella tularensis
Mycobacterium spp.
Enteric bacteria

Internal parasites

Trematodes
Cestodes
Nematodes and acanthocephalans

Arthropod parasites

Insect parasites
Arachnid parasites

References

Some references were seen as abstracts in electronic databases.

Acha PN, Szyfres B (1994). *Zoonoses and Communicable Diseases Common to Man and Animals*. 2nd editon. Pan American Health Organization, Washington. 963 pages.

Anderson CR, Spence L, Downs WG, Aitken TH (1961). Oropouche virus: a new human disease agent from Trinidad, West Indies. *American Journal of Tropical Medicine and Hygiene*, 10, 574-8.

Anonymous (2009). Global polio eradication initiative. http://www.polioeradication.org/casecount.asp, Accessed 4/3/2009.

Antonsson A, Hansson BG (2002). Healthy skin of many animal species harbors papillomaviruses which are closely related to their human counterparts. *Journal of Virology*, 76(24), 12537-42.

Balayan MS (1992). Natural hosts of hepatitis A virus. *Vaccine*, 10 Suppl 1, S27-31.

Bons N, Mestre-Frances N, Belli P, Cathala F, Gajdusek DC, Brown P (1999). Natural and experimental oral infection of nonhuman primates by bovine spongiform encephalopathy agents. *Proceedings of the National Academy of Sciences*, 96(7), 4046-51.

Bower WA, Nainan OV, Han X, Margolis HS (2000). Duration of viremia in hepatitis A virus infection. *Journal of Infectious Diseases*, 182(1), 12-7.

Bradley KK, Bergman DK, Woods JP, Crutcher JM, Kirchhoff LV (2000). Prevalence of American trypanosomiasis (Chagas disease) among dogs in Oklahoma. *Journal of the American Veterinary Medical Association*, 217(12), 1853-7.

Bronsdon MA, Homer MJ, Magera JM, Harrison C, Andrews RG, Bielitzki JT, Emerson CL, Persing DH, Fritsche TR (1999). Detection of enzootic babesiosis in baboons (*Papio cynocephalus*) and phylogenetic evidence supporting synonymy of the genera *Entopolypoides* and *Babesia*. *Journal of Clinical Microbiology*, 37(5), 1548-53.

Buller RM, Arif BM, Black DN, Dumbell KR, Esposito JJ, Lefkowitz EJ, McFadden g, Moss B, Mercer AA, Moyer RW, Skinner MA, Tripathy DN (2005). *Genus Yatapox.* In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. Pp. 121.

Bunning MI, Wilson TM, Bowen RA (2004). West Nile virus infection. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 1004-11.

CDC (2003). Update: multistate outbreak of monkeypox--Illinois, Indiana, Kansas, Missouri, Ohio, and Wisconsin, 2003. *Morbidity and Mortality Weekly Report*, 52(27), 642-6.

CDC (2004). Malaria.http://www.cdc.gov/Malaria/impact/index.htm, Accessed 25/3/2009.

CDC (2006). Japanese encephalitis.http://www.cdc.gov/ncidod/dvbid/jencephalitis/#top, Accessed 27/1/2009.

Connor RJ, Van den Bossche P (2004). African animal trypanosomoses. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 251-296

Cornelis GR (2000). Molecular and cell biology aspects of plague. *Proceedings of the National Academy of Sciences*, 97(16), 8778-83.

Cox-Singh J, Davis TM, Lee KS, Shamsul SS, Matusop A, Ratnam S, Rahman HA, Conway DJ, Singh B (2008). *Plasmodium knowlesi* malaria in humans is widely distributed and potentially life threatening. *Clinical Infectious Diseases*, 46(2), 165-71.

Culberson DE, Pindak FF, Gardner WA, Honigberg BM (1986). *Tritrichomonas mobilensis* n. sp. (Zoomastigophorea: Trichomonadida) from the Bolivian squirrel monkey *Saimiri boliviensis* boliviensis. *Journal of Protozoology*, 33(2), 301-4.

Davis DHS, Hallet AF, Isaacson M (1975). Plague. In: Hubert WT, McCullogh CC, Schurrenberger R, (eds.), *Diseases Transmitted From Animals to Man*, 6th edition. Charles C Thomas, Springfield Illinois. 147-173.

Davison AJ, Eberle R, Hayward GS, McGeogh DJ, Minson AC, Pellett PE, Roizman B, Studdert MJ, Thirly E (2005). Genus Variocellovirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 200-201.

de Villiers E-M, Bernard H-U, Broker, t., Delius H, zur Hausen H (2005). Family Papillomaviridae. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 239-253

Dienstag JL, Davenport FM, McCollum RW, Hennessy AV, Klatskin G, Purcell RH (1976). Nonhuman primate-associated viral hepatitis type A. Serologic evidence of hepatitis A virus infection. *Journal of the American Medical Association*, 236(5), 462-4.

Downie AW, Taylor-Robinson CH, Caunt AE, Nelson GS, Manson-Bahr PE, Matthews TC (1971). Tanapox: a new disease caused by a pox virus. *British Medical Journal*, 1(5745), 363-8.

Eberhard ML, Nace EK, Freeman AR (1999). Survey for Cyclospora cayetanensis in domestic animals in an endemic area in Haiti. *Journal of Parasitology*, 85(3), 562-3.

Eberhard ML, Njenga MN, DaSilva AJ, Owino D, Nace EK, Won KY, Mwenda JM (2001). A survey for *Cyclospora* spp. in Kenyan primates, with some notes on its biology. *Journal of Parasitology*, 87(6), 1394-7.

Escalante AA, Freeland DE, Collins WE, Lal AA (1998). The evolution of primate malaria parasites based on the gene encoding cytochrome b from the linear mitochondrial genome. *Proceedings of the Natlional Academy of Sciences*, 95(14), 8124-9.

ESR (2009). *New Zealand Public Health Surveillance Report*. March 2009.http://surv.esr.cri.nz/PDF_surveillance/NZPHSR/2009/NZPHSR2009March.pdf, Accessed 4/3/2009.

Felleisen RS (1998). Comparative genetic analysis of tritrichomonadid protozoa by the random amplified polymorphic DNA technique. *Parasitology Research*, 84(2), 153-6.

Fenner F (1996). Yatapoxvirus infections. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 2691-2693.

Fleischauer AT, Kile JC, Davidson M, Fischer M, Karem KL, Teclaw R, Messersmith H, Pontones P, Beard BA, Braden ZH, Cono J, Sejvar JJ, Khan AS, Damon I, Kuehnert MJ (2005). Evaluation of human-to-human transmission of monkeypox from infected patients to health care workers. *Clinical Infectious Diseases*, 40(5), 689-94.

Gonzalez-Scarano F, Nathanson N (1996). Oropouche virus. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 1485.

Gough AW, Barsoum NJ, Gracon SI, Mitchell L, Sturgess JM (1982). Poxvirus infection in a colony of common marmosets (Callithrix jacchus). *Laboratory Animal Science*, 32(1), 87-90.

Griffin DE, Bellini WJ (1996). Measles virus. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 1267-312.

Hailu A, Negesse Y, Abraham I (1995). Leishmania aethiopica: experimental infections in non-human primates. *Acta Tropica*, 59(3), 243-50.

Hall AS, McNulty WP (1967). A contagious pox disease in monkeys. *Jornal of the American Veterinary Association*, 151(7), 833-8.

Heath ACG (2002). Vector competence of *Haemaphysalis longicornis* with particular reference to blood parasites. *Surveillance*, 29(4), 12-4.

Hegner R (1928). The evolutionary significance of the protozoan parasites of monkey and men. *Quarterly Review of Biology*, 31 (2), 225-244.

Heneine W, Switzer WM, Sandstrom P, Brown J, Vedapuri S, Schable CA, Khan AS, Lerche NW, Schweizer M, Neumann-Haefelin D, Chapman LE, Folks TM (1998). Identification of a human population infected with simian foamy viruses. *Nature Medicine*, 4(4), 403-7.

Herzog C, Riviere J, Lescoutra-Etchegaray N, Charbonnier A, Leblanc V, Sales N, Deslys JP, Lasmezas CI (2005). PrPTSE distribution in a primate model of variant, sporadic, and iatrogenic Creutzfeldt-Jakob disease. *Journal of Virology*, 79(22), 14339-45.

Heymann DL, Szczeniowski M, Esteves K (1998). Re-emergence of monkeypox in Africa: a review of the past six years. *British Medical Bulletin*, 54(3), 693-702.

Holmes GP, Chapman LE, Stewart JA, Straus SE, Hilliard JK, Davenport DS (1995). Guidelines for the prevention and treatment of B-virus infections in exposed persons. The B virus Working Group. *Clinical Infectious Diseases*, 20(2), 421-39.

Hutin YJ, Williams RJ, Malfait P, Pebody R, Loparev VN, Ropp SL, Rodriguez M, Knight JC (2001) Outbreak of human monkeypox, Democratic Republic of Congo, 1996-1997. *Emerging Infectious Diseases* 7(3), 434-438

Hutson CL, Lee KN, Abel J, Carroll DS, Montgomery JM, Olson VA, Li Y, Davidson W, Hughes C, Dillon M, Spurlock P, Kazmierczak JJ, Austin C, Miser L, Sorhage FE, Howell J, Davis JP, Reynolds MG, Braden Z, Karem KL, Damon IK, Regnery RL (2007). Monkeypox zoonotic associations: insights from laboratory evaluation of animals associated with the multi-state US outbreak. *American Journal of Tropical Medicine and Hygiene*, 76(4), 757-68.

Jeneby MM, Ngeiywa M, Yole DS, Mwenda JM, Suleman MA, Carlson HE (2008). Enzootic simian piroplasm (Entopolypoides macaci) in wild-caught Kenyan non-human primates. *Journal of Medical Primatology*, 37(6), 329-36.

Jeneby MM, Suleman MA, Gichuki C (2002). Sero-epizootiologic survey of *Trypanosoma brucei* in Kenyan nonhuman primates. *Journal of Zoo and Wildlife Medicine*, 33(4), 337-41.

Johnston RE, Peters CJ (1996a). Chikungunya virus. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition..Lippincott Raven, Philadelphia. 858-865.

Johnston RE, Peters CJ (1996b). Mayaro virus. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 866-867.

Kapikian AZ, Chanock RM (1996). Rotaviruses. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 1657-1708.

Kaschula VR, Van Dellen AF, de Vos V (1978). Some infectious diseases of wild vervet monkeys (*Cercopithecus aethiops pygerythrus*) in South Africa. *Journal of the South African Veterinary Association*, 49(3), 223-7.

Kaufmann AF, Gary GW, Broderson JR, Perl DP, Quist KD, Kissling RE (1973). Simian virus 16 associated with an epizootic of obscure neurologic disease. *Laboratory Animal Science*, 23(6), 812-8.

Khan AS, Sears JF, Muller J, Galvin TA, Shahabuddin M (1999). Sensitive assays for isolation and detection of simian foamy retroviruses. *Journal Clinical Microbiology*, 37(8), 2678-86.

King NW (2001). Rhabdinoviruses. In: Williams ES, Barker IK (eds.), *Infectious Diseases of Wild Animals*, third edition. Blackwell publishing. 153-157.

King NW, Hunt RD, Daniel MD, Melendez LV (1967). Overt herpes-T infection in squirrel monkeys (*Saimiri sciureus*). *Laboratory Animal Care*, 17(4), 413-23.

Knight JC, Novembre FJ, Brown DR, Goldsmith CS, Esposito JJ (1989). Studies on Tanapox virus. *Virology*, 172(1), 116-24.

Kondova I, Simon MA, Klumpp SA, MacKey J, Widmer G, Domingues HG, Persengiev SP, O'Neil SP (2005). Trichomonad gastritis in rhesus macaques (*Macaca mulatta*) infected with simian immunodeficiency virus. *Veterinary Pathology*, 42(1), 19-29.

Koopmans M, K,, Green KY, Ando T, Clarke IN, Estes MK, Matson DO, Nakata S, Neill JD, Smith AW, Studdert MJ, Thiel H-J (2005). Genus Vesivirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 848-849.

Krinsky Wl (2002). True bugs (Hemiptera). In: Mullen G, Durden L, (eds.), *Medical and Veterinary Entomology*. Academic Press, Amsterdam. 67-86.

Lawyer PG, Githure JI, Anjili CO, Olobo JO, Koech DK, Reid GD (1990). Experimental transmission of Leishmania major to vervet monkeys (Cercopithecus aethiops) by bites of Phlebotomus duboscqi (Diptera: Psychodidae). *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 84(2), 229-32.

Learned LA, Reynolds MG, Wassa DW, Li Y, Olson VA, Karem K, Stempora LL, Braden ZH, Kline R, Likos A, Libama F, Moudzeo H, Bolanda JD, Tarangonia P, Boumandoki P, Formenty P, Harvey JM, Damon IK (2005). Extended interhuman transmission of monkeypox in a hospital community in the Republic of the Congo, 2003. *American Journal of Tropical Medicine and Hygiene*, 73(2), 428-34.

Leland MM, Hubbard GB, Sentmore HT, 3rd, Soike KF, Hilliard JK (2000). Outbreak of Orthoreovirus-induced meningoencephalomyelitis in baboons. *Comparative Medicine*, 50(2), 199-205.

Linial ML, Fan H, Hahn B, Lwer r, J. N, Quackenbush S, Rethwilm A, Sonigo P, Stoye J, Tristem M (2005). Family Retroviridae. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses*. Elsevier Academic Press, Amsterdam. 421-440.

London WT (1997). Epizootiology, transmission and approach to prevention of fatal simian haemorrhagic fever in rhesus monkeys. *Nature*, 268, 344-5.

Lopez FA, Manglicmot J, Schmidt TM, Yeh C, Smith HV, Relman DA (1999). Molecular characterization of Cyclospora-like organisms from baboons. *Journal of Infectious Diseases*, 179(3), 670-6.

MAF (2006). *Risk Analysis Procedures*. Version 1, Ministry of Agriculture and Forestry, Wellington, New Zealand.

Mansfield K, King N (1998). Viral diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York.. 1-49.

Manson-Bahr PE, Downie AW (1973). Persistence of tanapox in Tana River valley. *British Medical Journal*, 2(5859), 151-3.

Margaletic J (2003). [Small rodents in the forest ecosystem as infectious disease reservoirs]. *Acta Medica Croatica*, 57(5), 421-6.

Martino MA, Hubbard GB, Butler TM, Hilliard JK (1998). Clinical disease associated with simian agent 8 infection in the baboon. *Laboratory Animal Science*, 48(1), 18-22.

Matuschka FR, Fischer P, Heiler M, Richter D, Spielman A (1992). Capacity of European animals as reservoir hosts for the Lyme disease spirochete. *Journal of Infectious Diseases*, 165(3), 479-83.

Meyers WM, Gormus BJ, Walsh GP, Baskin GB, Hubbard GB (1991). Naturally acquired and experimental leprosy in nonhuman primates. *Americn Journal of Tropical Medicine and Hygiene*, 44 (4 Pt 2), 24-7.

Monath TP, Heinz FX (1996). Kyasnur forest disease virus. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 1022

Myers MG, Vincent MM, Hensen SA, Tauraso NM (1972). Problems in the laboratory isolation of simian hemorrhagic fever viruses and isolation of the agent responsible for the Sussex/69 epizootic. *Applied Microbiology*, 24(1), 62-9.

Njiokou F, Simo G, Nkinin SW, Laveissiere C, Herder S (2004). Infection rate of *Trypanosoma brucei* s.l., *T. vivax*, *T. congolense* "forest type", and *T. simiae* in small wild vertebrates in south Cameroon. *Acta Tropica*, 92(2), 139-46.

OIE (2008). World Animal Health Information Database (WAHID) Interface. http://www.oie.int/wahid-prod/public.php?page=home, Accessed 27/1/2009.

Ouwe-MissiI-Oukem-Boyer O, Mezui-Me-Ndong J, Boda AC, Lamine I, Labrousse F, Bisser S, Boutteille B (2006). The vervet monkey (Chlorocebus aethiops) as an experimental model for trypanosoma brucei gambiense human African trypanosomiasis : a clinical, biological and pathological study. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 100(5), 427-36.

Pawelczyk A, Ogrzewalska M, Zadrozna I, Sinski E (2004). The zoonotic reservoir of Borrelia burgdorferisensu lato in the Mazury Lakes district of North-Eastern Poland. *International Journal of Medical Microbiology*, 293 Suppl 37, 167-71.

Peters CJ, Buchmeier M, Rollin PE, Ksiazek TG (1996). Arenaviruses. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 1521-1551.

Phillips-Conroy JE, Lambrecht FL, Jolly CJ (1988). Hepatocystis in populations of baboons (*Papio hamadryas* s.l.) of Tanzania and Ethiopia. *Journal of medical Primatologyl*, 17(3), 145-52.

Picard JA, Vismer HF (2004). Mycoses. In: Coetzer JAW, Tustin RC (eds.). *Infectious Diseases of Livestock*. 2nd edition. Oxford University Press, Capetown: 2095-136.

Pinheiro FP, Travassos da Rosa AP, Gomes ML, LeDuc JW, Hoch AL (1982). Transmission of Oropouche virus from man to hamster by the midge *Culicoides paraensis*. *Science*, 215(4537), 1251-3.

Ramig RF, Ciarlet M, Mertens PPC, Dermody TS (2005). Genus Rotavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 484-496.

Saravani GA, Martin DR, Arriesgado MT (1992). *Haemophilus influenzae* in New Zealand: subtyping of isolates from invasive disease using outer membrane protein profiling. *New Zealand Medical Journal*, 105(935), 218-20.

Scimeca JM, Culberson DE, Abee CR, Gardner WA, Jr. (1989). Intestinal trichomonads (*Tritrichomonas mobilensis)* in the natural host *Saimiri sciureus* and *Saimiri boliviensis*. *Veterinary Pathology*, 26(2), 144-7.

Seethamchai S, Putaporntip C, Malaivijitnond S, Cui L, Jongwutiwes S (2008). Malaria and Hepatocystis species in wild macaques, southern Thailand. *American Journal of Tropical Medicine and Hygiene*, 78(4), 646-53.

Siegel JD, Rhinehart E, Jackson M, Chiarello L (2007). *Guideline for Isolation Precautions: Preventing Transmission of Infectious Agents in Healthcare Settings*. http://www.cdc.gov/ncidod/dhqp/pdf/guidelines/Isolation2007.pdf, Accessed 4/3/2009.

Sigurdson CJ, Miller MW (2003). Other animal prion diseases. *British Medical Bulletin*, 66, 199-212.

Smith HV, Paton CA, Girdwood RW, Mtambo MM (1996). *Cyclospora* in non-human primates in Gombe, Tanzania. *Veterinay Record*, 138(21), 528.

Snijder EJ, Brinton MA, Faaberg KS, Godeny EK, Gorbalenya AE, J. MN, Mengeling WL, Plagemann PGW (2005). Genus Arterivurus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 965-974.

Stich A, Meyer H, Kohler B, Fleischer K (2002). Tanapox: first report in a European traveller and identification by PCR. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 96(2), 178-9.

Sundberg JP, Shima AL, Adkison DL (1992). Oral papillomavirus infection in a pygmy chimpanzee (*Pan paniscus*). *Journal of Veterinary Diagostic Investigation*, 4(1), 70-4.

Switzer WM, Bhullar V, Shanmugam V, Cong ME, Parekh B, Lerche NW, Yee JL, Ely JJ, Boneva R, Chapman LE, Folks TM, Heneine W (2004). Frequent simian foamy virus infection in persons occupationally exposed to nonhuman primates. *Journal of Virology*, 78(6), 2780-9.

Takenaka T, Hashimoto K, Gotou H, Matsumoto S, Nishikawa T (1990). [Studies on Hepatocystis sp. in rhesus monkeys from Yunnan, China]. *Jikken Dobutsu*, 39(2), 273-9.

Taylor MA, Coop RL, Wall RL (2007). *Veterinary Parasitology*, 3rd edition. Blackwell Publishing, Oxford. Pp 904.

Tesh RB (1982). Arthritides caused by mosquito-borne viruses. *Annual Review of medicine*, 33, 31-40.

Toft JD, Eberhard ML (1998) Parasitic Diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York. 111-205.

Torres JR, Russell KL, Vasquez C, Barrera R, Tesh RB, Salas R, Watts DM (2004). Family cluster of Mayaro fever, Venezuela. *Emerging Infectious Diseases*, 10(7), 1304-6.

Van der Lugt JJ, Stewart CG (2004). Leishmaniosis. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 378-381.

Various Authors (2006). Fungal Diseases (12 chapters). In: Greene CE, (ed), *Infectious Diseases of the Dog and Cat*. Section III:, Saunders, Elsevier, St Louis. 533-665

Voorberg-vd Wel A, Kocken CH, Zeeman AM, Thomas AW (2008). Detection of new Babesia microti-like parasites in a rhesus monkey (Macaca mulatta) with a suppressed Plasmodium cynomolgi infection. *American Journal of Tropical Medicine and Hygiene*, 78(4), 643-5.

Whitley RJ (1996). Cercopithecine herpes virus 1 (B virus). In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 2623-2635.

Wikipedia (2010) *Haemogregarina.* http://en.wikipedia.org/wiki/Haemogregarina. Accessed 15/4/2010.

Williams CV, Van Steenhouse JL, Bradley JM, Hancock SI, Hegarty BC, Breitschwerdt EB (2002). Naturally occurring *Ehrlichia chaffeensis* infection in two prosimian primate species: ring-tailed lemurs (*Lemur catta*) and ruffed lemurs (*Varecia variegata*). *Emerging Infectious Diseases*, 8(12), 1497-1500.

Wolf RH, Gormus BJ, Martin LN, Baskin GB, Walsh GP, Meyers WM, Binford CH (1985). Experimental leprosy in three species of monkeys. *Science*, 227(4686), 529-31.

Wolfe LG, Griesemer RA, Farrell RL (1968). Experimental aerosol transmission of Yaba virus in monkeys. *Journal of the National Cancer Institute*, 41(5), 1175-95.

Wolfe ND, Switzer WM, Carr JK, Bhullar VB, Shanmugam V, Tamoufe U, Prosser AT, Torimiro JN, Wright A, Mpoudi-Ngole E, McCutchan FE, Birx DL, Folks TM, Burke DS, Heneine W (2004). Naturally acquired simian retrovirus infections in central African hunters. *Lancet*, 363(9413), 932-937

World Health Organization (2006). *Japanese encephalitis.* http://www.who.int/water_sanitation_health/diseases/encephalitis/en/, Accessed 27/1/2009

World Health Organization (2009). *Malaria.* http://www.who.int/mediacentre/factsheets/fs094/en/index.html, Accessed 25/3/2009.

Yabsley MJ, Norton TM, Powell MR, Davidson WR (2004). Molecular and serologic evidence of tick-borne Ehrlichiae in three species of lemurs from St. Catherines Island, Georgia, USA. *Journal of Zoo and Wildlife Medicine*, 35(4), 503-9.

Zamiatina NA, Andzhaparidze AG, Balaian MS, Sobol AV, Titova IP (1990). [Susceptibility of *Macaca rhesus* to infection with hepatitis A virus strains isolated from man and monkeys]. *Voprosy Virusologii*, 35(1), 30-3.

Zeiss CJ, Shomer N (2001). Hepatocystosis in a baboon (Papio anubis). *Contemporary Topics in Laboratory Animal Science*, 40(1), 41-2.

Zintl A, Mulcahy G, Skerrett HE, Taylor SM, Gray JS (2003). Babesia divergens, a bovine blood parasite of veterinary and zoonotic importance. *Clinical Microbiology Reviews*, 16(4), 622-36.

6. Chickungunya virus

6.1. HAZARD IDENTIFICATION

6.1.1. Aetiological agent

Family:*Togaviridae*. Genus: *Alphavirus*. Species: *Chikungunya virus* (Weaver et al 2005).

6.1.2. OIE list

Not listed.

6.1.3. New Zealand status

Does not occur in New Zealand.

6.1.4. Epidemiology

Chikungunya fever is endemic in large areas of Africa, Madagascar, India and associated islands, and South East Asia (Sudeep and Parashar 2008). In recent times a massive epidemic has been occurring in India and Indian Ocean islands and South East Asia. More than 200 reports of outbreaks have been posted on ProMED-mail[3] and the number of people infected runs into millions. Of the countries relevant to this risk analysis, Chikungunya fever has occurred only in Singapore (CDC 2008), and most recently in Italy (Stock 2009). However, because of its geographic location, northern Australia should be considered likely to become infected.

Outbreaks in Asia have resulted primarily from human-mosquito-human infections and primates are not considered to be significant in the epidemiology. In Africa, the situation is different and primates, particularly baboons and cercopithocene monkeys, are considered to be reservoirs of the virus. The disease is transmitted by mosquitoes, with *Aedes aegytii* and *A. albopictus* being the principal vectors in Asia while *A. furcifer* is the main vector in South Africa (Jupp and McIntosh 1990). Several other species of mosquitoes are also involved in Africa.

In Africa, primates are considered to be maintenance hosts. Vervet monkeys (Kaschula et al 1978) and baboons, great apes and numerous other simian primates and bush babies *(Galagao* spp.) are commonly infected (Mallnoski 1994). Infections in primates are subclinical. Baboons are the most important sources of infection. Macaques can also be infected but are not considered to be important sources of infection in Asian outbreaks. Baboons may have high titres of virus for up to 6 days (Mallnoski 1994). The incubation period in humans is from 2-12 days (CDC 2008) and viraemia lasts for about 5 days

[3] http://www.promedmail.org/

(Johnston and Peters 1996) It has been found that a viraemia of 7.0 $\log_{10} LD_{50}$/ml is infectious for mosquitoes (Jupp and McIntosh 1990). Virus has not been isolated from serologically positive primates (Harrison et al 1967). No reference could be found to suggest that primates are long-term carriers of the virus.

6.1.5. Conclusion

Primates may act as reservoir hosts for *Chikungunya virus* . It is possible, therefore, that primates could be imported during the incubation period of the disease while virus is present in the blood. However, as Chikungunya fever does not occur in any country of relevance to this risk analysis, with the exception of Singapore and Italy, only primates sourced from those two countries are assessed as being a potential hazard.

6.2. RISK ASSESSMENT

6.2.1. Entry assessment

A wide variety of primates may act as multiplying hosts for the virus, and recently infected primates could possibly be introduced from Singapore or Italy. However, it should be recognised that the small number of primates likely to be imported in zoos (which are registed containement facilities) constitute a very small risk compared to the very large number of human travellers who come from those countries where *Chikungunya virus* is endemic. In addition, the commodity definition specifies that primates must be sourced from a controlled environment which is under veterinary supervision. Since the incubation and viraemic periods are short, and long-term carriers are unknown, the likelihood of the virus being introduced is negligible.

6.2.2. Exposure assessment

Suitable vectors for the transmission of the virus are not found in New Zealand (Holder and Brown 1999) and therefore the virus would be unable to spread from any containment facility into which a potentially-infected primate might be introduced.

6.2.3. Risk estimation

Since the risk of entry is assessed to be negligible and the risk of introduction and establishment of *Chikungunya virus* are also assessed to be negligible, the virus is assessed not to be risk in the commodity.

References

Some references were seen as abstracts in electronic databases

CDC (2008). *Chikungunya*. http://www.cdc.gov/ncidod/dvbid/chikungunya/, Accessed 17/3/2009.

Harrison VR, Marshall JD, Guillous NB (1967). The presence of antibody to Chikungunya and other serologically related viruses in sera of sub-human primate imports to the United States. *Journal of Immunology*, 98, 979-91.

Holder P, Brown G (1999). The Mosquitoes of New Zealand. *Surveillance*, 26(4), 12-5.

Johnston RE, Peters CJ (1996). Chikungunya virus. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 858-65.

Jupp PG, McIntosh BM (1990). *Aedes furcifer* and other mosquitoes as vectors of Chikungunya virus at Mica, North Eatern Transvaal. South Africa. *Journal of the American Mosquito Control Programme*, 6(3), 415-20.

Kaschula VR, van Dellen AF, de Vos V (1978). Some infectious diseases of wild vervet monkeys (*Cercopithecus aethiops pygerythrus*) in South Africa. *Journal of the South African Veterinary Association*, 49(3), 223-7.

Mallnoski FJ (1994). Chikungunya fever. In: Beran GW, Steele JH, (eds.), *Handbook of Zoonoses*. CRC Press, Boca Raton, Florida. 103.

Stock I (2009). [Chikungunya fever- expanded distribution of a re-emerging tropical infectious disease]. *Medizinische Monatsschrift für Pharmazeuten*, 32(1), 17-26.

Sudeep AB, Parashar D (2008). Chikungunya: an overview. *Journal of Bioscience*, 33(4), 443-9.

Weaver SC, Frey TK, Huang HV, M. KR, Rice CM, Roehrig JT, Shope RE, Straus EG (2005). Genus Alphavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 1003-1008.

7. Dengue virus

7.1. HAZARD IDENTIFICATION

7.1.1. Aetiological agent

Family: *Flaviviridae*. Genus: *Flavivirus*. Species: *Dengue virus.* Four strains of the virus are known termed Dengue virus 1, 2, 3 and 4 (Thiel et al 2005).

7.1.2. OIE list

Not listed.

7.1.3. New Zealand status

Does not occur in New Zealand.

7.1.4. Epidemiology

Dengue fever occurs in South and Central America , some African countries, India, South East Asia, Pacific Island countries and north eastern parts of Australia. Of the countries of relevance to this risk analysis, dengue fever occurs in Singapore and part of Australia. The vectors for the disease are present in southern areas of the USA but dengue fever, other than introduced cases are rare, having last occurred in 1999. Since 1980 there has been an increasing incidence of the disease in Asia and South America. Tens of millions of cases occur each year and several billion people are at risk (CDC 2008).

Dengue fever generally runs a mild course in humans although a small percentage of cases develop into the serious and sometimes fatal haemorrhagic form of the disease. The incubation period is from 3-14 days and the viraemia lasts from 3-10 days (CDC 2008). The virus is transmitted by mosquitoes, particularly *Aedes aegyptii*, but other *Aedes* spp., including *A. albopictus*, may play a role. No information could be found to suggest that long-term carriers of *dengue virus* occur. In epidemics, transmission occurs in a human-mosquito-human cycle.

Monkeys of the species *Macacus*, *Cynomolgus*, *Cercopithecu*s, *Cercocebus* and *Papio* have been infected experimentally by mosquito bite or injection. Infection results in viraemia lasting 1-7 days but is essentially subclinical (Halstead 1981). Possible sylvatic monkey-mosquito cycles in Malaysia and Nigeria have been suggested. However, in epidemic situations, the disease occurs in a human-mosquito-human urban cycle (Monath and Heinz 1996).

7.1.5. Conclusion

Viraemia may occur in subclinically infected monkeys. Therefore, there is a non-negligible likelihood of introduction in primates sourced from countries where the dengue fever occurs. That is, from Singapore, the north eastern part of Australia and the southern USA. The virus is considered to be a potential hazard in primates imported from those countries.

7.2. RISK ASSESSMENT

7.2.1. Entry assessment

The commodity definition restricts primates to be imported to those which originate in a controlled environment under veterinary supervision. It should be recognised that the likelihood of introduction of *dengue virus* in a few primates imported into a containment facility is extremely small when compared to the likelihood of infected travellers arriving from endemically infected regions. Over 100 cases of dengue fever occur in New Zealand each year in people who have travelled abroad (ESR 2009). Compared to the current, demonstrable risk of *dengue virus* entering New Zealand, any additional risk from importation of primates is negligible.

7.2.2. Exposure assessment

As stated, many cases of dengue fever in travellers occur annually in New Zealand. However, because vectors for further transmission of the virus are not present (Holder and Brown 1999), the virus has been unable to not establish here. The likelihood of *dengue virus* establishing following importation of primates into a containment facility is thus assessed to be negligible.

7.2.3. Risk estimation

Since the likelihood of introduction and establishment of *dengue virus* is assessed as negligible, the virus is assessed not to be a risk in the commodity.

References

CDC (2008). *Dengue fever*.**http://www.cdc.gov/ncidod/dvbid/dengue/index.htm**, Accessed 19/3/2009.

ESR (2009). *New Zealand Public Health Surveillance Report*. March 2009.**http://surv.esr.cri.nz/PDF_surveillance/NZPHSR/2009/NZPHSR2009March.pdf**, Accessed 4/3/2009.

Halstead SB (1981). Dengue and dengue hemorrhagic fever. In: Beran GW, (eds.), *CRC Handbook Series in Zoonoses. Section B: Viral Zoonoses* Volume 1. CRC Press, Boca Raton, Florida. 421-436,

Holder P, Brown G (1999). The Mosquitoes of New Zealand. *Surveillance*, 26(4), 12-5.

Monath TP, Heinz FX (1996). Dengue fever viruses. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 997-1004,

Thiel HJ, Collett MS, Gould EA, Heinz FX, Houghton M, Meyers G, Purcell RH, Rice CM (2005). Genus Flavivirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.). *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 982-988,

8. Ebola virus

8.1. HAZARD IDENTIFICATION

8.1.1. Aetiological agent

Family:*Filoviridae*. Genus: *Ebolavirus*. Species: *Cote d'Ivoire ebolavirus, Reston ebolavirus, Sudan ebolavirus* and *Zaire ebolavirus* (Feldman et al 2005).

8.1.2. OIE list

Not listed.

8.1.3. New Zealand status

Does not occur in New Zealand.

8.1.4. Epidemiology

Ebola haemorrhagic fever is a disease of primates and humans with mortality rates of up to 88% with some strains of the virus. The disease in humans occurs in central and west African countries and Sudan. A variant of the virus is the Reston Ebola strain which causes disease and mortality in monkeys but, to date, has not caused mortality in humans (CDC 2006; Peters et al 1996). Reston Ebola virus was introduced into a primate colony in Reston, Virginia, with the importation of rhesus macaques from the Philippines and Indonesia. It was also introduced into Italy with macaques from the same institute in the Philippines. The virus causes morbidity and mortality in primates but although human contacts seroconverted they remained asymptomatic (Peters et al 1996). African viruses are virulent for both humans and primates but may vary in pathogenicity.

Outbreaks of Ebola haemorrhagic fever in humans are usually associated with hunting and butchering gorillas, chimpanzees or other primates to eat. Further human to human transmission occurs. Outbreaks occur in humans when active outbreaks are occurring in primates. (Bermejo et al 2006; CDC 2006; Formenty et al 1999; Leroy et al 2004; Nkoghe et al 2005; Rouquet et al 2005). However, primates, like humans, are accidental hosts and the maintenance hosts of the virus have not been identified. Bats have been shown to be infected and it has been suggested that they may be primary hosts (Leroy et al 2005). Contact between the index cases and bats has been described in two cases (Peters et al 1996).

The incubation period for the disease as appears to be 4-16 days (Peters et al 1996) or 2-21 days (CDC 2006). Virus can be isolated from serum or tissues during the first 15 days after infection but, if death does not supervene, is cleared from the blood in parallel with the appearance of antibody which appears after 14-21 days (Fisher-Hoch et al 1992). Although there have been isolated reports of longer persistence of virus, such as a case where it

persisted in the semen and anterior eye chamber of a patient for 12 weeks (Peters et al 1996), there is nothing in the literature to suggest that long-term human or primate carriers occur or play any role in the maintenance of the disease.

Experimental infection of rhesus and African green monkeys results in a disease similar to that seen in humans (Peters et al 1996).

Following initial outbreaks of mortality of imported primates, the USA instituted quarantine measure and standards for isolation facilities for imported monkeys (Roberts and Andrews 2008). The mortality rates of imported primates dropped markedly after introduction of a quarantine period of 31 days and titres of antibody against Ebola virus were very low and, in the few animals with antibody titres, virus could not be isolated (CDC 1991). There appears to be little risk of Ebola (or Marburg) virus infections after an adequate quarantine period (Schou and Hansen 2000).

No reports of outbreaks of Ebola haemorrhagic fever in primates in zoos could be located. The likelihood of occurrence and persistence of Ebola virus in zoos of the type considered in this analysis is negligible.

8.1.5. Conclusions

Ebola virus is very unlikely to occur in zoo primate populations. Long-term carriers do not occur and play no part in the epidemiology of the disease. Therefore, the likelihood of introduction in primates that originate in a controlled environment in any of the countries considered in this analysis, is assessed to be negligible. Therefore, Ebola virus is assessed not to be a hazard in the commodity.

References

Some references were seen as abstracts in electronic databases

Bermejo M, Rodriguez-Teijeiro JD, Illera G, Barroso A, Vila C, Walsh PD (2006). Ebola outbreak killed 5000 gorillas. *Science*, 314(5805), 1564.

CDC (1991). Update: nonhuman primate importation. *Morbity and Mortality Weekly Rreport*, 40(40), 684-5, 91.

CDC (2006). *Ebola hemorrhagic fever*. http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/ebola.htm, Accessed 25/3/2009.

Feldman H, Geisbert TW, Jahrling PB, Klenk H-D, Netesov SV, Peters CJ, Sanchez A, Swanepoel R, E. VV (2005). Genus Ebolavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 651-653.

Fisher-Hoch SP, Perez-Oronoz GI, Jackson EL, Hermann LM, Brown BG (1992). *Filovirus* clearance in non-human primates. *Lancet*, 340(8817), 451-3.

Formenty P, Boesch C, Wyers M, Steiner C, Donati F, Dind F, Walker F, Le Guenno B (1999). Ebola virus outbreak among wild chimpanzees living in a rain forest of Cote d'Ivoire. *Journal of Infectious diseases*, 179 Suppl 1, S120-6.

Leroy EM, Kumulungui B, Pourrut X, Rouquet P, Hassanin A, Yaba P, Delicat A, Paweska JT, Gonzalez JP, Swanepoel R (2005). Fruit bats as reservoirs of *Ebola virus*. *Nature*, 438(7068), 575-6.

Leroy EM, Rouquet P, Formenty P, Souquiere S, Kilbourne A, Froment JM, Bermejo M, Smit S, Karesh W, Swanepoel R, Zaki SR, Rollin PE (2004). Multiple Ebola virus transmission events and rapid decline of central African wildlife. *Science*, 303(5656), 387-90.

Nkoghe D, Formenty P, Leroy EM, Nnegue S, Edou SY, Ba JI, Allarangar Y, Cabore J, Bachy C, Andraghetti R, de Benoist AC, Galanis E, Rose A, Bausch D, Reynolds M, Rollin P, Choueibou C, Shongo R, Gergonne B, Kone LM, Yada A, Roth C, Mve MT (2005). [Multiple Ebola virus haemorrhagic fever outbreaks in Gabon, from October 2001 to April 2002]. *Bulletin de la Société de pathologie exotique*, 98(3), 224-9.

Peters CJ, Sanchez A, Rollin PE, Ksiazek TG, Murphy FA (1996). Marburg and Ebola viruses. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 1161-1176.

Roberts JA, Andrews K (2008). Nonhuman primate quarantine: its evolution and practice. *ILAR Journal*, 49(2), 145-56.

Rouquet P, Froment JM, Bermejo M, Yaba P, Delicat A, Rollin PE, Leroy EM (2005). Wild animal mortality monitoring and human Ebola outbreaks, Gabon and Republic of Congo, 2001-2003. *Emerging Infectious Diseases*, 11(2), 283-90.

Schou S, Hansen AK (2000). Marburg and Ebola virus infections in laboratory non-human primates: a literature review. *Comparative Medicine*, 50(2), 108-23.

9. Marburg virus

9.1. HAZARD IDENTIFICATION

9.1.1. Aetiological agent

Family:*Filoviridae*. Genus: *Marburgvirus*. Species: *Lake Victoria Marburgvirus* (Feldman et al 2005b).

9.1.2. OIE list

Not listed.

9.1.3. New Zealand status

Does not occur in New Zealand.

9.1.4. Epidemiology

Marburg virus is closely related to *Ebola virus*. Ebola haemorrhagic fever and Marburg haemorrhagic fever are similar with respect to epidemiology, symptoms and pathogenesis. *Marburg virus* was first isolated in Germany and Yugoslavia from humans who were infected through contact with tissues and blood of subclinically infected monkeys (*Cercopithecus aethiops*) imported from Uganda. Outbreaks of disease have occurred in Kenya, Uganda, the Congo and Angola. Several outbreaks have been described and the mortality rate varies from 25% to greater than 70% (Feldman et al 2005a). In a large outbreak in Uganda, the mortality rate was 88% (World Health Organization 2009). Although the initial outbreaks were associated with contact with monkeys, subsequent major outbreaks have not been attributed to an identified source and spread through human to human transmission occured. A major outbreak in the Congo was associated with workers in a mine (Bausch et al 2003; Bertherat et al 1999; Borchert et al 2006) and another outbreak involving more than 300 cases and greater than 80% mortality occurred in Angola (Smetana et al 2006; World Health Organization 2009).

The incubation period of Marburg haemorrhagic fever is 3-9 days (World Health Organization 2009) and long-term carriers have not been reported. Outbreaks are controlled by isolation of infectious cases. The success of this measure supports the contention that long-term carriage of the virus does not occur.

There is still uncertainty regarding what the natural reservoir host for the virus is, but bats have been suggested (Swanepoel et al 2007; Towner et al 2007).

9.1.5. Conclusion

Marburg virus is unlikely to occur in zoo primates. Long-term carriage does not occur. Therefore, the likelihood of introduction of the virus in primates that originate in a controlled environment in any of the countries considered in this analysis, is assessed to be negligible and *Marburg virus* is assessed to not be a hazard in the commodity.

References

Some references were seen as abstracts in electronic databases

Bausch DG, Borchert M, Grein T, Roth C, Swanepoel R, Libande ML, Talarmin A, Bertherat E, Muyembe-Tamfum JJ, Tugume B, Colebunders R, Konde KM, Pirad P, Olinda LL, Rodier GR, Campbell P, Tomori O, Ksiazek TG, Rollin PE (2003). Risk factors for Marburg hemorrhagic fever, Democratic Republic of the Congo. *Emerging Infectious Diseases*, 9(12), 1531-7.

Bertherat E, Talarmin A, Zeller H (1999). [Democratic Republic of the Congo: between civil war and the Marburg virus. International Committee of Technical and Scientific Coordination of the Durba Epidemic]. *MédecineTropicale :Rrevue du Corps de Santé Colonial*, 59(2), 201-4.

Borchert M, Mulangu S, Swanepoel R, Libande ML, Tshomba A, Kulidri A, Muyembe-Tamfum JJ, Van der Stuyft P (2006). Serosurvey on household contacts of Marburg hemorrhagic fever patients. *Emerging Infectious Diseases*, 12(3), 433-9.

CDC (1991). Update: nonhuman primate importation. *Morbity and Mortality Weekly Rreport*, 40(40), 684-5, 91.

Feldman H, Geisbert TW, Jahrling PB, Klenk H-D, Netesov SV, Peters CJ, Sanchez A, Swanepoel R, E. VV (2005a). Family Filoviridae. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 645-653.

Feldman H, Geisbert TW, Jahrling PB, Klenk H-D, Netesov SV, Peters CJ, Sanchez A, Swanepoel R, E. VV (2005b). Genus Marburgvirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 650-651.

Smetana J, Chlibek R, Vackova M (2006). [Outbreak of Marburg hemorrhagic fever in Angola]. *Epidemiologie, Mikrobiologie, Imunologie*. 55(2), 63-7.

Swanepoel R, Smit SB, Rollin PE, Formenty P, Leman PA, Kemp A, Burt FJ, Grobbelaar AA, Croft J, Bausch DG, Zeller H, Leirs H, Braack LE, Libande ML, Zaki S, Nichol ST, Ksiazek TG, Paweska JT (2007). Studies of reservoir hosts for Marburg virus. *Emerging Infectious Diseases*, 13(12), 1847-51.

Towner JS, Pourrut X, Albarino CG, Nkogue CN, Bird BH, Grard G, Ksiazek TG, Gonzalez JP, Nichol ST, Leroy EM (2007). Marburg virus infection detected in a common African bat. *PLoS One*, 2(1), e764.

World Health Organization (2009). *Marburg haemorrhagic fever*. http://www.who.int/mediacentre/factsheets/fs_marburg/en/, Accessed 25/3/2009.

10. Hepatitis A virus

10.1. HAZARD IDENTIFICATION

10.1.1. Aetiological agent

Family: *Picornaviridae*. Genus: *Hepatovirus*. Species: *Hepatitis A virus*. There are two distinct biotypes that are associated with different preferred hosts. The human biotype infects all species of primates including humans and the simian biotype infects green monkeys (*Chlorocebus* spp.) and cynomolgus monkeys (*Macaca fascicularis*). The biotypes have specific epitopes and can be distinguished by monoclonal antibodies, but they also have cross reacting antigens (Stanway et al 2005).

10.1.2. OIE list

Not listed.

10.1.3. New Zealand status

Hepatitis A occurs sporadically in humans in New Zealand. Ninety one cases were notified in the year to December 2008 (ESR 2009). It is a Section A disease notifiable to the Medical Officer of Health.

10.1.4. Epidemiology

Hepatitis A virus may infect humans and primates and occurs globally. In endemically infected countries with low standards of general hygiene, up to 100% of young children may be infected. Young children are readily infected but are resistant and often remain asymptomatic, while adults are more likely to develop symptoms (CDC 2009; Hollinger and Ticehurst 1996). In endemically infected countries, widespread infection of children results in a population that is largely immune and clinical disease is rare, but non-immune travellers are at risk. In developed countries with high standards of sanitation and a naïve population, sporadic outbreaks of disease occur (Hollinger and Ticehurst 1996).

The incubation period varies from 10-50 days with a mode of 1 month. *Hepatitis A virus* replicates in the liver during the incubation phase. Typically, viraemia` does not persist past the time when symptoms appear (Hollinger and Ticehurst 1996). However, one researcher has reported that viraemia may persist for 36-391 days, with an average of 95 days (Bower et al 2000). Relapses may occur up to 6 months after recovery from an initial attack. Excretion of the virus in the faeces occurs from 2 weeks before to 1 week after the onset of symptoms (CDC 2009). However, in two infants, using PCR viral RNA was detected in faeces for 4-5 months (Rosenblum et al 1991). However, detection of RNA does not necessarily equate to the presence of viable virus. Infection is self-limiting and does not result in chronic infection. Immunity is life-long (CDC 2009). Infections usually result in mild disease and mortality rates are low.

Human strains of *hepatitis A virus* produce disease in chimpanzees, owl monkeys, stump-tailed monkeys and several species of marmoset, but the condition is usually milder than in humans. Other primate species are susceptible to infection but the infections are subclinical (Hollinger and Ticehurst, 1996). The simian biotype of the virus does not infect humans (Hollinger and Ticehurst 1996). The virus has been transmitted from primates (usually chimpanzees) to humans on at least 35 occasions involving more than 200 persons. Infected persons were usually staff involved in care of the primates and infections resulted in mild or subclinical infections (Hollinger and Ticehurst 1996). A persistent state of infection does not occur in the primates involved.

Diagnosis is generally made using serological tests such as ELISA. High levels of IgM hepatitis A antibody indicate acute infection, high IgG antibody levels indicate past infection (Lemon 1997).

10.1.5. Conclusion

Since *Hepatitis A virus* infection may occur in primates and could be transmitted to humans, the likelihood of introduction in the commodity is non-negligible and the virus is considered to be a hazard in the commodity.

10.2. RISK ASSESSMENT

10.2.1. Entry assessment

Hepatitis A virus is distributed worldwide and may occur in primates. Therefore, the likelihood of entry in the commodity is non-negligible. However, the likelihood of the virus being introduced through importation of small numbers of primates imported into containment facilities (zoos) is extremely small compared to the likelihood of introduction by the hundreds of thousands of humans who enter New Zealand annually.

10.2.2. Exposure assessment

Imported primates will not be in contact with humans except for the few trained staff who work with them. Since transmission of *Hepatitis A virus* is by the feacal oral route, and usually occurs only through close contact, there is a non-negligible likelihood of transmission from an infected imported primate to a zoo staff member.

10.2.3. Consequence assessment

Hepatitis A virus causes a mild disease with a very low mortality. Infected humans excrete the virus for a short time in their faeces. The virus is already present in New Zealand and does not cause serious disease in humans. Since outbreaks of hepatitis A are usually restricted to a few people and the disease is mild, the consequences of introduction in primates imported into a containment facility (a zoo) is assessed to be negligible.

10.2.4. Risk estimation

Since the likelihood of entry or exposure is low and consequences are assessed to be negligible, the risk from *Hepatitis A virus* is assessed to be negligible and the virus is assessed not to not be a risk in the commodity.

References

Some references were seen as abstracts in electronic databases

Bower WA, Nainan OV, Han X, Margolis HS (2000). Duration of viremia in hepatitis A virus infection. *Journal of Infectious Diseases*, 182(1), 12-7.

CDC (2009). *Hepatitis A*. http://www.cdc.gov/hepatitis/HAV.htm, Accessed 6/3/2009.

ESR (2009). *New Zealand Public Health Surveillance Report*. March 2009. http://surv.esr.cri.nz/PDF_surveillance/NZPHSR/2009/NZPHSR2009March.pdf, Accessed 4/3/2009.

Hollinger FB, Ticehurst JR (1996). Hepatitis A virus. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 735-782.

Lemon SM (1997). Type A viral hepatitis:epidemiology, diagnosis and prevention. *Clinical Chemistry*, 43, 1494-9.

Rosenblum LS, Villarino ME, Nainan OV, Melish ME, Hadler SC, Pinsky PP, Jarvis WR, Ott CE, Margolis HS (1991). Hepatitis A outbreak in a neonatal intensive care unit: risk factors for transmission and evidence of prolonged viral excretion among preterm infants. *Journal of Infectious Diseases*, 164(3), 476-82.

Stanway G, Brown F, Christian P, Hovi T, Hyypia T, King A, M, Q., N.J. K, M. LS, Minor PD, Pallansch MA, Palmenberg AC, Skern T (2005). *Genus Hepatovirus.* In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball L, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 770-771.

11. Hepatitis B virus

11.1. HAZARD IDENTIFICATION

11.1.1. Aetiological agent

Family: Hepadnaviridae. Genus: Orthohepadnavirus. Species: *Hepatitis B virus.*

Hepatitis B viruses are currently classified into eight genotypes and a number of related viruses isolated from primates (Mason et al 2005). However, on-going research means that the classification is in a state of flux (Myers et al 2006; Simmonds 2001a; Simmonds 2001b; Starkman et al 2003). Several recombinants have been identified (Simmonds and Midgley 2005). The *Woolly monkey hepatitis B virus* found in woolly monkeys, which are a new world species, is distinct from other *Hepatitis B virus* species found in the great apes.

11.1.2. OIE list

Not listed.

11.1.3. New Zealand status

Hepatitis B virus is endemic in humans here. The prevalence of carriers may be as high as 1-3% of the European and 5.4-16% of the Maori population (Robinson et al 2005). Another study estimated that there could be 56,000 carriers of the virus amongst the 915,000 Maori, Asian and Pacific Islanders over 15 years of age (Gane 2005) . Hepatitis B is a Section B disease notifiable to the Medical Officer of Health.

11.1.4. Epidemiology

Hepatitis B virus occurs globally and causes a serious disease of humans in which it may become chronic, and carriers who excrete the virus in their faeces are common. Chronic hepatitis may result in the development of carcinoma.

Hepatitis B virus has been isolated from primates, particularly the great apes, and has been found in apes in zoos (Thornton et al 2001; Zuckerman et al 1978). It was previously assumed, since monkeys and great apes can be experimentally infected with human strains of hepatitis B virus (Zuckerman et al 1975), that infection resulted from transmission from humans. However, it is now clear that great apes (but not monkeys) are commonly infected in the wild (Makuwa et al 2006; Makuwa et al 2003; Starkman et al 2003). The genotypes affecting great apes and woolly monkeys are distinct from human genotypes (Grethe et al 2000; Hu et al 2000; Makuwa et al 2006; Mason et al 2005; Payne 2004; Robertson and Margolis 2002; Takahashi et al 2000; Warren et al 1999).

An animal attendant in contact with infected apes has been reported to have seroconverted asymptomatically (Linneman et al 1984). No records were found of infection with human genotypes of *Hepatitis B virus* occurring naturally in apes, or of ape genotypes causing disease in humans. It has been stated that "it is unlikely that the presence of HBVs indigenous to primates would have important public health implications" (Linneman et al 1984). There is no evidence that infection of humans with ape genotypes occurs more than occasionally or causes disease, but this question has not been resolved conclusively (Robertson 2001).

A recombinant vaccine is available for use in humans and its efficacy has been proven in challenge studies conducted in chimpanzees. The vaccine induces protection against various subtypes of the virus (Hollinger and Ticehurst 1996).

Diagnosis of *Hepatitis B virus* infection can be confirmed by detection of either antigen or antibody. Serological tests, including radioimmunoassay and ELISA, to detect surface antigen of the virus (HBsAg) can be used to diagnose early acute infection. Antibody against HBsAg indicates previous infection and immunity, or vaccination. Detection of antibody to the core antigen (HBcAg) indicates acute or chronic infection. Interpretation of serological results can be complex (Hollinger and Ticehurst 1996). Recently, many workers have favoured the use of PCR to detect viral nucleotide sequences. Real time PCR methods have been found to be sensitive and specific (Abe et al 1999; Chen et al 2001). PCR test kits are available commercially.

11.1.5. Conclusion of hazard identification

Hepatitis B virus can infect humans and great apes and causes serious disease in humans. Although it is unlikely that genotypes that infect apes will infect humans, this supposition remains to be resolved conclusively. Therefore, the likelihood that imported apes could expose their humans handlers is non-negligible and *Hepatitis B virus* is considered to be a hazard in the commodity.

11.2. RISK ASSESSMENT

11.2.1. Entry assessment

Hepatitis B virus has a worldwide distribution and some genotypes have been found in captive primates in zoos in England (Thornton et al 2001; Zuckerman et al 1978), the USA (Linneman et al 1984) and Australia (Payne 2004). Therefore, the likelihood of entry of the virus in the commodity is non-negligible

11.2.2. Exposure assessment

Hepatitis B virus has spread between apes held in zoo populations and amongst free-living wild apes and the primate genotype was transmitted to a human who, nevertheless, did not develop symptoms. The likelihood of transmission of virus introduced in imported primates to zoo primates and, possibly, to their human handlers is non-negligible.

11.2.3. Consequence assessment

Hepatitis B virus infects human and primates only and is not a risk to other animals, whether wild or domesticated. There is evidence that the genotypes of *Hepatitis B virus* found in primates are different from those found in humans and are unlikely to be a risk to humans. Since hepatitis B is already endemic in New Zealand, the introduction of the virus in a few primates imported into a containment facility would not alter the prevalence of the disease significantly. For these reasons, introduction of the virus in imported primates would not be important from a biosecurity perspective. However, it has not been unequivocally established that apes never carry human genotypes of the virus or that humans are resistant to primate genotypes. Since the *Hepatitis B virus* can cause serious disease in some humans, the potential consequences of introducing the virus are assessed to be non-negligible.

11.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. As a result, the risk from *Hepatitis B virus* in the commodity is assessed to be non-negligible and it is a risk in the commodity and risk management measures can be justified.

11.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of *Hepatitis B virus* in the commodity:

- Quarantine is not a suitable option for preventing the importation of the virus because infected primates may remain subclinical long-term carriers of the virus.
- There is no treatment that will eliminate the virus.
- The *Code* makes recommendations with respect to the disease (see below)
- Serological tests are available to detect antibodies in carrier animals.
- Virus isolation or PCR are available for detection of the virus in blood of carriers.
- The genotypes of the virus that infect apes are different from those that infect humans.

Article 6.12.6. of the *Code* makes the following relevant recommendations;

Certification and quarantine requirements for […] nonhuman primates [other than marmosets and tamarins] from premises under veterinary supervision

Veterinary Authorities of *importing countries* should require:

for prosimians, New World monkeys, Old World monkeys, gibbons and great apes from premises under veterinary supervision

1. the presentation of an *international veterinary certificate* attesting that the shipment meets the requirements specified in Article 6.12.3., and that the *animals*:

    a. are either born in the premises of origin or have been kept there for at least 2 years;

b. come from premises which are under permanent veterinary supervision, and where a suitable health monitoring programme is followed, including microbiological and parasitological tests as well as necropsies.
c. […]
d. […]
e. […]
f. […]
g. […]
h. were subjected to a diagnostic test for hepatitis B virus and their current status documented (gibbons and great apes only);

2. the placement of the *animals* in a *quarantine station* for at least 30 days, and during this period:

a. all *animals* to be monitored daily for signs of illness and, if necessary, subjected to a clinical examination;

b. all *animals* dying for any reason to be subjected to complete post-mortem examination at a laboratory approved for this purpose;

c. any cause of illness or death to be determined before the group to which the *animals* belong is released from quarantine;

d. […].

This risk analysis does not examine the importation of nonhuman primates from uncontrolled environments. Nevertheless, it should be noted that Article 6.12.4 of the *Code* makes recommendations for quarantine requirements for nonhuman primates from an uncontrolled environment. The recommendations relating to hepatitis B in Article 6.12.4 are that the animals should be tested twice while being held in quarantine, with the first test being carried out during the first week and the second test 3-4 weeks later The serological tests recommended are for anti-hepatitis B core antigen and for hepatitis B surface antigen.

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

Option 1: All the measures recommended in the *Code* (see above) could be required. No testing other than physical examination would be required except for gibbons and great apes which should be subjected to a diagnostic test.

Note: This option implicitly assumes that veterinary supervision in the zoo of origin is sufficient to ensure that hepatitis B does not occur in the colony. It is justified by the assumption that genotypes of *Hepatitis B virus* found in apes differ from those found in humans and are not harmful for humans. Further, it is a reasonable option since the virus is endemic in this country and the small increase in risk posed by limited importations into a containment facility make no significant difference to the overall biosecurity risk.

Option 2: Animals to be imported could be tested serologically on two occasions while being held in quarantine, with an interval of 3-4 weeks between tests. Tests used should be for detection of antibodies against hepatitis B core antigen and hepatitis B surface antigen.

Note: This is equivalent to the measure recommended in the *Code* for importation of nonhuman primates from an uncontrolled environment.

Option 3: Animals to be imported could be tested serologically on two occasions while being held in quarantine, with an interval of 3-4 weeks between tests. The test used should be a sensitive PCR method for the detection of antibodies against hepatitis B core antigen and hepatitis B DNA sequences.

Note: Since PCR is more sensitive for detection of hepatitis B antigen this is more stringent than Options 1 and 2.

References

Some references were seen as abstracts in electronic databases.

Abe A, Inoue K, Tanaka T, Kato J, Kajiyama N, Kawaguchi R, Tanaka S, Yoshiba M, Kohara M (1999). Quantitation of hepatitis B virus genomic DNA by real-time detection PCR. *Journal of Clinical Microbiology*, 37(9), 2899-903.

Chen RW, Piiparinen H, Seppanen M, Koskela P, Sarna S, Lappalainen M (2001). Real-time PCR for detection and quantitation of hepatitis B virus DNA. *Journal of Medical Virology*, 65(2), 250-6.

Gane E (2005). Screening for chronic hepatitis B infection in New Zealand: unfinished business. *New Zealand Medical Journal*, 118(1211), 1-5.

Grethe S, Heckel JO, Rietschel W, Hufert FT (2000). Molecular epidemiology of hepatitis B virus variants in nonhuman primates. *Journal of Virology*, 74(11), 5377-81.

Hollinger FB, Ticehurst JR (1996). Hepatitis B virus. In: Fields BN, Knipe DM, Howley PM (eds.), *Fields Virology*. Third edition. Lippincott Raven. 2739-2807.

Hu X, Margolis HS, Purcell RH, Ebert J, Robertson BH (2000). Identification of hepatitis B virus indigenous to chimpanzees. *Proceedings of the National Academy of Science, New York*, 4(1661-4)

Linneman CC, Kramer LW, Askey PA (1984). Familial clustering of hepatitis B infections in gorillas. *American Journal of Epidemiology*, 119(3), 424-30.

Makuwa M, Souquiere S, Telfer P, Bourry O, Rouquet P, Kazanji M, Roques P, Simon F (2006). Hepatitis viruses in non-human primates. *Journal of Medical Primatology*, 35(6), 384-7.

Makuwa M, Souquiere S, Telfer P, Leroy E, Bourry O, Rouquet P, Clifford S, Wickings EJ, Roques P, Simon F (2003). Occurrence of hepatitis viruses in wild-born non-human primates: a 3 year (1998-2001) epidemiological survey in Gabon. *Journal of Medical Primatolgy*, 32(6), 307-14.

Mason WS, Burrell CJ, Casey J, H. GW, R. HC, Kahn M, Langford R, Newbold J, Schaefer S, Taylor JM, Will H (2005). Genus Orthohepadnovirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 379-381.

Myers R, Clark C, Khan A, Kellam P, Tedder R (2006). Genotyping Hepatitis B virus from whole- and sub-genomic fragments using position-specific scoring matrices in HBV STAR. *Journal of General Virology*, 87(Pt 6), 1459-64.

Payne KL (2004). *Hepatitis B Virus in Silvery Gibbons.* Masters in Philosophy in Zoo and Wildlife Medicine Thesis, Murdoch University, Perth. http://wwwlib.murdoch.edu.au/adt/browse/view/adt-MU20050204.154840, Accessed 1/4/2009.

Robertson BH (2001). Viral hepatitis and primates: historical and molecular analysis of human and nonhuman primate hepatitis A, B, and the GB-related viruses. *Journal of Viral Hepatitis*, 8(4), 233-42.

Robertson BH, Margolis HS (2002). Primate hepatitis B viruses - genetic diversity, geography and evolution. *Review of Medical Virology*, 12(3), 133-41.

Robinson T, Bullen C, Humphries W, Hornell J, Moyes C (2005). The New Zealand Hepatitis B Screening Programme: screening coverage and prevalence of chronic hepatitis B infection. *New Zealand Medical Journal*, 118(1211), U1344.

Simmonds P (2001a). The origin and evolution of hepatitis viruses in humans. *Journal of General virology*, 82(Pt 4), 693-712.

Simmonds P (2001b). Reconstructing the origins of human hepatitis viruses. *Philosophical Transactions of the Royal Society, London. Biological Science*, 356(1411), 1013-26.

Simmonds P, Midgley S (2005). Recombination in the genesis and evolution of hepatitis B virus genotypes. *Journal of Virology*, 79(24), 15467-76.

Starkman SE, MacDonald DM, Lewis JC, Holmes EC, Simmonds P (2003). Geographic and species association of hepatitis B virus genotypes in non-human primates. *Virology*, 314(1), 381-93.

Takahashi K, Brotman B, Usuda S, Mishiro S, Prince AM (2000). Full-genome sequence analyses of hepatitis B virus (HBV) strains recovered from chimpanzees infected in the wild: implications for an origin of HBV. *Virology*, 267(1), 58-64.

Thornton SM, Walker S, Zuckerman JN (2001). Management of hepatitis B virus infections in two gibbons and a western lowland gorilla in a zoological collection. *Vetertinary Record*, 149(4), 113-5.

Warren KS, Heeney JL, Swan RA, Heriyanto, Verschoor EJ (1999). A new group of hepadnaviruses naturally infecting orangutans (Pongo pygmaeus). *Journal of Virology*, 73(9), 7860-5.

Zuckerman AJ, Scalise G, Mazaheri MR, Kremastinou J, Howard CR, Sorensen K (1975). Transmission of hepatitis B to the rhesus monkey. *Developments in Biological Standardization*, 30, 236-9.

Zuckerman AJ, Thornton A, Howard CR, Tsiquaye KN, Jones DM, Brambell MR (1978). Hepatitis B outbreak among chimpanzees at the London Zoo. *Lancet*, 23(2(8091)), 652-4.

12. Lymphocytic choriomeningitis virus

12.1. HAZARD IDENTIFICATION

12.1.1. Aetiological agent

Family: Arenaviridae. Genus: Arenavirus. Species: *Lymphocytic choriomeningitis virus*.

12.1.2. OIE list

Not listed.

12.1.3. New Zealand status

No publication was found to suggest that *lymphocytic choriomeningitis virus* occurs in New Zealand. When other countries require it, MAF certifies that New Zealand is free from lymphocytic choriomeningitis.[4]

12.1.4. Epidemiology

Overseas, *lymphocytic choriomeningitis virus* is found commonly in mice and other rodents. Five percent of mice in the USA carry the virus (CDC 2007) and in some buildings up to 50% may be carriers (Acha and Szyfres 1994). Mice infected *in utero* become persistent carriers. Transmission to humans is invariably from contact with rodents or their secretions or faeces. Transmission most commonly occurs from the house mouse, but pet hamsters and other rodents may also be responsible. Mice are essential to the maintenance of the virus (Acha and Szyfres 1994). Transmission from human to human is rare and has been recorded only *in utero* or by by blood transfusion.. In humans, the incubation period is 8-13 days (CDC 2007) and long-term carriers have not been reported. The disease is usually benign and infections are often asymptomatic, although severe disease, with a mortality of around 1%, may occur (CDC 2007). The disease typically has a biphasic febrile period corresponding to a biphasic viraemia (Moody and Johnson 2009).

Lymphocytic choriomeningitis virus causes callitrichid hepatitis (Stephensen et al 1991) in some species of New World primates known as callitrichids (marmosets and tamarins). Disease outbreaks have occurred in the USA and the United Kingdom (Childs and Wilson 1994) and in Germany (Asper et al 2001).The feeding of newborn mice to callitrichids has resulted in transmission of the virus (Childs and Wilson 1994; Montali et al 1993). There is no evidence that the callitrichids are long-term carriers. Although rhesus and cynomolgus monkeys are susceptible to experimental infection (Childs and Wilson 1994), natural infection and disease have not been described. Seroconversion without clinical signs may occur in New and Old World monkeys.

[4] For example, see http://www.biosecurity.govt.nz/exports/animals/omars/rabaniec.sin.htm

12.1.5. Conclusion of hazard identification

Primates are not maintenance hosts or long-term carriers of the virus. Since primates will be imported only from a controlled environment, where they will have been held under veterinary supervision, the likelihood of introduction of *lymphocytic choriomeningitis virus* in the commodity is negligible and it is considered not to be a hazard in the commodity

References

Some references were seen as abstracts in electronic databases.

Acha PN, Szyfres B (1994). *Zoonoses and Communicable Diseases Common to Man and Animals*. 2nd editon. Pan American Health Organization, Washington. 963 pages.

Asper M, Hofmann P, Osmann C, Funk J, Metzger C, Bruns M, Kaup FJ, Schmitz H, Gunther S (2001). First outbreak of callitrichid hepatitis in Germany: genetic characterization of the causative lymphocytic choriomeningitis virus strains. *Virology*, 284(2), 203-13.

CDC (2007). *Lymphocytic Choriomeningitis*. http://www.cdc.gov/ncidod/dvrd/spb/mnpages/dispages/lcmv/qa.htm, Accessed 2/4/2009.

Childs JE, Wilson LJ (1994). Lymphocytic choriomeningitis. In: Beran GW, Steele JH, (eds.), *Handbook of Zoonoses*. Section B: Viral. Second edition. CDC press, Boca Raton. 463-71.

Montali RJ, Scanga CA, Pernikoff D, Wessner DR, Ward R, Holmes KV (1993). A common-source outbreak of callitrichid hepatitis in captive tamarins and marmosets. *Journal of Infectious Diseases*, 167(4), 946-50.

Moody RD, Johnson DH (2009). *Lymphocytic Choriomeningitis*. http://emedicine.medscape.com/article/220796-overview, Accessed 2/4/2009.

Stephensen CB, Jacob JR, Montali RJ, Holmes KV, Muchmore E, Compans RW, Arms ED, Buchmeier MJ, Lanford RE (1991). Isolation of an arenavirus from a marmoset with callitrichid hepatitis and its serologic association with disease. *Journal of Virology*, 65(8), 3995-4000.

13. Rabies virus

13.1. HAZARD IDENTIFICATION

13.1.1. Aetiological agent

Family: *Rhabdoviridae*. Genus *Lyssavirus*. Species*: Rabies virus* (Tordo et al 2005). In addition to the true *Rabies virus* there are six closely related lyssaviruses which cause similar diseases.

13.1.2. OIE list

Listed.

13.1.3. New Zealand status

Rabies is an exotic, notifiable disease (Ministry of Agriculture and Forestry 2008) and a Section B disease notifiable to the Medical Officer of Health.

13.1.4. Epidemiology

Rabies is a disease of all mammals, including humans. Worldwide rabies causes up to 50,000 human deaths annually (Tordo et al 2005) It occurs in the United States, Canada and some European countries. Australia and several countries in Europe, including Great Britain, Ireland, Sweden, Norway, Denmark, Finland, Portugal and Greece are free from rabies, but some of these countries have common borders with infected countries (OIE 2008b). In all endemically infected countries, the virus is maintained in a population of domestic or wild carnivores or bats. In some European countries and in Australia, related lyssaviruses occur in bats (Fooks et al 2003; Swanepoel 2004; Thompson 1999).

Rabies is characterised by severe nervous signs and is invariably fatal. The virus is carried mainly by carnivores and, in the final stages of the disease, they excrete the virus in saliva and transmit it to other animals and humans when they bite them. In countries where monkeys are present, it is not unusual for humans to be bitten by them (Eslamifar et al 2008; Ichhpujani et al 2008). Rabies is rare in primates although a few cases have been confirmed in chimpanzees, cebus, and squirrel monkeys (Richardson and Humphrey 1971; Anonymous 1988b). Rabies has been transmitted to humans by monkey bites (Anonymous 1988a; Ichhpujani et al 2008).

Rabies may be transmitted by means other than bites, but transmission by aerosol, such as may occur in bat colonies (Swanepoel 2004) or *per os,* as has been reported in kudu (Hubschle 1988), is rare.

Following deposition in a bite wound, rabies virus enters peripheral nerves and is transported through the nerves to the central nervous system. After entering the peripheral nerves, the virus is

not found in any other body tissues or in the blood. Amputation of limbs of mice experimentally inoculated into the footpads has been shown to prevent the virus from progressing to the brain (Swanepoel 2004). The passage of virus through the nervous system is a slow process and, depending on the site of infection, the dose of virus and the animal concerned, the incubation period may vary from weeks to, rarely, years (Dietzschold et al 1996). Viraemia is an exceptional event except in experimental infections of young mice with large doses of virus (Swanepoel 2004). *Rabies virus* spreads to the salivary glands at about the stage that there is generalized dissemination of infection in the brain. It then multiplies in the salivary glands and is excreted in the saliva. In the terminal stages of the disease, animals become incoordinated and may be aggressive. The disease invariably ends fatally.

Killed vaccines are used in humans for both pre- and post-exposure immunization. Vaccines used include inactivated virus produced in diploid human cells, duck embryos, rhesus monkey diploid cells and chick embryo cells (CDC 1988; CDC 1998; Dietzschold et al 1996). Vaccination of primates is not commonly practised, but primates have been used for experimental testing of many types of vaccines, including those used in humans (Kessler et al 1982; Lavender 1973; Lodmell et al 1998; Rupprecht et al 1992) .

There are no tests that can detect infection during the incubation stage of rabies.

13.1.5. Conclusion of hazard identification

Rabies is a serious, invariably fatal, disease that can affect all mammals. Although it rarely occurs in primates, it is present in several of the countries considered in this risk analysis. Therefore, it is considered to be a hazard in the commodity

13.2. RISK ASSESSMENT

13.2.1. Entry assessment

The likelihood of introducting rabies virus in primates imported from Australia England, Luxembourg, Belgium, Switzerland and the Scandinavian countries is negligible since the disease does not occur in these countries. Since the disease is rare in primates, and extremely rare in those sourced from the controlled environment of a zoo, the likelihood of introducing the virus is extremely low but non-negligible.

13.2.2. Exposure assessment

If an infected animal were to be imported it would become rabid, and could bite other primates with which it is in contact and zoo staff would also be at risk. Therefore, the likelihood of exposure is assessed to be non-negligible.

13.2.3. Consequence assessment

The consequences of an introduction of rabies would be severe; it could result in the death of primates other than the imported case or even human attendants. However, it would be extremely unlikely for rabies to spread beyond the containment facility.

13.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible and the risk estimate for *Rabies virus* is non-negligible. Therefore, risk management measures can be justified.

13.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of *Rabies virus* in the commodity:

- Rabies is an inevitably fatal disease of humans and other mammals
- The incubation period for rabies may, on rare occasions, extend to a year or more
- Effective vaccines are available for use in humans and nonhuman primates

The *Code* (Article 6.12.6.) recommends that nonhuman primates should "come from premises in which no case of tuberculosis or other zoonoses including rabies has occurred during the last 2 years prior to shipment in the building where the animals were kept"

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

Option 1: Animals to be imported should be certified as having been born in, and lived their entire life in, a captive primate population in which no case of rabies has occurred during the previous 2 years.

Note: this is equivalent to the *Code* recommendation.

Option 2: Animals to be imported should be certified as having been born in, and lived their entire life in, a country free from rabies.

Note: this would exclude importation from several of the countries considered in this risk analysis.

Option 3: When importing from a country where rabies occurs, animals to be imported should be certified as having been vaccinated at least 6 months prior to export with an effective inactivated rabies vaccine; and

a. Subjected to a serological test to confrm seroconversion following vaccination; or
b. Been kept in isolation from other primates not part of the importation for the 6 months immediately prior to shipment.

References

Some references were seen as abstracts in electronic databases.

Anonymous (1988a). Imported human rabies -- Australia, 1987. *Morbidity and Mortality Weekly Report*, 37(22), 351-3.

Anonymous (1988b). *Significant Zoonotic Disease in Nonhuman Primates*. Report from the Division of Veterinary Medicine, Walter Reed Army Institute, Washington DC. http://netvet.wustl.edu/species/primates/primzoon.txt, Accessed 2/4/2009.

CDC (1988). A new rabies vaccine for use in humans. *Morbidity and Mortality Weekly Report*, 37(14)(217-218), 223.

CDC (1998). Availability of new rabies vaccine for human use. *Morbidity and Mortality Weekly Report*, 47(01)(12), 19.

Dietzschold R, Rupprecht CE, Fu ZF, Koproowske H (1996). Rhabdoviruses. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 1137-1159.

Eslamifar A, Ramezani A, Razzaghi-Abyaneh M, Fallahian V, Mashayekhi P, Hazrati M, Askari T, Fayaz A, Aghakhani A (2008). Animal bites in Tehran, Iran. *Archives of Iranian Medicine*, 11(2), 200-2.

Fooks AR, Brookes SM, Johnson N, McElhinney LM, Hutson AM (2003). European bat lyssaviruses: an emerging zoonosis. *Epidemiology and Infection*, 131(3), 1029-39.

Hubschle OJ (1988). Rabies in kudu antelope. *Reviews of Infectious Diseases*, 19(Supplement 4), S629-33.

Ichhpujani RL, Mala C, Veena M, Singh J, Bhardwaj M, Bhattacharya D, Pattanaik SK, Balakrishnan N, Reddy AK, Samnpath G, Gandhi N, Nagar SS, Shiv L (2008). Epidemiology of animal bites and rabies cases in India. A multicentric study. *Journal of Communicable Diseases*, 40(1), 27-36.

Kessler MJ, Summer JW, Baer GM (1982). Evaluation of killed rabies vaccine for rhesus monkeys (*Macaca mulatta*). *Journal of Zoo Animal Medicine*, 13, 74-7.

Lavender JF (1973). Immune respose in primates vaccinated with duck embryo cell culture rabies vaccine. *Applied and Environmental Virology*, 25(3), 327-31.

Lodmell DL, Ray NB, Parnell MJ, Ewalt LC, Hanlon CA, Shaddock JH, Sanderlin DS, Rupprecht CE (1998). DNA immunization protects nonhuman primates against rabies virus. *Nature Medicine*, 4(8), 949-52.

Ministry of Agriculture and Forestry (2008). The Unwanted Organisms Register, http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, Accessed 27/1/2009.

OIE (2008a). World Animal Health Information Database (WAHID) Interface. http://www.oie.int/wahid-prod/public.php?page=home, Accessed 27/1/2009.

OIE (2008b). World Animal Health Information Database (WAHID) Interface. http://www.oie.int/wahid-prod/public.php?page=home, Accessed 27/1/2009.

Richardson JH, Humphrey GL (1971). Rabies in imported nonhuman primates. *Laboratory Animal Science*, 21(6), 1083.

Rupprecht CE, Hanlon CA, Cummins LB, Koprowski H (1992). Primate responses to vaccinia-rabies recombinant virus vaccine. *Vaccine*, 10(6), 368-74.

Swanepoel R (2004). Rabies. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 1123-1182.

Thompson GK (1999). Veterinary surgeon's guide to Australian bat lyssavirus. *Australian Veterinary Journal*, 77(11), 710-2.

Tordo N, Benmansour A, Calisher C, Dietzgen RG, Fang R-X, Jackson AO, Kurath GN, Nadin-Davis S, Tesh RB, Walker PJ (2005). Genus Lyssavirus. In: Fauquet CM, Mayo MA, Maniloff J, Desselberger U,

Ball LA, (eds.), *Eighth Report of the International Committee on Taxonomy of Viruses.* Elsevier Academic Press, Amsterdam. 630-633.

14. Yellow fever virus

14.1. HAZARD IDENTIFICATION

14.1.1. Aetiological agent

Family: *Flaviviridae*. Genus: *Flavivirus*. Species: *Yellow fever virus*.

14.1.2. OIE list

Not listed.

14.1.3. New Zealand status

Does not occur in New Zealand. It is a Section B disease notifiable to the Medical Officer of Health.

14.1.4. Epidemiology

Yellow fever virus is mosquito-borne and causes serious, frequently fatal, disease in humans. It is confined to tropical parts of South America and Africa, where the virus infects primates (CDC 2007). The virus has never established in Asia (Vainio and Cutts 1998). In South America, cases are confined to those transmitted in a monkey-mosquito-human cycle in people who enter the jungle. In Africa, large outbreaks occur in a human-mosquito-human cycle (Monath and Heinz 1996). The incubation period of yellow fever in humans varies from 3-6 days and viraemia is of short duration (Monath and Heinz 1996). In primates the virus is eliminated as antibodies are produced and viraemia generally lasts for about 6 days only, with a range of 2-9 days (Mansfield and King 1998; Vainio and Cutts 1998). There is solid immunity in humans and primates who recover from yellow fever. In African monkeys, viraemia occurs but infection is subclinical or is associated with minimal signs only (Mansfield and King 1998). Species of African primates which may be infected include baboons, magabeys, African green, colobus and patas monkeys. In South America, epizootics have been described in howler, spider and squirrel monkeys and capuchin and woolly monkeys may be infected but are less susceptible to disease (Mansfield and King 1998).

Yellow fever virus is transmitted primarily by *Aedes aegypti* mosquitoes. *Aedes* (*Stegomyia*) *aegypti*, *A. (Stegomyia) africanus*, *A.* (*Stegomyia*) *opok*, *A.* (*Stegomyi*a) *luteocephalus*, *A.* (*Stegomyia*) *simpsoni* group, *A.* (*Diceromyia*) *furcifer*, and *A.* (*Diceromyia*) *taylori* play a role in Africa and *Haemagogus* spp.are important in South American sylvatic cycles. Other species of mosquito, including *A. albopictus*, have also been implicated (Vainio and Cutts 1998). The virus is maintained by transovarial transmission in mosquitoes.

Yellow fever has remained confined to tropical Africa and South America and competent vectors are not found in New Zealand.

14.1.5. Conclusion of hazard identification

Yellow fever is not present in any of the countries considered in this risk analysis. Competent vectors for the virus are not found in New Zealand. Therefore, yellow fever is not considered to be a hazard in the commodity.

References

CDC (2007). *Yellow fever. Yellow fever global map.* http://www.cdc.gov/ncidod/dvbid/yellowfever/YF_GlobalMap.html, Accessed 8/4/2009.

Mansfield K, King N (1998). Yellow fever virus. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York.. 32-33.

Monath TP, Heinz FX (1996). Yellow fever. In: Fields BN, Knipe DM, Howley PM (eds.).*Fields Virology*. Third edition. Lippincott Raven, Philadelphia. 1009-1016.

Vainio J, Cutts F (1998). *Yellow Fever*. World Health Organization, Geneva. http://www.who.int/vaccines-documents/DocsPDF/www9842.pdf. Accessed 8/4/2009.

15. *Francisella tularensis*

15.1. HAZARD IDENTIFICATION

15.1.1. Aetiological agent

The bacterium *Franciscella tularensis.* There are two main types; *F. tularensis* subsp. *tularensis* (Type A) occurs in North America and *F. tularensis* subsp. *palaeartica* (Type B) in America and Eurasia.

15.1.2. OIE list

Not listed.

15.1.3. New Zealand status

Does not occur in New Zealand.

15.1.4. Epidemiology

Tularaemia occurs mainly in the northern hemisphere. *F. tularensis* subsp *tularensis* (Type A) is mainly associated with lagomorphs and is highly virulent for humans and domestic rabbits. It is transmitted primarily by ticks and biting flies. *F. tularensis* subsp. *palaeartica* (Type B) is found mainly in aquatic rodents such as beavers and muskrats in North America, and in hares and small rodents in Eurasia. It is less virulent for humans (Morner 1992; Morner 2008). In a large outbreak in Kosovo, in which 715 cases were reported, no deaths occurred (World Health Organization 2009a). Tularaemia has been reported in over 250 species including humans, mammals, birds, fish, amphibians, arthropods and protozoa (Morner 1992). Many animals are subclinically infected but develop antibody (Acha and Szyfres 1994).

The disease is commonly transmitted by ticks, especially in North America, but also through handling infected animals or carcasses. The Eurasian strains of *F. tularensis* are commonly transmitted by mosquitoes or direct contact, but also by water, contaminated food or inhalation (Morner 2008). The infective dose is extremely small and, since infected animals excrete the organism in urine and faeces, there is a high risk of transmission to humans (Morner 2008). Nevertheless, humans are accidental hosts and human to human transmission is believed not to occur (New South Wales Government: Department of Health Undated; Tidy 2007; World Health Organization 2009a; World Health Organization 2009b).

Tularaemia usually occurs sporadically but occasional outbreaks are also reported. The incubation period in humans varies from 1-10 days (Acha and Szyfres 1994).

Tularaemia is uncommon in primates although outbreaks have been reported. Little has been written about the disease in primates. The disease has been described in lemurs and New World monkeys, a gorilla and orangutans in the USA and in a golden-headed lion tamarin and a common marmoset in Switzerland (Matz-Rensing et al 2007). An epidemic in which six of 35 cynomolgus monkeys (*Macaca fascicularis*) died and 12 seroconverted over a 2 year period has been reported (Matz-Rensing et al 2007). No evidence could be found that primate to primate transmission occurs and, in several cases, there has been circumstantial evidence of contact with squirrels or rodents with possible transmission to primates by fleas. It is probable that primates, like humans, are accidental hosts and not contagious.

15.1.5. Conclusion of hazard identification

Tularaemia occurs rarely in primates. It is seldom reported from the southern hemisphere, indicating that it has not established in this part of the world. There is no evidence that primates are contagious or carriers of the disease. *Francisella tularensis* is considered to not be a hazard in the commodity.

References

Acha PN, Szyfres B (1994). *Zoonoses and Communicable Diseases Common to Man and Animals*. 2nd editon. Pan American Health Organization, Washington. 963 pages.

Matz-Rensing K, Floto A, Schrod A, Becker T, Finke EJ, Seibold E, Splettstoesser WD, Kaup FJ (2007). Epizootic of tularemia in an outdoor housed group of cynomolgus monkeys (*Macaca fascicularis*). *Veterinary Pathology*, 44(3), 327-34.

Morner T (1992). The ecology of tularaemia. *Revue Scientifique et Technique*, 11(4), 1123-30.

Morner T (2008). *Tularaemia.* In: *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*, sixth edition. OIE, Paris. 361-366,

New South Wales Government: Department of Health (Undated). *Tularaemia*. http://www.health.nsw.gov.au/factsheets/guideline/tularaemia.html, Accessed 9/4/2009.

Tidy C (2007). *Tularaemia*. http://www.patient.co.uk/showdoc/40000341/, downloaded 9/4/2009.

World Health Organization (2009a). *2002-Tularaemia in Kosovo -Update 2*. http://www.who.int/csr/don/2002_02_06/en/index.html, Accessed 9/2/2009.

World Health Organization (2009b). *Tularaemia*. http://www.who.int/csr/delibepidemics/tularaemia/en/, Accessed 9/4/2009.

16. *Mycobacterium* spp.

16.1. HAZARD IDENTIFICATION

16.1.1. Aetiological agent

Mycobacterium spp. cause tuberculosis in animals and humans. In particular *M. tuberculosis* and the closely-related *M. africanum* (Mostowy et al 2004) typically cause tuberculosis in humans. *M. bovis* and the closely-related *M. caprae* (Prodinger et al 2005) cause disease in cattle and a wide variety of other animals and, less commonly, humans. *M. microti* (the vole bacillus) and *M. pinipedii* (marine mammals) are closely-related species but are unlikely to occur in primates. Mycobacteria of the avium-intracellulare complex are widespread in the environment and cause disease in birds and occasional disease in mammals and humans, particularly in immunocompromised individuals (Anonymous 2004). Infection with mycobacteria of the avium-intracellulare complex leads to sensitisation to the tuberculin skin test. *M. avium* subsp. *paratuberculosis* is the cause of Johne's disease in ruminants. In addition, a large number of "atypical" mycobacteria are occasional or opportunistic pathogens. *M. kansasii, M. genavense, M. marinum, M. simiae, M. scrofulaceum, M. szulgai, M. haemophilum, M. intracellulare, M. malmoense, M. ulcerans, M. xenopi, M. abscessus, M. chelonae, M. fortuitum,* and (rarely) *M. smegmatis* have occasionally been implicated as a cause of disease (Scheinfeld 2008). Other species of mycobacteria have been reviewed (Anonymous 2004) and new species are constantly being described. *M. leprae* causes leprosy in both humans and primates (Gibson 1998).

16.1.2. OIE list

M. bovis and *M, avium* subsp. *paratuberculosis* are listed.

16.1.3. New Zealand status

M. tuberculosis, M. bovis, M. avium subsp. *paratuberculosis* and members of the avium-intracellulare complex and many of the opportunistic pathogens are present in this country. *M. bovis* is the subject of a national Pest Management Strategy. It occurs in cattle and farmed deer, and in wild or feral animals including deer, possums, ferrets and pigs. *M. tuberculosis* occurs in humans and is notifiable to the Medical Officer of Health under the Tuberculosis Act of 1948. *M. avium* subsp. *paratuberculosis* is endemic in ruminants.

16.1.4. Epidemiology

Most cases of tuberculosis in primates are caused by *M. tuberculosis* although cases caused by *M. bovis* also occur (Stetter et al 1995; Wilson et al 1984). Members of the *M. tuberculosis* group have closely related biological and immunological characteristics. The lesions they cause and the methods used for diagnosis are similar. *M. microti* is considered not to be pathogenic for humans unless they are immunocompromised, but it has been

identified as cause of disease in squirrel monkeys. For the purposes of this risk analysis the *M. tuberculosis* group is considered as a single pathogenic group

The atypical mycobacteria are not true pathogens and many, if not all, are already present in New Zealand and are, therefore, not considered in this risk analysis.

Johne's disease occurs commonly in ruminants in New Zealand but is not recognised as a disease of humans. Only one reference was found to *M. avium* subsp. *paratuberculosis* causing disease in primates (McClure et al 1987). The case was characterised by chronic diarrhoea, refractory to treatment, that was clinically similar to *M. avium-intracellulare* infections in macaques and to Johne's disease in ruminants.

Some members of the *M. avium-intracellulare* group are pathogenic for birds, and cause localised lesions in lymph nodes of humans, pigs and deer. In humans, they most frequently cause disease in immunocompromised patients, especially those infected with HIV (De Lisle 1987). In primates, a disease resembling Johne's disease in cattle has been ascribed to organisms of the *M. avium-intracellulare* group but attempts to transmit it experimentally were unsuccessful (Gibson 1998).

Rare cases of leprosy have been described in a sooty mangabey and in chimpanzees (Gibson 1998). Signs and lesions are similar to those seen in humans

Tuberculosis caused by *M. tuberculosis* and *M. bovis* has been frequently described in a wide variety of primates (Gibson 1998). It is likely that all primates are susceptible. In the USA, tuberculosis was previously common in primates but improved hygiene and methods of handling and testing imported primates (Roberts and Andrews 2008) has resulted in a great reduction of the number of cases. The prevalence of tuberculosis in 22,913 primates imported into the United States was 0.4% in one study (CDC 1993).

Transmission of mycobacteria is most commonly by the respiratory route but food-borne infection, infection from contact with contaminated fomites, and wound infections are also possible. The deleterious effects of crowding in unhygienic buildings has been demonstrated by the high incidence of cases in wild baboons that slept in an abandoned building (Keet et al 2000). Signs of infection and pathology vary depending on the route of infection and the susceptibility of the animal. In general, the disease resembles that seen in humans, although chronic infections of the type seen in humans are less common in primates, and some primates die without showing any signs. The incubation period may vary from weeks to years. The disease may run an acute course or may be chronic or, in some cases, may be latent, with no overt signs. Latent infections may be reactivated after years, resulting in outbreaks of disease (Lerche et al 2008).

Diagnosis of mycobacterial infections can be made by culturing the organism from suitable specimens or by identification of the organism by PCR. Collection of suitable specimens may be difficult and culturing may take up to 6 weeks or longer, especially in the case of *M. bovis.*

The most common established method of diagnosis in primates is the intradermal tuberculin test (Lerche et al 2008; Roberts and Andrews 2008). Tests based on cell mediated immunity, such as the tuberculin test and the measurement of the gamma

interferon response to specific antigens, are relatively sensitive in recently-infected primates but lose sensitivity over time and frequently fail to detect latently infected cases. Serological tests using a variety of newly identified protein antigens, particularly ESAT 6, are generally more sensitive in detecting latent infections. Although serological tests have not yet been validated stringently, testing regimens involving both cell mediated and serological tests are likely to be most sensitive.

Treatment of infected animals may be undertaken but involves long-term administration of multiple drugs. It is, therefore, unsuitable for prophylactic use when importing primates. Vaccines have seldom been used and are not recommended for use in primates.

16.1.5. Conclusion of hazard identification

M. avium subsp. *paratuberculosis* has only rarely been described in primates. It does not cause disease in humans and is endemic in New Zealand ruminants. Therefore, it is not considered to be a hazard in the commodity.

Organisms of the *M. avium-intracellulare* group occur commonly in New Zealand and are not important pathogens of immunologically competent humans. Therefore, they are not considered to be a hazard in the commodity.

Mycobacterial species termed "atypical mycobacteria" are saprophytes or opportunistic pathogens and are not considered to be a hazard in the commodity.

Leprosy is rare and the signs typical. It is highly improbale that infected primates would not be detected in the type of controlled environment from which the primates considered in this risk analysis would be sourced. Therefore it is not considered to be a hazard in the commodity.

M. tuberculosis occurs worldwide in humans and has been found in zoo and laboratory primate colonies. *M. bovis* occurs commonly in those countries from which it has not been eradicated. Both *M. tuberculosis* and *M. bovis* are present in New Zealand. On the other hand, other members of the *M. tuberculosis* group are not known to be present here. It would be undesirable to introduce new species of the *M. tuberculosis* group. It would also be undesirable to introduce a new source of *M. bovis* (which is the subject of a national Pest Management Strategy in cattle and farmed deer) or *M. tuberculosis* (which is a notifiable disease of humans). For these reasons, all members of the *M. tuberculosis* group are considered to be a potential hazard in the commodity.

16.2. RISK ASSESSMENT

16.2.1. Entry assessment

Primates imported from any of the countries considered in this risk analysis could, possibly, be infected with *M. tuberculosis* . *M. bovis* has been eradicated from Australia, Canada, Singapore and several EU countries but is still reported in France, Germany, Ireland, Italy, Poland, Portugal, Spain, the United Kingdom and, rarely, the USA (OIE 2008). The likelihood of introduction of *M. bovis* is negligible in imports from those

countries from which it has been eradicated and is non-negligible in imports from countries in which it still occurs. Other members of the *M. tuberculosis* group could be introduced from countries where they occur.

Tuberculosis is a rare disease in zoo primate populations that are under proper veterinary supervision. However, the likelihood of introduction of *M. tuberculosis* complex organisms is assessed to be non-negligible.

16.2.2. Exposure assessment

If infected primates were to be imported, it is likely that infection would be transmitted to primates or other animal species in the zoo. *M. tuberculosis* would probably be transmitted only to primates or their handlers. In cages or enclosures that are not enclosed with glass, infection could be transmitted to members of the public by droplet infection. Therefore, the likelihood of exposure is assessed to be non-negligible

16.2.3. Consequence assessment

Introduction of infected animals could result in infections and deaths in primates in the zoo to which the imported animals are introduced. Infections of humans could occur. There is also a slight possibility that introduction of *M. bovis* could result in spread of infection to zoo ruminants. Therefore, the consequences are assessed to be non-negligible.

16.2.4. Risk estimation

The likelihood of entry, exposure and consequence have all been assessed to be non-negligible. The risk estimate for *M. tuberculosis*, *M bovis* and related species, therefore, is non-negligible. All are assessed to be a risk in the commodity and risk management measures can be justified.

16.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of *M. tuberculosis* and *M. bovis* in the commodity;

- Quarantine is not a suitable measure for preventing introduction of mycobacteria as chronic and latent infections occur.
- Treatments and vaccination are not useful.
- All *M. tuberculosis* group species are closely related and infections can be detected using the same tests.
- With regard to tuberculin skin tests, the following recommendation is made in Article 6.12.4. of the *Code*: “Of the skin tests, the Mantoux test is the most reliable of all and has the advantage over others in that the size of the reaction to the test is related to the severity of infection. Skin tests in marmosets, tamarins or small prosimians should be performed in the abdominal skin rather than in the eyelid. In some species (e.g. orangutan), skin tests for tuberculosis are notorious for false

positive results. Comparative tests using both mammalian and avian PPD, together with cultures, radiography and ELISA may eliminate confusion".

The *Code* makes the following recommendations to manage the risk of tuberculosis when importing nonhuman primates;

Article 6.12.5.

Certification and quarantine requirements for marmosets and tamarins from premises under veterinary supervision

Veterinary Authorities of *importing countries* should require:

for marmosets and tamarins from premises under veterinary supervision

1. the presentation of an *international veterinary certificate* attesting that the shipment meets the requirements specified in Article 6.12.3., and that the animals:
    a. are either born in the premises of origin or have been kept there for at least 2 years;
    b. come from premises which are under permanent veterinary supervision, and where a suitable health monitoring programme is followed, including microbiological and parasitological tests as well as necropsies;
    c. have been kept in buildings and enclosures in which no *case* of tuberculosis has occurred during the last 2 years prior to shipment;
2. a description of the health monitoring programme implemented by the establishment of origin;
3. the placement of the animals in a *quarantine station* meeting the standards set in Chapter 5.9. for at least 30 days; and during this period:
    a. all animals to be monitored daily for signs of illness and, if necessary, be subjected to a clinical examination;
    b. all animals dying for any reason to be subjected to complete post-mortem examination at a laboratory approved for this purpose;
    c. […]

Veterinary Authorities of *importing countries* should not normally require any tests for […] tuberculosis. However, stringent precautions to ensure human health and safety should be followed as recommended in Article 5.9.4.

Article 6.12.6.

Certification and quarantine requirements for other nonhuman primates from premises under veterinary supervision

Veterinary Authorities of *importing countries* should require:

for prosimians, New World monkeys, Old World monkeys, gibbons and great apes from premises under veterinary supervision

1. the presentation of an *international veterinary certificate* attesting that the shipment meets the requirements specified in Article 6.9.3., and that the *animals*:

a. are either born in the premises of origin or have been kept there for at least 2 years;

b. come from premises which are under permanent veterinary supervision, and where a suitable health monitoring programme is followed, including microbiological and parasitological tests as well as necropsies;

c. have been kept in buildings and enclosures in which no *case* of tuberculosis has occurred during the last 2 years prior to shipment;

d. come from premises in which no *case* of tuberculosis or other *zoonoses* including rabies has occurred during the last 2 years prior to shipment in the building where the *animals* were kept;

e. were subjected to a tuberculosis test on two occasions with negative results, at an interval of at least 2 weeks between each test during the 30 days prior to shipment;

f. […]

g. […]

h. […]

2. the placement of the *animals* in a *quarantine station* for at least 30 days, and during this period:

a. all *animals* to be monitored daily for signs of illness and, if necessary, subjected to a clinical examination;

b. all *animals* dying for any reason to be subjected to complete post-mortem examination at a laboratory approved for this purpose;

c. any cause of illness or death to be determined before the group to which the *animals* belong is released from quarantine;

d. *animals* to be subjected to the following diagnostic tests and treatments in accordance with Chapter 4.16.:

Disease/agent	**Animal groups**	**Schedule**	**Methods**
Tuberculosis	All species	One test	Skin test or serology. (See further comments in the Table of Article 6.12.4.)
[…]			
[…]			

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

Option 1: Primates to be imported could have been born in or lived for the two years prior to shipment in premises in which tuberculosis has not been diagnosed in any animal for at least 2 years.

Option 2: Primates to be imported could be subjected to a tuberculin skin test on two occasions with negative results; once immediately on entry to a pre-export quarantine and again 4 weeks later.

Option 3: Primates to be imported could be subjected to a tuberculin skin test, and a serological test with negative results; both tests being conducted immediately on entry to a pre-export quarantine and again 4 weeks later.

Note: These options are applicable to all primates and do not differentiate marmosets and tamarins from other primates as the *Code* does in Articles 6.12.5. and 6.12.6.

Note: However, it should be noted that the *Code* explicitly states that Veterinary Authorities of an importing country should not normally require that marmosets or tamarins be subjected to a test for tuberculosis.

Note: Each tuberculin test in a primate requires a general anaesthetic which is stressful and compromises the welfare of the animal. A requirement for unnecessary tuberculin tests should therefore be avoided.

False positive results are relatively common in nonhuman primates, especially orangutans. Such false positives are not a biosecurity issue but will be of concern to an importer. An importer who suspects that a test result is a false positive could apply for a derogation on the grounds of equivalence by supplying additional evidence such as chest X-rays and/or the result of a blood test such as Chembio's *PrimaTB STAT-PAK* test.[5]

References

Some references were seen as abstracts in electronic databases.

Anonymous (2004). Mycobacteria. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 1965-1969.

CDC (1993). Tuberculosis in imported nonhuman primates - United States June 1990-May 1993. *Morbidity and Mortality Weekly Report*, 42(29), 572-6.

De Lisle GW (1987). *Mycobacterium avium* complex. *Surveillance*, 14(4), 20.

Gibson SV (1998). Bacterial and mycotic diseases. X Mycobacterium. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York.. 84-89,

Keet DF, Kriek NPJ, Bengis RG, Grobler DG, Michel A (2000). The rise and fall of tuberculosis in a free-ranging chacma baboon troop in the Kruger National Park. *Onderstepoort Journal of Veterinary Research*, 67(2), 115-22.

Lerche NW, Yee JL, Capuano SV, Flynn JL (2008). New approaches to tuberculosis surveillance in nonhuman primates. *Ilar Journal*, 49(2), 170-8.

[5] www.chembio.com/pdfs/PrimaTB-Sell-Sheet-Final.pdf

McClure HM, Chiodini RJ, Anderson DC, Swenson RB, Thayer WR, Coutu JA (1987). Mycobacterium paratuberculosis infection in a colony of stumptail macaques (*Macaca arctoides*). *Journal of Infectious Diseases*, 155(5), 1011-9.

Mostowy S, Onipede A, Gagneux S, Niemann S, Kremer K, Desmond EP, Kato-Maeda M, Behr M (2004). Genomic analysis distinguishes *Mycobacterium africanum*. *Journal of Clinical Microbiology*, 42(8), 3594-9.

OIE (2008). World Animal Health Information Database (WAHID) Interface. http://www.oie.int/wahid-prod/public.php?page=home, Accessed 27/1/2009.

Prodinger WM, Brandstatter A, Naumann L, Pacciarini M, Kubica T, Boschiroli ML, Aranaz A, Nagy G, Cvetnic Z, Ocepek M, Skrypnyk A, Erler W, Niemann S, Pavlik I, Moser I (2005). Characterization of Mycobacterium caprae isolates from Europe by mycobacterial interspersed repetitive unit genotyping. *Journal of Clinical Microbiology*, 43(10), 4984-92.

Roberts JA, Andrews K (2008). Nonhuman primate quarantine: its evolution and practice. *Ilar Journal*, 49(2), 145-56.

Scheinfeld NS (2008). *Atypical mycobacterial diseases*. http://emedicine.medscape.com/article/1105570-overview, Accessed 9/4/2009.

Stetter MD, Mikota SK, Gutter AF, Monterroso ER, Dalovisio JR, Degraw C, Farley T (1995). Epizootic of Mycobacterium bovis in a zoologic park. *Journal of the American Veterinary Medical Association*, 207(12), 1618-21.

Wilson P, Weavers E, West B, Taylor M, Kavanagh J, Jones P (1984). *Mycobacterium bovis* infection in primates in Dublin Zoo: epidemiological aspects and implications for management. *Laboratory Animals*, 18(4), 383-7.

17. Enteric bacteria

17.1. HAZARD IDENTIFICATION

17.1.1. Aetiological agents

The main enteric bacteria found in primates are;

Escherichia coli (enterotoxigenic, enteropathogenic and verocytotoxin producing strains)
Salmonella spp.
Shigella spp.
Yersinia entrocolitica and *Y. pseudotuberculosis*
Campylobacter coli and *C. jejuni*

17.1.2. OIE list

Not listed.

17.1.3. New Zealand status

All the enteric bacteria listed above are found in New Zealand in humans and animals. In 2007 ESR's Enteric Reference Laboratory reported 96 isolates of *Shigella* spp.(including *S. boydii, S. dysenteriae, S. flexneri* and *S. Sonnei*), 1,341 isolates of at least 68 serotypes of *Salmonella* from humans and 1,001 isolates from at least 43 serotypes from nonhuman sources. There were 120 isolates of verocytotoxic *E. coli*, (ESR 2008). Isolations of *E. coli*, *Y. enterocolitica* and *Y. pseudotuberculosis* are commonly reported in MAF's quarterly magazine, *Surveillance*. *Campylobacter* spp. are isolated very commonly (Baker and Feely 2006; Savill et al 2003).

17.1.4. Epidemiology

Enteric bacteria are universally distributed and there have been many reports of their isolation from primates (Gibson 1998). All species listed above are transmitted by the faecal-oral route and cause gastrointestinal illness characterised by diarrhoea and vomiting. The incubation period of the illness caused by these enteric bacteria is short and infections often run an acute course. However, subclinically infected carriers of all the species listed occur. Diagnosis is always by faecal culture and identification of the organism.

17.1.5. Conclusion of hazard identification

The enteric bacteria occur commonly in New Zealand. The importation of a small number of infected primates into zoos, which are containment facilities, is unlikely to alter the prevalence of human infection in any detectable way. On these grounds it could be argued that these enteric bacteria are not a biosecurity risk in the commodity. However, the *Code*

specifically mentions enteric bacteria and recommends testing of imported primates. Therefore, enteric bacteria are considered to be a hazard in the commodity.

17.2. RISK ASSESSMENT

17.2.1. Entry assessment

Enteric bacteria are universally distributed and are frequently isolated from primates. Therefore, the likelihood that they would be introduced in imported primates is relatively high.

17.2.2. Exposure assessment

Imported primates are intended to be introduced into in the controlled environment of a zoo, which is a containment facility. Nevertheless, the imported primates would come into contact with those already in the collection. Enteric bacteria introduced in imported primates could be transmitted to other primates and their human handlers. Therefore, the likelihood of exposure is assessed to be non-negligible.

17.2.3. Consequence assessment

Introduction of enteric bacteria could result in outbreaks of enteric disease in captive primate populations and sporadic cases of disease in their human handlers.

17.2.4. Risk estimation

Entry, exposure and consequence have all been assessed as non-negligible. The risk estimate for enteric bacteria is therefore non-negligible and they are assessed to be a risk in the commodity. Therefore, risk management measures can be justified.

17.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of enteric bacteria in the commodity;

- Asymptomatic carriage of enteric bacteria is common and a period of quarantine will not eliminate this risk.
- Reliable vaccines are not available to protect against enteric bacteria.
- Administration of antibacterial drugs cannot be relied upon to eliminate carriage of enteric bacteria.

The *Cod*e makes recommendations for managing the risk of enteric bacteria in primates imported from a controlled environment. For nonhuman primates, Articles 6.12.5. and 6.12.6. recommend faecal culture daily for 3 days within the first 5 days of arrival (or into pre-export quarantine).

If the importation were to be from an uncontrolled environment (and hence outside the scope of this risk analysis), the *Code* recommends an additional one or two faecal cultures carried out at intervals of 2 to 4 weeks.

The following options could be considered in order to manage the risk from enteric bacteria;.

Option 1: No restrictions on importation except that the animals should be clinically healthy.

Note: This option implies that, since these enteric bacteria are universally distributed and occur commonly in New Zealand, they are not a significant biosecurity risk in the few primates being imported into a containment facility.

Option 2: Carry out faecal culture for enteric bacteria, as recommended in Articles 6.12.5. and 6.12.6. of the *Code*, with negative results.

Option 3: Carry out faecal culture for enteric bacteria, as recommended in Article 6.12.4. of the *Code*, with negative results.

References

Some references were seen as abstracts in electronic databases.

Baker M, Feely B (2006). Study reveals New Zealand campylobacter rates highest in world http://www.otago.ac.nz/news/news/2006/09-07-06_press_release.html, Accessed 15/4/2009.

ESR (2008). Database of the Enteric Reference Laboratory. http://www.surv.esr.cri.nz/enteric_reference/enteric_reference.php, Accessed 15/4/2009.

Gibson SV (1998). Bacterial and mycotic diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York.. 60-110,

Savill M, Hudson A, Devane M, Garrett N, Glpin B, A. B (2003). Elucidation of potential transmission routes of *Campylobacter* in New Zealand. *Water Science and Technology*, 47(3), 33-8.

18. Internal metazoan parasites

18.1. HAZARD IDENTIFICATION

18.1.1. Aetiological agent

The Australian *Draft Generic Import Risk Analysis of Nonhuman Primates* lists the following parasites;

Trematodes and cestodes

Athesmia foxi (A. heterolecithoides) and *Controrchis biliophilus*
Other Dicrocoeliidae
Phaneropsolus spp.
Primatotrema spp.
Paragonimus spp.
Gastrodiscoides hominis
Aototrema dorsogenitalis
Schistosoma mansoni and *Schistosoma* spp.
Bertiella studeri, Bertiella spp.
Atriotaenia spp. and *Paratriotaenia*
Taenia solium (Cysticercus cellulosae)
Diphyllobothrium spp.
Echinococcus granulosus and *Echinococcus multilocularis*

Nematodes

Probstmayria spp.
Trypanoxyuris tamarini
Subulura spp. and *Primasubulura* spp.
Ternidens deminutus (false hookworm)
Oesophagostomum spp.
Necator spp. and *Ancylostoma* spp.
Nematodirus weinbergi.
Molineus spp. and *Longistriata* spp.
Nochtia nochti
Trichospirura (Spirura) leptostoma and *Spirura* spp.
Streptopharagus spp.
Gongylonema spp.
Pterygodermatites nycticebi, P. alphi
Physaloptera spp. and *Abbreviata spp.*
Oxyuriasis (*Enterobius vermicularis*)
Strongiloidiasis (*Strongiloides* spp.)
Trichuris spp.

Filarial nematodes

Meningonema peruzzii.
Mansonella spp. (affecting Old World apes)
Brugia malayi and *B. pahangi*
Macacanema formosana
Edesonfilaria malayensis
Onchocerca volvulus.
Loa papionis
Dracunculus medinensis (Dracunculus spp.*)*
Filariopsis and *Filaroides* spp.
Angiostrongylus costaricensis.
Anatrichosoma spp.

Acanthocephalans

Monilliformis monilliformis
Prosthenorchis elegans
Prosthenorchis spirula

This list is not exhaustive. One book lists over 200 species of nematodes, more than 70 trematodes and over 40 cestodes that can infect primates (Toft and Eberhard 1998).

18.1.2. OIE list

Echinococcosis/hydatidosis and bovine cysticercosis are listed.

18.1.3. New Zealand status

The status of primates held in zoos and registered facilities is not known. A complete listing of parasites identified in terrestrial mammals in New Zealand, including humans but not including primates, has been compiled (McKenna 2009).

18.1.4. Epidemiology

Since the potential list of parasites is so large, it is neither practical nor useful to consider each parasite individually. In the text below, some species are mentioned but this does not imply that these are the only species that could be found in the commodity. Rather than attempting to deal with each parasite species individually, it is more useful to consider general principles and methods to prevent their introduction.

18.1.4.1. Cestodes and trematodes

Cestodes and trematodes have complex life cycles involving one or two intermediate hosts and sometimes additional paratenic hosts. A wide range of species including snails, reptiles, fish, crustaceans, invertebrates such as copepods, insects and even mammals may act as intermediate hosts for various cestodes and trematodes. Typical examples are *Bilharzia* spp. that require a particular snail species as the intermediate host, *Taenia*

saginata that uses cattle as an intermediate host, while *Paragonimus* spp. require both copepods and crustaceans to complete their life cycle.

For these reasons, the opportunity for successful completion of the life cycle in primates held in the controlled environment of a zoo, is so unlikely that it is negligible. Whether primates are the intermediate host of a parasite, or the accidental host of an intermediate stage of a parasite, they are non-infectious unless eaten by a competent host.

Echinococcosis, listed by the OIE, is an infestation with either *Echinococcus granulosus* or *E. multilocularis* which has been observed in monkeys. However, monkeys are accidental intermediate hosts and hydatid cysts in these species are often sterile. Even if the cysts are not sterile, the life cycle of the parasite could not be completed unless the hydatid cysts were eaten by a competent canid. The likelihood of this happening in a New Zealand zoo is so unlikely that it is assessed to be negligible.

Diagnosis of infestations in the live animal that is a primary host, depends on examination of faeces. Even in species where the parasite resides in the lungs, eggs are coughed up and swallowed and then found in faeces. Faecal examination may require careful gross examination to identify tapeworm segments, or microscopic examination for parasite eggs using sedimentation or flotation methods.

Effective treatments are available for trematodes and cestodes. The most commonly used and broadly effective parasiticide is praziquantel. Therefore the prophylactic use of such parasiticides is a practical risk mitigation measure.

18.1.4.2. Nematodes and acanthocephalans

Nematodes and acanthocephalans are found in the intestines, lungs or other organs. They vary in pathogenicity from significant to harmless. Life cycles are direct or indirect. Species with indirect life cycles include;

Nematodes

Subulura spp.
Primasubulura spp.
Trichospirura (Spirura) leptostoma.
Spirura spp.
Streptopharagus spp.
Gongylonema spp.
Pterygodermatites nycticeb, P. alphi
Physaloptera spp.
Abbreviata spp.
Angiostrongylus costaricensis.

Acanthocephalans

Prosthenorchis elegans
Prostheorchis spirula
Monilliformis monilliformis

Intermediate hosts of these parasites are most commonly cockroaches, but also dung beetles, grasshoppers, other insects, and molluscs for some species. Since the intermediate hosts do not form part of the human diet in New Zealand, the likelihood of transmission of these parasites to humans is negligible. Therefore, although they may be of interest to importers, they are not a biosecurity risk in the commodity.

Filarial parasites of Old and New World monkeys are transmitted by an extensive variety of biting and blood or lymph sucking insects (Toft and Eberhard 1998). Species infesting New World primates include five species of *Dipetalonema* and nine of *Mansonella*. Species infesting Old World primates include three of *Dirofilaria*, *Macacanema formosana*, *Edesonfilaria malayensis*, *Lao papionis*, *Brugia malayensis*, *B. phalangi*, *Meningonema peruzzi*, *Oncocerca volvulus*, four species of *Mansonella* and two of *Dirofilaria* (Toft and Eberhard 1998). Toft and Eberhard conclude "that transmission of these filarial parasites within a primate colony is unlikely and no special control measures are required other than extermination for possible arthropod vectors". Filarial infestations are restricted to tropical countries and, despite numerous introductions of infested, people have not established in temperate countries (Lipner et al 2007). In New Zealand, filarial parasites have never established in the human population. The zoo population of primates in this country is very small There are few species of mosquitoes, no *Culicoides*, and only one tick species that parasitises mammals. Therefore, the likelihood of establishment of filarial parasites subsequent to the importation of a few primates is considered to be negligible.

Zoonotic parasites with direct life cycles include *Stongyloides* spp., oxyurids (pinworms), strongylids (*Oesophagostomum* spp.), ancylostomids (hookworms), trichstrongylids and ascarids (Toft and Eberhard 1998). In addition, there are several species of spirurids for which details of life cycles and possible significance for public health are not known (Toft and Eberhard 1998). However, of these, only *Strongyloides* spp. and hookworms are considered to be of public health significance.

Dracunculus medinensis is a rare parasite that is found in only a few countries in Africa, in areas where people depend on unhygienic water sources in which the intermediate copepod hosts are present (Kelly and Pereira 2006). Animals are not important in maintaining the infestation. The likelihood of introduction of *Dracunculus medinensis* in the few primates likely to be imported, and the likelihood that the parasite could establish here, are negligible.

Anatichosoma spp. are commonly found in the nasal passages of wild *Macaca mulatta* and for effective diagnosis nasal swabs should be examined (Karr et al 1980). The parasite has been recorded in humans but is rare (Toft and Eberhard 1998).

The nematodes of primates have been reviewed by Toft and Eberhard (1998). All species, except *Capillaria hepatica*, are diagnosed by examination of faecal samples to detect eggs, even of parasites of lungs and nasal cavities (although in the latter case examination of faeces is insensitive.). *Capillaria hepatica* is pathogenic for humans. However, because the eggs are released only from the liver only after death, primates in zoos do not constitute a source of infestation for humans.

There are many effective treatments for nematode parasites (Taylor et al 2007; Toft and Eberhard 1998)

18.1.5. Conclusion of hazard identification

Trematode and cestode parasites are not considered to be a hazard in the commodity because they would be unable to complete their life cycle when their primate hosts are housed in a zoo. Therefore, they are not considered to be a hazard in the commodity.

There are many nematode parasites found in primates and, as some are zoonotic or potentially zoonotic, they are considered to be a hazard in the commodity.

18.2. RISK ASSESSMENT

18.2.1. Entry assessment

There are many nematode parasites that are zoonotic or potentially zoonotic. They occur in a wide variety of primates and, collectively, are distributed worldwide. Therefore, when importing primates, the likelihood of their carrying parasites not found in New Zealand is non-negligible

18.2.2. Exposure assessment

Imported primates will be housed with other primates and transmission of nematodes is likely for parasites with direct life cycles. Therefore, parasites are of concern to importers. On the other hand, in the controlled environment of a zoo, only a few staff will have contact with the animals or their faeces. When good hygienic procedures are followed, transmission to zoo staff and further spread to other humans is improbable. However, since transmission to humans or other susceptible mammals could occur under some circumstances, the likelihood of exposure is assessed to be non-negligible.

18.2.3. Consequence assessment

The introduction of novel nematode parasites into zoos primate colonies could have consequences of concern to zoo management. However, many parasite species are non-pathogenic or only mildly pathogenic for humans. Those which are true human pathogens are far more likely to be introduced by hundreds of thousands of humans who enter New Zealand annually. In addition, there are effective treatments for nearly all known internal nematode parasites. However, since some may cause disease in humans and even be transmitted to other animals, the potential consequences are assessed to be non-negligible.

18.2.4. Risk estimation

Entry, exposure and consequence have all been assessed to be non-negligible. As a result, the risk estimate for introducing nematode parasites is assessed to be non-negligible. Therefore, risk management measures can be justified.

18.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of helminth parasites in the commodity;

- Therefore, Since there are a large number of individual parasites to consider, options for effective management should be based on general principles which will be effective to prevent introduction of all species.
- Trematodes and cestodes are not considered to be a hazard in the commodity.
- Nematode infestations can be diagnosed by examination of faecal samples. Faeces should be examined by larval culture and for eggs by flotation and sedimentation methods (Taylor et al 2007).
- Effective treatments are available for all important parasites and could be used prophylactically.

Articles 6.12.5. and 6.12.6. of the *Code* recommend that primates being imported from a controlled environment be subjected to diagnostic tests for, and appropriate treatment against, endoparasites. The test procedure should consist of at least two tests, one of which should be at the start, the other towards the end of a quarantine period. Testing methods should be appropriate to species of primate and species of parasite.

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

Option 1: No measures against internal parasites could be required on the importation of primates from a controlled environment.

Note: This option implies that introduction of internal parasites harmful to humans or other mammals is so unlikely that it can be regarded as negligible.

Option 2: Primates to be imported could be treated twice, once shortly after introduction into a quarantine, and again at least 4 weeks later. Different anthelmintics should be used for the treatments nad both anthelmintics should be known to be broadly effective against nematode parasites.

Note: This option relies solely on treatment without testing to determine whether it has been effective.

Option 3: Primates to be imported could be subjected to a faecal examination using larval culture, sedimentation and flotation methods and any additional appropriate tests (e.g. testing of nasal swabs where there are clinical signs suggestive of *Anatichosoma* spp. infection) immediately on entry into quarantine. A positive test result would be followed by identification of the species of parasite and appropriate treatment of all primates in the consignment. Two weeks after treatment all animals in the group could again be tested and if they are still positive they could be treated with a different anthelmintic. This procedure could be repeated until all animals in the group have negative faecal tests.

References

Some references were seen as abstracts in electronic databases.

Karr SL, Henrickson RV, Else JG (1980). A survey for intestinal helminths in recently wild-caught Macaca mulatta and results of treatment with mebendazole and thiabendazole. *Journal of Medical Primatology*, 9(3), 200-4.

Kelly JK, Pereira G (2006). The problem of water contamination with *Dracunculus medinensis* in southern Sudan. *Journal of Rural and Tropical Public Health.*, 5, 49-58.

Lipner EM, Law MA, Barnett E, Keystone JS, von Sonnenburg F, Loutan L, Prevots DR, Klion AD, Nutman TB (2007). Filariasis in travelers presenting to the GeoSentinel surveillance network. *PLoS Neglected Tropical Diseases*, 1(3), e88.

McKenna, P B (2009) An updated checklist of helminth and protozoan parasites of terrestrial mammals in New Zealand. *New Zealand Journal of Zoology*, 36, 89-113.

Taylor MA, Coop RL, Wall RL (2007). *Veterinary Parasitology*..Blackwell Publishing, Oxford. 904 pages..

Toft JD, Eberhard ML (1998) Parasitic Diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York. Pp. 111-205.

19. Insect parasites

19.1. HAZARD IDENTIFICATION

19.1.1. Aetiological agents

The only flea of importance in primates is *Tunga penetrans* (stick-tight, jigger, or chigoe flea) (Toft and Eberhard 1998). Other fleas sometimes found on primates are incidental infestations by parasites of other animals (e.g. *Ctenocephalides felis* and *Ctenocephalides canis*) and are not considered in this section.

Larvae of certain flies may be responsible for causing dermal myiasis in primates. Examples include *Cuterebra* spp., *Dermatobia hominis, Alouattamyia* spp., *Cordylobia anthropophaga* and *Cordylobia hominivora* (Toft and Eberhard 1998).

At least 12 species of biting lice of the order *Mallophaga* and 22 sucking lice of the order *Anoplura* are known to infest primates (Toft and Eberhard 1998).

19.1.2. OIE list

No arthropod parasites are listed.

19.1.3. New Zealand status

Tunga penetrans is an exotic parasite.

No reports were found of the occurrence in New Zealand of the species causing dermal myiasis in primates.

Pediculus humanus capitis (head louse), *Pediculus humanus humanus* (body louse) and *Phthiris pubis* (pubic louse or crabs) occur universally and are present in New Zealand. No reports were found of the occurrence of the other species of lice in humans and it is concluded that, if they occur, they are rare and not significant.

19.1.4. Epidemiology

Tunga penetrans is a parasite of a wide variety of animals and humans (Taylor et al 2007) and may infest primates (Toft and Eberhard 1998). The flea penetrates into the skin and remains attached to the host with only the posterior part exposed. Male fleas take a blood meal and then leave the host (Witt et al 2004). Females remain attached in the host skin while taking blood and expanding until they may reach the size of a pea. Mobile males mate with the embedded females which shed their eggs through the opening in the skin . The entire life cycle takes about 17 days (Taylor et al 2007). The parasite is found in tropical parts of South and Central America, Asia and Africa (Taylor et al 2007). In

primates *T. penetrans* is a problem only in animals taken from the wild (Toft and Eberhard 1998).

The likelihood of humans becoming infested from animals kept in a controlled environment is remote (Toft and Eberhard 1998).

Treatment of infested quarantine premises and primates with appropriate insecticides is an effective means of controlling *T. penetrans*. Since the primates under consideration in this analysis are to be sourced from a controlled environment, and since Toft and Eberhard (1998) state that *T. penetrans* is a problem only in animals captured from the wild, the likelihood of introducing the parasite is assessed to be negligible.

Myiasis is caused by the larvae of several insect species that parasitise the skin of animals. At least 26 species of *Cuterebra* occur in the USA and Canada. Larvae of *Cuterebra* spp. penetrate the skin of their hosts and develop into subdermal nodules which mature in 3-7 weeks. The larvae leave the host and drop to the ground where they develop into pupae and then adults in the spring. In temperate regions there is a single generation each year, with pupae overwintering in the ground (Taylor et al 2007). *Cutebra* spp.would be unable to complete their life cycle in a controlled environment where buildings are cleaned regularly, disinfected and disinsected. Larvae are sensitive to macrocyclic lactone insecticides (Colwell 2001). Because the primates to be imported are to be sourced from a controlled environment, *Cuterebra* spp. are not considered to be a hazard in the commodity.

Dermatobia hominis parasitises humans and many other species, including primates (Taylor et al 2007; Toft and Eberhard 1998). Larvae that have penetrated the skin may develop for about 3 months before emerging. The adult fly captures a vector host, usually a mosquito, and lays eggs on the mosquito which then carries the eggs to another mammalian host when feeding on it. The distribution of *D. hominis* is restricted to Central America.

The large, and often purulent swellings containing *D. hominis* larvae are easily detected during clinical inspection and emerging larvae would not be able to complete their life cycle in a controlled environment that is cleaned regularly, disinfected and disinsected. Therefore, *D. hominis* is not considered to be a hazard in the commodity.

Allouattamyia spp. are considered to be host-specific for howler monkeys and are rarely found on humans or other primates (Colwell 2001). Since howler monkeys are not included in the scope of this risk analysis, *Allouattamyia* spp. is not a hazard in the commodity.

Cordylobia anthropophaga is another insect whose larvae cause myiasis. These larvae mature and leave the host 7-15 days after infestation. The parasite is confined to tropical areas of Sub-Saharan Africa and this risk analysis considered importation only from controlled environments outside the range of this fly. For this reason, *C. anthropophaga* is not considered to be a hazard in the commodity.

The New World screwworm fly, *Cochliomyia hominivorax*, and the Old World screwworm fly, *Chrysomya bezziana*, are both obligate parasites of mammals during their larval stages. The two species occupy an equivalent parasitic niche in their natural ranges and their life

cycles are essentially the same. Adult females lay eggs on wounds or in body orifices of live mammals. Infestation of a loris (a strepsirrhine primate of the family Lorisidae) with the larvae of *Chrysomya bezziana* has been reported from the Malaysian national zoo (Spradbery & Vanniasingham 1980). However, infestations are easily detected on clinical inspection and would not escape notice in a controlled environment under veterinary supervision. The New World screwworm fly is restricted to tropical countries of South and Central America and the Caribbean while the Old World screwworm fly has a more extensive range, being found throughout much of tropical and subtropical Africa, some countries of the Middle East along the Persian Gulf, the Indian subcontinent, South-East Asia and Papua New Guinea. The degree to which screwworm flies can tolerate cold has had a major influence on their distributions and, for this reason, they are highly unlikely to be able to establish in New Zealand. Neither *Cochliomyia hominivorax* nor *Chrysomya bezziana* is considered to be a hazard in the commodity.

Lice are broadly host specific. However, sucking lice may be able to infest humans, great apes and New World monkeys, with the possible exception of marmosets and tamarins. However, Old World monkeys do not become infested with human sucking lice. Biting lice are rare in primates (Toft and Eberhard 1998). All lice have similar life cycles. Adult lice stick their eggs to hairs on the host's body. The hatched nymphs go through 3 moults before becoming adults, mating and laying eggs. The whole life cycle takes 2-3 weeks (Taylor et al 2007). Lice are typically sensitive to many insecticides but resistance has developed to some of the more frequently used products (Heath 2007). Treatments should be spaced 10-14 days apart to kill all hatched lice before they become adult and lay eggs. Transmission is by direct contact between infested hosts or by contact with objects on which infested hosts have rubbed themselves. Diagnosis of louse infestations is made by careful examination of the primates. It may be necessary to sedate or anaesthetise the animals in order to make a careful examination and a magnifying glass should be used. Some of the louse species carried by primates may not be present in New Zealand.

Human body lice are vectors for epidemic typhus (*Rickettsia prowazekii*) and trench fever (*Bartonella quintanta*) (Azad and Beard 1998; CDC 2007) and, should human body lice be introduced on imported primates, there is a theoretical possibility that they might be carrying these pathogens.

19.1.5. Conclusion of hazard identification

Neither *Tunga penetrans* nor the insect agents of dermal myiasis are considered to be a hazard in the commodity. Since some species of lice that have been found on primates and could infest humans may not be present in New Zealand, lice are considered to be a hazard in the commodity.

19.2. RISK ASSESSMENT

19.2.1. Entry assessment

At least 34 species of lice have been described on primates (Toft and Eberhard 1998) and unless a careful examination of the primates to be imported is carried out, it may not be

obvious that an animal is infested. The likelihood of introducing lice on imported primates is non-negligible.

19.2.2. Exposure assessment

Imported animals would be mixed with other primates in the zoo into which they are introduced and transmission of any lice they may carry would be likely. Transmission to zoo staff would also be possible. Therefore, the likelihood that exposure and transmission to humans will occur is assessed to be non-negligible.

19.2.3. Consequence assessment

The consequences of introducing lice would not be severe. Infested animals may suffer discomfort from skin irritation and there may be loss of hair from rubbing to relieve itching. However, in the unlikely event that introduced lice were to be carrying the agents of epidemic typhus or trench fever those agents could be transmitted to zoo staff should they become infested with the lice. However, this is an improbable scenario, given the sources from which the primates are to be imported, and treatments are available for both lice and the diseases they might be carrying. Nevertheless, since human lice and diseases for which they are vectors could be introduced, the consequences are assessed to be non-negligible.

19.2.4. Risk estimation

The likelihood of entry and exposure, and the consequences have all been assessed to be non-negligible. As a result ,the risk estimate for introduction of new species of lice is non-negligible and they are classified as a risk in the commodity and risk management measures can be justified.

19.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of lice in the commodity;

- New species of lice which are able to parasitise humans are unlikely to be found on primates imported from a controlled environment.
- Effective treatments are available for lice-infested primates and for the diseases they could be carrying.
- Although the *Code* does not make specific recommendations relating to lice, it does make general recommendations for all ectoparasites.
- Effective treatment of lice requires at least two treatments at an interval of 10-14 days.
- The insecticide chosen should be one effective against all ectoparasites. Treatments for all types of ectoparasites should be integrated and reviewed regularly so as to insure the most effective insecticides are used.

- The examination and treatment of primates for ectoparasites may require the administration of a tranquillizer or anaesthetic. This is likely to be stressful and compromise the animal's welfare, so should not be required unless absolutely essential.

The *Code* recommends that nonhuman primates should be tested and treated for ectoparasites at least twice, once at the start of a quarantine period and once toward the end. Testing methods and treatments should be appropriate to the species of animal and parasitic agent.

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

Option 1: Primates could be imported without restrictions provided that their skin and hair appears to be healthy.

Note: This option implies that lice are not likely to be present on primates that are sourced from a controlled environment under veterinary supervision, and that infestations with lice are of minor importance.

Option 2: While held in a pre-export quarantine, primates to be imported could be treated twice, 14 days apart, with a broad spectrum insecticide.

Note: This option is less stringent than the *Code* recommendations since inspections are not required. It does not provide assurance that treatments have been effective.

Option 3: While held in a pre-export quarantine, primates to be imported could be treated twice, 14 days apart, with a broad spectrum insecticide and inspected carefully after each treatment. Should viable lice be present after the second treatment, the procedure could be repeated using a different insecticide and treatment and inspections could be repeated until the animals are parasite-free.

References

Azad AF, Beard CB (1998). Rickettsial pathogens and their arthropod vectors. *Emerging Infectious Diseases*, 4(2), 179-86.

CDC (2007). Prevention of specific Infectious Diseases. Rickettsial infections. In: CDC, (eds.), *Yellow Book/CDC Travellers Health*. CDC. http://wwwn.cdc.gov/travel/yellowBookCh4-Rickettsial.aspx,

Colwell DD (2001). Bot flies and warble flies (Order Diptera: Family Oestridae). In: Samuel WM, Kocan AA, Pybus MJ, (eds.). *Parasitic Diseases of Wild Animals,* Second edition. Wiley Blackwell. 46-71.

Heath ACG (2007). *Sheep Biting-Louse Resistance Management*. http://ww.nzpps.org/resistance/sheeplouse.php

Spradbery JP, Vanniasingham JA (1980). Incidence of the screw-worm fly, *Chrysomya bezziana*, at the Zoo Negara, Malasia. *Malaysian Veterinary Journal* 7: 28-32.

Taylor MA, Coop RL, Wall RL (2007). *Veterinary Parasitology*. Blackwell Publishing, Oxford. 904 pages.

Toft JD, Eberhard ML (1998) Parasitic Diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York. 111-205.

Witt LH, Linardi PM, Meckes O, Schwalfenberg S, Ribeiro RA, Feldmeier H, Heukelbach J (2004). Blood-feeding of *Tunga penetrans* males. *Medical and Veterinary Entomology l*, 18(4), 439-41.

20. Ticks

20.1. HAZARD IDENTIFICATION

20.1.1. Aetiological agent

Ticks belong to the families *Ixodidae* (hard ticks) and *Argasida*e (soft ticks). Worldwide there are around 179 species of *Argasidae* and 690 species of *Ixodidae* (Norval and Horak 2004). It is not known how many of these species could be found on primates but one source lists 37 ixodid and two argasid ticks (Toft and Eberhard 1998).

20.1.2. OIE list

Not listed.

20.1.3. New Zealand status

The only tick parasite of mammals in New Zealand is *Haemaphysalis longicornis*. Five genera of ticks are listed as unwanted notifiable organisms (MAF 2008).

Amblyomma spp.
Boophilus spp.
Dermacentor spp.
Ixodes spp.
Rhipicephalus spp.

20.1.4. Epidemiology

Ticks are important vectors for many diseases of humans; they transmit more diseases than any other vector. These diseases include Lyme's disease, ehrlichiosis, anaplasmosis, relapsing fever, tularemia, tick-borne encephalitis, tick-borne babesiosis and tick-borne rickettsiosis. At least 16 rickettsial diseases are carried by ticks. Ticks are also vectors for many diseases of livestock. At least 33 tick-borne diseases and toxicoses of livestock are present in southern Africa. Ticks may also carry many diseases of cats, dogs and wildlife species. Although there is uncertainty about the susceptibility of primates to tick-borne diseases, they may carry ticks infected with the agents of many human and animal diseases (Allan 2001; Bitam and Raoult 2009; CDC 2007; Norval and Horak 2004).

Hard ticks (*Ixodidae*) have a four stage life cycle; egg, larva with six legs, nymphs with eight legs and no genital pore, and adults with eight legs and a genital pore. Different species of ticks may have one-host, two-host or three-host life cycles. Adults lay batches of several thousand eggs that hatch and the larvae climb up grass stems or other vegetation and await a passing animal host. Larvae are pinhead-sized and not easily seen in grass or on an animal's body. When fully engorged, the larvae moult to develop to the next stage. Three-host ticks leave the host and moult off the host. Two- and one-host ticks moult on the host and then continue to feed on the same host. Mature nymphs of two-host ticks leave

the host when engorged and moult off the host before finding a new host on which to develop to the adult stage. One-host ticks remain on the same host throughout larval, nymph and adult feeding periods. Finally, when the adult females are engorged, they mate with a male while still on the host. Male ticks remain on the host and may mate repeatedly. Three-host ticks, such as *Rhipicephalus appendiculatus*, may remain on the host for only 3 days while one-host ticks, such as *Boophilus microplus*, may be on the host for about 3 weeks (Norval and Horak 2004).

Soft ticks (*Argasidae*) are less important than hard ticks in terms of economic harm caused. Many live off the host in cracks, burrows, nests, or buried in the sand, and take repeated short meals from a resting host. Therefore, soft ticks are unlikely to be imported on live animals.

Acaricides can be used to control ticks. However, many species of tick in several countries have developed resistance to acaricides (Jongejan and Uilenberg 1994; Jonsson et al 2000; Li et al 2003; Li et al 2004; Mekonnen et al 2002)

20.1.5. Conclusion of hazard identification

Many species of tick have been reported on primates. As inspection may fail to reveal pinhead-sized larvae on hairy primates, ticks are considered to be a hazard on the commodity.

20.2. RISK ASSESSMENT

20.2.1. Entry assessment

Many species of tick may infest primates. Since ticks occur in all the countries considered in this risk analysis, the likelihood of introducing ticks on primates is assessed to be non-negligible.

20.2.2. Exposure assessment

Imported primates are likely to be introduced into premises where other primates are present and any ticks they might be carrying could breed in the premises and infest the resident primates and even zoo staff. The likelihood of exposure, therefore, is non-negligible.

20.2.3. Consequence assessment

Infestation of resident primates and zoo staff could lead to ticks being transported to other areas inside or outside the zoo, possibly resulting in the establishment of new tick species and even the occurrence of diseases that the ticks might be carrying. Therefore, the consequences of introducing ticks are assessed to be non-negligible.

20.2.4. Risk estimation

The likelihood of entry and exposure, and consequences have all been assessed to be non-negligible. The risk estimate, therefore, is non-negligible and ticks are classified as a risk in the commodity. Therefore, risk management measures can be justified.

20.3. RISK MANAGEMENT

The following points have been considered in drafting options to manage the risk of introducing ticks on the commodity;

- A large number of tick species are capable of infesting primates.
- Ticks are potential vectors of a number of diseases of humans and animals.
- Resistance to acaricides is common and treatment cannot be relied upon as the only means of preventing the introduction of ticks.
- Treatments for all types of ectoparasites should be integrated and reviewed regularly so as to insure the most effective insecticides are used.
- It may be possible to manage quarantine of primates in a manner that would prevent the introduction of ticks, even without the use of acaricides. If primates were to be held in a quarantine facility for a sufficient length of time, say 6 weeks, any ticks that might be infesting on entry to the premises would have engorged and dropped off. Provided the premises were regularly cleaned and treated in a manner that would kill all ticks, re-infestation would be prevented.
- The examination and treatment of primates for ectoparasites may require the administration of a tranquillizer or anaesthetic. This is likely to be stressful and compromise the animal's welfare, so should not be required unless absolutely essential.

The *Code* recommends that nonhuman primates should be tested and treated for ectoparasites at least twice, once at the start of a quarantine period and once toward the end. Testing methods and treatments should be appropriate to the species of animal and parasitic agent.

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

Option 1: While held in a pre-export quarantine, primates to be imported could be treated twice, 14 days apart, with an effective acaricide.

Note: This option relies solely on the effectiveness of the acaricide.

Option 2: While held in a pre-export quarantine, primates to be imported could be treated twice, 14 days apart, with an effective acaricide and inspected carefully after each treatment. Should viable ticks be present after the second treatment, the procedure could be repeated using a different acaricide and treatment and inspections could be repeated until the animals are parasite-free.

Note: Whichever option is selected, effective management of the risk requires that the quarantine premises have impervious floors and smooth painted walls.

The premises should be cleaned thoroughly with a high pressure hose followed by steam cleaning or spraying with a suitable acaricide. Bedding should consist of material that will not harbour ticks. Grass and straw are not suitable while wood shavings or sawdust are. Bedding should be removed every 7 days and the premises cleaned thoroughly and sprayed with acaricide.

References

Some references were seen as abstracts in electronic databases.

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

Bitam I, Raoult D (2009). Other tick-borne diseases in Europe. *Current Problems in Dermatology*, 37, 130-54.

CDC (2007). Rickettsial (Spotted and Typhus Fevers) and Related Infections (Anaplasmosis and Ehrlichiosis). In: CDC, (eds.), *Yellow Book/CDC Travellers Health*. http://wwwnc.cdc.gov/travel/yellowbook/2010/chapter-5/rickettsial-and-related-infections.aspx. Accessed 29/04/2010.

Jongejan E, Uilenberg G (1994). Ticks and tick control methods. *Revue Scientifique et Technique.* 13(4), 1201-20.

Jonsson NN, Mayer DG, Green PE (2000). Possible risk factors on Queensland dairy farms for acaricide resistance in cattle tick (Boophilus microplus). *Veterinary Parasitology*, 88(1-2), 79-92.

Li AY, Davey RB, Miller RJ, George JE (2003). Resistance to coumaphos and diazinon in *Boophilus microplus* (Acari: Ixodidae) and evidence for the involvement of an oxidative detoxification mechanism. *Journal of Medical Entomology*, 40(4), 482-90.

Li AY, Davey RB, Miller RJ, George JE (2004). Detection and characterization of amitraz resistance in the southern cattle tick, *Boophilus microplus* (Acari: Ixodidae). *Journal of Medical Entomology*, 41(2), 193-200.

MAF (2008). Unwanted Organisms Register. http://mafuwsp6.maf.govt.nz/uor/searchframe.htm, accessed 11/1/2009.

Mekonnen S, Bryson NR, Fourie LJ, Peter RJ, Spickett AM, Taylor RJ, Strydom T, Horak IG (2002). Acaricide resistance profiles of single- and multi-host ticks from communal and commercial farming areas in the Eastern Cape and North-West Provinces of South Africa. *Onderstepoort Journal of Veterinary Research*, 69(2), 99-105.

Norval RAI, Horak IG (2004). Vectors: Ticks. In: Coetzer JAW, Tustin RC, (eds.). *Infectious Diseases of Livestock.* 2nd edition. Oxford University Press, Capetown: 3-42.

Toft JD, Eberhard ML (1998) Parasitic Diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York. 111-205.

21. Mites

21.1. HAZARD IDENTIFICATION

21.1.1. Aetiological agent

Toft and Eberhard (1998) list 18 species of respiratory mite and 36 of skin mite that have been recorded in primates. Of these, only *Sarcoptes scabiei*, trombiculid mites and *Demodex* spp. are known to parasitise humans.

The *Trombiculidae* contain approximately 700 species (Centre for Food Security and Public Health and the Institute for International Cooperation in Animal Biologics 2005)

21.1.2. OIE list

Not listed.

21.1.3. New Zealand status

Nothing is known about the occurrence of mites in primates already in zoos in New Zealand.

According to a MAF Overseas Market Access Requirement Notification, *Sarcoptes scabiei* infests "sheep and goats in New Zealand (May 2005). However, it has been reported in dogs, ferrets, pigs, lamoids, hedgehogs, and humans" (MAF 2006). Thirteen species of demodicid mites have been recorded from 10 mammalian host species, including humans (Nutting et al 1975). Trombiculid mites are present (Spain and Luxton 1971).

21.1.4. Epidemiology

Cutaneous acariasis in primates can be caused by a large variety of mites. However, only *Sarcoptes scabiei* and *Demodex* spp.are of significance in humans.

Sarcoptes scabiei, the cause of sarcoptic mange, may be transmitted to humans. This mite causes hyperkeratosis and crusting of the skin accompanied by intense itching. Although the sarcoptic mange mites found on different species are morphologically similar, they are regarded as different varieties, subspecies, or forms (Cordoro et al 2008), and have a high degree of species specificity. Humans infested with mites from other species develop mild symptoms which resolve spontaneously (Cordoro et al 2008). It is not known whether primates are infested with true human strains or with varieties adapted to their primate hosts. However, transfer from primates to humans, and vice versa, has been recorded on several occasions. Sarcoptic mange can be diagnosed by recognition of typical signs and confirmed by demonstration of mites in skin scrapings. Several effective treatments are available, including injection of ivermectin and application of topical insecticides. The life cycle on the host is completed in about 3 weeks (Zajac and Conboy 2006). Treatments

need to be repeated to kill newly-hatched larvae before they can develop into adults. Treatments with ivermectin at 2 week intervals, or bathing in insecticidal solutions at weekly intervals for 4 weeks, are effective treatments.

Demodex folliculorum and *D. brevis* occur commonly in humans in hair follicles and, particularly, in eyelash follicles (Baima and Sticherling 2002; Hu and Wang 2001). There is some dispute as to whether they are harmless commensals or pathogens. However, they have frequently been associated with blepharitis (Czepita et al 2005a; Czepita et al 2005b). These mites are universally distributed and occur in New Zealand (Nutting et al 1975). The two species specific to humans are not included in the list of parasites known to occur in primates, but *Demodex* spp. are listed as parasites of primates (Toft and Eberhard 1998).

No references were found to human infestations with other skin mites of primates.

Respiratory and nasal mites may infest a wide variety of primates including Old and New World monkeys, baboons and great apes (Toft and Eberhard 1998). Lung mite infestations usually cause minimal disease signs or are subclinical. However, in some cases lung lesions resembling tubercles may be observed. *Pneumonyssus semicola* is found in virtually 100% of wild *Macacca mulatta*. Although mites may be demonstrated in tracheobronchial washings, the method is not reliable and diagnosis is difficult. Complete control can be achieved by raising newborn monkeys in isolation from their mothers, and mite-free colonies can be established by this means. None of the respiratory mites are known to infest humans. Similarly, the five known species of nasal mite and one laryngeal mite of primates have not been reported as being able to infest humans.

Trombiculid mites (known as harvest mites, chiggers) are universally distributed and the family *Trombiculidae* includes around 700 species. They are extremely small, almost microscopic, being about 0.4 mm long. They are parasitic only in their larval stage, with other stages living in the environment. They act as vectors for *Orientia* (*Rickettsia*) *tsutsugamashi* the agent of scrub typhus (Azad and Beard 1998).

Scrub typhus is endemic to a part of the world known as the ‘tsutsugamushi triangle, which extends from northern Japan and far-eastern Russia in the north, to northern Australia in the south, and to Pakistan and Afghanistan in the west (Seong, Choi and Kim 2001).

Effective insecticides are available for the treatment of all species of mite. Treatment should be repeated at suitable intervals to kill all newly-hatched larvae before they reach maturity. Treatments at 2 week intervals are appropriate.

21.1.5. Hazard identification conclusion

Except for *Sarcoptes scabiei* and trombiculid mites, the mites of primates are not known to infest humans. *Sarcoptes scabiei* may be found on many species in New Zealand and so is not a hazard on the commodity. Trombiculid mites could carry *Orientia tsutsugmashi*, a pathogen not present in New Zealand and so are considered to be a hazard on the commodity.

Demodex folliculorum and *D. brevis*, the two *Demodex* parasites of humans, are not listed as primate parasites (Toft and Eberhard 1998). In addition, *Demodex* infestations of

humans occur universally and usually do not cause disease. Therefore, *Demodex* spp. are not considered to be a hazard on the commodity.

None of the other species of mite which infest skin or the respiratory system are parasites of humans. Therefore, although they may be important for importers, they are not considered to be a hazard in the commodity.

21.2. RISK ASSESSMENT

21.2.1. Entry assessment

Since trombiculid mites occur universally and can be carried by primates, they could be introduced into a zoo on imported primates. However, trombiculid mites remain on their hosts for a few days only before dropping off. It then takes 50-70 days for them to complete their life cycle (Taylor et al 2007). If primates were to be held in quarantine for a period before or after importation, any trombiculid mites they might be carrying would be unable to re-infest them before they were removed from quarantine. Therefore, the likelihood of entry of trombiculid mites is assessed to be extremely low.

21.2.2. Exposure assessment

The likelihood that trombiculid mites being introduced on imported primates is extremely low. However, if they were introduced into a premised in New Zealand, they could complete their life cycle and infest other primates, or zoo staff. The likelihood of exposure is, therefore, assessed as non-negligible.

21.2.3. Consequence assessment

Trombiculid mites are already present in New Zealand and so the consequences of introducing the mites themselves would be negligible. However, should the introduced mites be carrying *Orientia tsutsugamashi*, primates or their handlers could become infected. Scrub typhus can be fatal if untreated. Although it is highly unlikely that *O. tsutsugamashi* would be carried by trombiculid mites on primates imported from a controlled environment in those countries considered in this risk analysis, the consequences of introduction could be significant.

21.2.4. Risk estimation

The likelihood of entry is assessed as being extremely low for tromiculid mites. The likelihood of exposure is assessed as being non-negligible but possible consequences significant. The risk estimate is, therefore, non-negligible and trombiculid mites are assessed to be a risk on the commodity. risk management measures can be justified.

21.3. RISK MANAGEMENT

The following points have been considered in drafting options to manage the risk of introducing trombiculid mites on the commodity;

- Trombiculid mites are extremely small (0.4 mm) and are unlikely to be seen on inspection.
- Trombiculid mites are a potential vector of scrub typhus.
- Trombiculid mites are present on their host for a few days only.

The *Code* recommends that nonhuman primates should be tested and treated for ectoparasites at least twice, once at the start of a quarantine period and once toward the end. Testing methods and treatments should be appropriate to the species of animal and parasitic agent.

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

Option 1: While held in a pre-export quarantine, primates to be imported could be treated twice, 14 days apart, with an effective ectoparasiticide.

Note: Effective management of the risk requires that the quarantine premises have impervious floors and smooth painted walls. The premises should be cleaned thoroughly with a high pressure hose followed by steam cleaning or spraying with a suitable insecticide. Bedding should consist of wood shavings or sawdust. Bedding should be removed every 7 days and the premises cleaned thoroughly and sprayed with insecticide.

References

Some references were seen as abstracts in electronic databases.

Azad AF, Beard CB (1998). Rickettsial pathogens and their arthropod vectors. *Emerging Infectious Diseases*, 4(2), 179-86.

Baima B, Sticherling M (2002). Demodicidosis revisited. *Acta Dermato-Venereologica*, 82(1), 3-6.

Centre for Food Security and Public Health and the Institute for International Cooperation in Animal Biologics (2005). *Acariaisis. Mange*. http://www.cfsph.iastate.edu/Factsheets/pdfs/acariasis.pdf.

Seong S, Choi M, Kim I (2001). *Orientia tsutsugamushi* infection: overview and immune responses. *Microbes and Infection* 3 (1): 11–21.

Cordoro KM, Wilson BB, Cawley EP, Kauffman CD (2008). *Scabies.* http://emedicine.medscape.com/article/1109204-overview, Accessed 30/4/2009.

Czepita D, Kuzna-Grygiel W, Kosik-Bogacka D (2005a). [Demodex as an etiological factor in chronic blepharitis]. *Klinika Oczna*, 107 (10-12), 722-4.

Czepita D, Kuzna-Grygiel W, Kosik-Bogacka D (2005b). [Investigations on the occurrence as well as the role of Demodex follicuforum and Demodex brevis in the pathogensis of blepharitis]. *Klinika Oczna*, 107 (1-3), 80-2.

Hu Q, Wang Y (2001). [Investigation on the prevalence of human Demodex among 2,248 medical students in inner Mongolia]. *Zhongguo Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi*, 19 (4), 239-40.

MAF (2006). *Exporting Sheep and Goats to Argentina*. Overseas Market Access Requirements Notification. http://www.biosecurity.govt.nz/exports/animals/omars/saganiec.arg.htm, Accessed 20/4/2009.

Nutting WB, Kettle PR, Tenquist JD, Whitten LK (1975). Hair follicle mites (*Demodex* spp.) in New Zealand. *New Zealand Journal of Zoology*,, 2(2), 219-22.

Spain AV, Luxton M (1971). Catalog and bibliography of Acari of the new Zealand subregion. *Pacific Insects Monograph* (25), 179-226.

Taylor MA, Coop RL, Wall RL (2007). Mites. In: *Veterinary Parasitology*. Blackwell Publishing, Oxford. 712-5.

Toft JD, Eberhard ML (1998) Parasitic Diseases. In: Taylor Bennett B, Abee CR, Henrickson R (eds.). *Nonhuman Primates in Biomedical Research: Diseases.* Academic Press, New York. Pp. 111-205.

Zajac AM, Conboy GA (2006). Sarcoptes scabiei. In: (eds.), *Veterinary Clinical Parasitology*, 7th edition. Blackwell Publishing, Ames, Iowa. 50.

22. Weed seeds

22.1. HAZARD IDENTIFICATION

22.1.1. Aetiological agent

All viable seeds of weeds and exotic plants.

22.1.2. OIE list

Not listed.

22.1.3. New Zealand status

All exotic plants are unwanted.

22.1.4. General considerations

Weed seeds could be found attached to the hair of primates. Large seed heads and pieces of plant material would be easily visible and could be removed before shipment but small seeds might not be visible. It is unlikely that seeds or plant material would remain attached to a primate's hair or skin for more than a short time since most primates actively groom each other and themselves.

Seeds are specifically adapted to survive unfavourable environmental conditions and most will at least survive from one growing season to another. Many will survive for several years and germinate when favourable conditions occur. Most seeds are highly resistant to dehydration and retain viability better in dry conditions although some are specifically adapted to remain viable in water.

Weed seeds may survive passage through an animal’s digestive system and be passed out in faeces (Katovich et al undated). One hundred percent of radioopaque markers were voided from the gut of 25 human subjects in 25-169 hours (Hinton et al 1969). It is highly probable that weeds seeds ingested by primates would be voided in a similar period during a relatively short quarantine period.

22.1.5. Hazard identification conclusion

It is concluded that weed seed could be introduced on a primate’s hair or in its digestive tract and weed seeds are considered to be a hazard in the commodity.

22.2. RISK ASSESSMENT

22.2.1. Entry assessment

As seeds could be introduced attached to a primate's hair or in its faeces, the likelihood of entry is non-negligible

22.2.2. Exposure assessment

Weed seeds could become detached from primate's hair or passed in faeces. They are generally resistant to most environmental conditions and may remain dormant until conditions are favourable for germination. Therefore, the likelihood that seeds could germinate and grow if released into a suitable environment is non-negligible.

22.2.3. Consequence assessment

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

22.2.4. Risk estimation

Since the likelihood of entry and exposure, and consequences are assessed to be non-negligible, the risk estimate for weed seeds is non-negligible and they are classified as a risk in the commodity. Therefore, risk management measures can be justified.

22.3. RISK MANAGEMENT

The following points have been considered when drafting options to manage the risks associated with introduction of weed seeds in the commodity:

- Weed seeds will not be present on a primate's hair or in their faeces unless weeds or weed seeds have been present in the premises where primates are held prior to export or in food eaten by the primates prior to export.
- Weed seeds are likely to be able to survive harsh environmental conditions.
- The examination of a primate's skin for the presence of weed seeds may require the administration of a tranquillizer or anaesthetic. This is likely to be stressful and compromise the animal's welfare, so should not be required unless absolutely essential.
- Examination of a primate's skin for ectoparasites would detect weed seeds.

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

Option 1: Primates could be imported without restrictions on the assumption that they are unlikely to be carrying plant material or seed on their hair or skin or in their alimentary tract.

Option 2: Prior to export, primates could be fed a diet that is free from viable seeds. A normal primate diet is likely to be suitable.

Options 3: Premises where primates are held prior to export could be free from all bedding material that could contain weeds or weed seeds. Wood shavings, sawdust or artificial bedding materials would be suitable.

Option 4: Immediately prior to shipment primates could be inspected to ensure that they are free from plant material. If necessary they could be groomed thoroughly.

References

Hinton JM, Lennard-Jones JE, Young AC (1969). A new method for studying gut transit times using radioopaque markers. *Gut*, 10(10), 842-7.

Katovich J, Becker R, Doll J (undated). *Weed Seed Survival in Livestock Systems*. A publication of University of Minnesota extension service http://www.manure.umn.edu/assets/WeedSeedSurvival.pdf, Accessed 17/7/2009.