Australian Government

Biosecurity Australia

Import Risk Analysis Report for Horses from Approved Countries

Final Report

March 2010

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Acronyms and abbreviations

AGID	agar gel immunodiffusion
AHIC	Australian Horse Industry Council
AHS	African horse sickness
ALOP	appropriate level of protection
AQIS	Australian Quarantine and Inspection Service
ARB	Australian Racing Board
AUSVETPLAN	Australian Veterinary Emergency Plan
BDV	Borna disease virus
CEM	contagious equine metritis
CFT	complement fixation test
C-ELISA	competitive ELISA
Code	OIE Terrestrial Animal Health Code 2009
EADRA	Emergency Animal Disease Response Agreement
EAV	equine arteritis virus
EEE	Eastern equine encephalitis
EEV	equine encephalosis virus
EHV	equid herpesvirus
EGA	equine granulocytic anaplasmosis
EI(V)	equine influenza (virus)
EIA	equine infectious anaemia
ELISA	enzyme-linked immunosorbent assay
EPM	equine protozoal myeloencephalitits
EVA	equine viral arteritis
FEI	Fédération Equestre Internationale (International Equestrian Federation)
HI	haemagglutination inhibition
IFAT	indirect fluorescent antibody test
IgG	immunoglobulin G
IgM	immunoglobulin M

IRA	import risk analysis
JE	Japanese encephalitis
LP-ELISA	liquid-phase blocking ELISA
OIE	World Organisation for Animal Health (formerly known as the Office International des Epizooties)
OIE Manual	OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009
PAQ	post-arrival quarantine
PCR	polymerase chain reaction
PEQ	pre-export quarantine
RVF(V)	Rift Valley fever (virus)
RT-PCR	reverse-transcriptase PCR
SOPs	Standard operating procedures
SPS	sanitary and phytosanitary
SPS Agreement	WTO Agreement on the Application of Sanitary and Phytosanitary Measures.
SRH	single radial haemolysis
USDA	United States Department of Agriculture
VEE	Venezuelan equine encephalitis
VN	virus neutralisation
VS	vesicular stomatitis
WEE	Western equine encephalitis
WNV	West Nile virus
WTO	World Trade Organization

Summary

Horses have been imported into Australia since European settlement. Importation by air began in the 1970s and was routine by the mid-1990s. Risks associated with shorter travel times were taken into account in developing subsequent quarantine measures.

Following the outbreak of equine influenza in Australia in August 2007, Biosecurity Australia recommended to the Australian Quarantine and Inspection Service (AQIS) that strengthened quarantine measures be implemented on an interim basis for the temporary and permanent importation of horses from all approved countries except New Zealand. Additional quarantine measures announced in September 2007 included testing requirements for equine influenza, strengthened operational standards for quarantine facilities during pre-export quarantine (PEQ) and post-arrival quarantine (PAQ), and an extended PAQ period for consignments originating from multiple PEQ facilities. Further amendments to the interim measures were announced in December 2007, July 2008 and September 2008.

On 12 June 2008, the Australian Government announced that it had accepted all 38 recommendations from the Commission of Inquiry into the August 2007 equine influenza outbreak in Australia. The Government's response to recommendation 34 stated that Biosecurity Australia would conduct an import risk analysis (IRA) for horses from countries and regions from which Australia currently permits such importation.

The Chief Executive of Biosecurity Australia announced the formal commencement of the IRA on 30 September 2008 to be conducted according to the IRA Handbook 2007 using a non-regulated pathway. The draft IRA report was released for 60 days public comment on 30 November 2009.

The IRA report for horses from approved countries was developed by Biosecurity Australia with the assistance of technical and scientific experts, including an Expert Panel. This report provides an assessment of the risks of introduction and spread of potential disease agents associated with the importation of horses from approved countries and, where appropriate, recommends risk management measures in accordance with Australia's conservative approach to quarantine.

Countries, administrative regions and territories from which Australia currently permits the importation of horses, are referred to in the IRA as approved countries. These are Canada, certain Member States of the European Union (Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Luxembourg, the Netherlands, Portugal, Spain, Sweden and the United Kingdom), Hong Kong, Macau, New Zealand, Singapore, Switzerland, the United Arab Emirates, and the United States.

Australia suspended the direct importation of horses from Japan following the outbreak of equine influenza in Japan in August 2007. Revised quarantine measures for the importation of horses from Japan are considered as part of the generic measures recommended in this IRA. Thus for the purposes of this IRA, Japan is considered an approved country.

This final IRA report recommends quarantine measures necessary to achieve Australia's appropriate level of protection (ALOP) for the importation of horses, donkeys and mules from approved countries. Biosecurity Australia has made a number of changes to the IRA report following consideration of stakeholder comments on the draft IRA report. These changes include:

- the duration of PAQ has been amended to include a 14-day PAQ for commingled consignments originating from the same region. After further consideration of risks of commingling and effects of prior exposure and heterogeneous strains of EI virus, the Expert Panel considered that the risks could be managed with a 14-day PAQ for commingled consignments originating from the same region
- certification for contagious equine metritis (CEM) has been amended to allow samples to be taken during the 30 days immediately before export, including during PEQ
- timing of sampling for diagnostic tests has been aligned, where possible, in order to provide less restrictive options without compromising the quarantine risk. This will improve management of test results before export, facilitate certification and minimise veterinary attendance. This amendment also applies to the timing for taking reference sera
- editorial corrections and clarification have been incorporated, and additional supporting technical information and new references included.

The hazard identification chapter compiled a list of potential disease agents and 43 were retained for risk assessment. Following risk assessment, the unrestricted risk of entry, establishment and/or spread was estimated for each disease agent. For 22 disease agents, the unrestricted risk was assessed as being too high to achieve Australia's ALOP, and risk management measures were considered and recommended to reduce the risk to an acceptable level. In addition, for two diseases — Borna disease and West Nile fever — that are emerging diseases of potential quarantine concern, measures are recommended to manage the health of the animal while in quarantine and minimise delays in quarantine and subsequent consignments if there is a clinical case and a disease investigation is necessary. The IRA recommends premises freedom for Borna disease and vaccination against West Nile virus for horses from countries where clinical disease is known to occur.

It is the view of the Expert Panel that risk management measures, in accordance with the World Organisation for Animal Health (OIE) Terrestrial Animal Health Code 2009 (the Code) recommendations and taking account of the equine health status of the approved countries, are appropriate for the majority of disease agents requiring risk management. These are included in Australia's recommended quarantine measures for:

- African horse sickness
- anthrax
- contagious equine metritis
- dourine
- Eastern equine encephalomyelitis
- epizootic lymphangitis
- equid herpesvirus-1 (abortigenic and neurological strains)
- equine infectious anaemia
- equine viral arteritis

- glanders
- horse pox
- Japanese encephalitis
- New World screw-worm fly
- Old World screw-worm fly
- rabies
- Western equine encephalomyelitis
- Venezuelan equine encephalomyelitis.

Where there were no recommendations in the Code, Australia's existing quarantine measures differ from the Code or the recommendations in the Code did not achieve Australia's ALOP for a disease agent, risk management options were considered. These disease agents were equine influenza, equine piroplasmosis, Lyme disease, surra and vesicular stomatitis. It is the Expert Panel's view that the risk management measures summarised below would achieve Australia's ALOP.

Equine influenza

Country freedom without vaccination, or premises freedom, vaccination, diagnostic testing, PEQ and PAQ.

Equine piroplasmosis

Country freedom, or premises freedom, inspection and treatment for ticks, diagnostic testing, PEQ and PAQ.

Lyme disease

Country freedom, or premises freedom, inspection and treatment for ticks, PEQ and PAQ.

Surra

Country freedom, or no disease in equids, premises freedom, protection from vectors, diagnostic testing, PEQ and PAQ.

Vesicular stomatitis

Country freedom, or premises freedom, diagnostic testing and PEQ.

Two disease agents that did not previously require quarantine measures for the importation of horses into Australia are anthrax and screwworm-fly myiasis.

For all other disease agents retained for risk assessment, the unrestricted risk was estimated as being sufficiently low to achieve Australia's ALOP. This included three disease agents previously requiring quarantine measures, namely, equine granulocytic anaplasmosis (*Anaplasma phagocytophilum,* formerly *Ehrlichia equi*) and Potomac horse fever (*Neorickettsia risticii* formerly *Ehrlichia risticii*), both formerly referred to as equine ehrlichiosis, and *Taylorella asinigenitalis*.

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

1 Introduction

The import risk analysis (IRA) report for the importation of horses from approved countries was developed by Biosecurity Australia with the assistance of the horse IRA Expert Panel. The IRA assesses the risks of introduction and spread of potential disease agents associated with the importation of horses and, where appropriate, recommends risk management measures in accordance with Australia's conservative approach to quarantine.

1.1 Background

Horses have been imported into Australia since European settlement. Importation by air began in the 1970s and became routine from the mid-1990s. The associated risks with shorter travel times were taken into account in developing quarantine measures.

Australia has a range of conditions covering the temporary and permanent importation of horses and the return of Australian horses. Horses have been permitted importation from Canada, certain Member States of the European Union, Hong Kong, Japan, Macau, New Zealand, Singapore, Switzerland, United Arab Emirates and the United States.

In August 2007, there was an outbreak of equine influenza in Australia that spread in New South Wales and Queensland. The Australian Government established a Commission of Inquiry into the outbreak headed by the Hon Ian Callinan AC. Through cooperative efforts of government, industry and the general public, equine influenza was eradicated. Australia met the World Organisation for Animal Health (OIE) criteria as free from equine influenza on 25 December 2008.

In response to the quarantine risk arising from the equine influenza outbreak, on 28 September 2007, Biosecurity Australia informed stakeholders (Biosecurity Australia Advice (BAA) 2007/21) that it had recommended to the Australian Quarantine and Inspection Service (AQIS) that strengthened quarantine measures be implemented on an interim basis for the temporary and permanent importation of horses from all approved countries except New Zealand. Additional quarantine measures included vaccination and testing requirements for equine influenza, the operation of quarantine facilities during pre-export quarantine (PEQ) and post-arrival quarantine (PAQ) and an extended PAQ period.

Further amendments were advised on 6 December 2007 (BAA 2007/23) to clarify the pre-export equine influenza vaccination requirements and included an additional test for equine influenza within 24 hours of arrival in Australia. The amendments were included in interim measures for the importation of horses from all approved countries except New Zealand.

Biosecurity Australia announced on 14 July 2008 updated interim conditions for importation of horses from the United States and European Union (BAA 2008/22). In September 2008 (BAA 2008/31), further advice was provided to AQIS to amend interim quarantine conditions for imported horses from other approved countries except New Zealand. Amendments included specifying equine influenza strains in vaccines if such vaccines are commercially available, collection of blood samples

during PEQ, additional PAQ equine influenza testing requirements and changes to the operational arrangements during PEQ and PAQ.

On 12 June 2008, the Australian Government announced that it had accepted all 38 recommendations from the Commission of Inquiry into the equine influenza outbreak in Australia. The Government's response to recommendation 34 stated that:

> 'Biosecurity Australia will undertake, in accordance with the Import Risk Analysis Handbook 2007, a comprehensive import risk analysis relating to the importation of horses from the countries and regions from which Australia currently permits such importation. Biosecurity Australia will make such recommendations for any changes to policies for importation as are warranted to the Director of Animal and Plant Quarantine (copies will also be provided to the officer responsible for the importation of horses and the Executive Director of AQIS).'

Countries from which Australia currently permits the importation of horses include Canada, the European Union, Hong Kong, Macau, New Zealand, Singapore, Switzerland, United Arab Emirates and the United States. Import conditions for horses from Japan were suspended following the equine influenza outbreak in Japan in 2007; however, Japan has been included in the scope of the IRA report.

There were conditions for the importation of horses from Fiji, New Caledonia, Norfolk Island and Norway but they have not been used for a considerable time. Although not specified in the IRA, any applications to import horses from these countries will be based on the generic quarantine measures recommended in the IRA.

The Chief Executive of Biosecurity Australia announced the formal commencement of the IRA on 30 September 2008 (BAA 2008/32). The IRA report was developed according to the 2007 IRA Handbook using a non-regulated pathway. This approach includes the release of an IRA report for 60 days public comment.

1.2 Import risk analysis process

Australia's biosecurity policies and risk management measures aim to prevent or control the entry, establishment and/or spread of pests and diseases that could cause significant harm to people, animals, plants and other aspects of Australia's environment.

IRAs are based on the latest scientific information and are undertaken by Biosecurity Australia with the assistance of technical and scientific experts, where necessary, and in consultation with stakeholders at various stages during the process. An Expert Panel has assisted in the development of this IRA report. The membership of the Expert Panel is detailed in section 1.3. Stakeholder comments are encouraged and comments will be carefully considered in finalising the IRA. The final IRA and recommendations for a policy determination are provided to Australia's Director of Animal and Plant Quarantine (the Secretary of the Australian Government Department of Agriculture, Fisheries and Forestry). Once a policy determination is made, AQIS will take this into account, together with other relevant information, in considering applications for import permits for horses. AQIS is responsible for implementing risk management measures, pre-border, border and post-border.

The IRA process is an important part of developing and reviewing Australia's biosecurity policies. It enables the Australian Government to consider formally the risks that could be associated with current imports or proposals to import new products into Australia. If a risk does not achieve Australia's appropriate level of protection (ALOP), risk management measures are recommended.

1.2.1 Australia's appropriate level of protection

Australia is a member of the World Trade Organization (WTO) and must adhere to the WTO Agreement on the Application of Sanitary and Phytosanitary Measures (SPS Agreement). A science-based process is required for assessing quarantine risks. The SPS Agreement defines the concept of an 'appropriate level of sanitary and phytosanitary protection' as the level of protection deemed appropriate by a WTO Member establishing a sanitary or phytosanitary measure to protect human, animal or plant life or health within its territory. Among a number of obligations, a WTO Member, when applying risk management measures, should ensure that these are not more trade restrictive than required to achieve its ALOP.

Successive Australian governments have maintained a conservative approach to the management of biosecurity risks. This approach is expressed in terms of Australia's ALOP, which reflects community expectations through Government policy. Australia's ALOP is currently described as providing a high level of protection aimed at reducing risk to a very low level, but not to zero. This definition of the ALOP is illustrated in a risk estimation matrix, in Table 3.3 of the Methods chapter (chapter 3). Australian State and Territory governments have indicated their support through the Primary Industries Ministerial Council, which agreed in 2002 that Australia's needs are met by this definition of the ALOP.

1.3 Expert Panel

An IRA Expert Panel assisted Biosecurity Australia in its consideration of the scientific issues during the risk analysis. The development of the IRA was led by Dr Mike Nunn, Principal Scientist, Animal Biosecurity. The Expert Panel comprised:

- Dr Patricia Ellis, Animal Health Consultant with longstanding involvement in the horse industry. She was involved in the response to the equine influenza outbreak in Australia.
- Dr James Gilkerson, Director, Equine Infectious Disease Laboratory and Centre for Equine Virology, University of Melbourne. He is an experienced veterinary virologist.
- Dr Hugh Millar, Executive Director Biosecurity Victoria and Chief Veterinary Officer, Victoria. He has experience with biosecurity policy and quarantine operations.

A number of Biosecurity Australia staff contributed to the development of this IRA report.

1.4 Scope

The IRA report considers the quarantine risks that may be associated with the importation of horses into Australia from the countries and regions from which Australia currently permits such importation. The countries and regions from which Australia currently permits imports of horses are referred to as approved countries and are listed in section 1.4.1.

Potential disease agents of quarantine concern that could be introduced into Australia through the importation of horses were considered in the IRA, regardless of their presence or absence in approved exporting countries. Risk assessments were not conducted for diseases that are present in Australia or are not of quarantine concern.

However, recommended quarantine conditions include measures to manage any incident of disease that is not of quarantine concern, which might occur in horses during export to Australia or in PAQ, and that may impact adversely on quarantine arrangements.

Quarantine risks associated with transport routes, transit and transhipment were assessed, and the current transport conditions were reviewed. Measures to manage these risks have been incorporated in this IRA report and include contingencies for unforseen events that might pose an increased quarantine risk to Australia or disrupt quarantine arrangements.

Horses are imported into Australia for breeding, racing, competition and other purposes. Horses, donkeys and mules are considered in the IRA. Quarantine measures currently exist for the importation of zoo equids (including zebras, Przewalski's horses and other non-domesticated equids) from some approved countries. Importation of equids into zoos poses different risks to the importation of domestic equids, thus zoo equids are not considered in the IRA.

In accordance with the SPS Agreement, IRAs assess risks to human, animal and plant life or health. Under Australian administrative arrangements, Biosecurity Australia provides advice to Australia's Director of Animal and Plant Quarantine on the life or health of animals and plants. Risks to human health are the responsibility of the Australian Government Department of Health and Ageing.

1.4.1 Approved countries

Countries, administrative regions and territories from which Australia currently permits the importation of horses, are referred to in the IRA as approved countries. These include:

- Europe: Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland (Republic of), Italy, Luxembourg, the Netherlands, Portugal, Spain, Sweden, Switzerland and the United Kingdom
- North America: Canada and the United States
- Asia: Hong Kong (Special Administrative Region), Macau and Singapore
- Middle East: United Arab Emirates
- Pacific Region: New Zealand.

The direct importation of horses from Japan was suspended following the outbreak of equine influenza in Japan in August 2007. Biosecurity Australia received information from Japan regarding this outbreak and subsequent activities that were undertaken by animal health authorities in Japan. Officers from Biosecurity Australia visited Japan in June 2009 to obtain further information on monitoring and surveillance for equine influenza and to observe horse quarantine facilities and procedures. Revised quarantine measures for the importation of horses from Japan are considered as part of the generic measures in this IRA. Thus for the purposes of this IRA, Japan is considered an approved country.

As detailed in Animal Quarantine Policy Memorandum 1999/62 Australia takes into account the following criteria when considering the approval of countries to export animals and their products to Australia:

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

If other countries with a long history of trade with Australia wish to be added to the list of approved countries, a detailed assessment taking into account these criteria would be required to determine if Australia's quarantine requirements could be met.

2 The horse industry in Australia

2.1 Industry structure

The horse industry in Australia is a large industry, diverse in structure and function. It uses large areas of land, contributes to export earnings and creates considerable economic activity in rural and urban communities (Pilkington and Wilson 1993). The significant size and economic impact of the racing sector is well documented but reliable and precise information on the relative importance of other sectors is difficult to obtain.

Horses in Australia are used for racing, breeding, sporting activities, recreation, regulatory purposes (police horses), tourism, stock work and meat production (pet food and meat exported for human consumption).

A broad range of ancillary service providers depend for their livelihood on the horse industry. Many others work part-time in related industries.

Many people in the horse industry belong to breed and activity organisations. Records held by the Australian Horse Industry Council (AHIC) suggest that nationally there are 15–20 large horse industry organisations and approximately 100 smaller ones. AHIC member organisations claim memberships in excess of 100 000 people, with horse registrations exceeding 500 000. However, large numbers of horse owners do not belong to any particular organisation while many horse owners belong to more than one organisation or breed society. Thus, it is difficult to draw conclusions from registry information or membership records.

Horse-related activities play an important part in the social amenity of many Australians, across all age groups, both actively as riders or drivers or passively as spectators.

A large number of Australians ride horses for recreation. In a 1998 survey of nearly two million Victorian households, 8.8% of respondents had ridden a horse at least once in the previous three months and 90.9% cited recreation as the main reason for riding a horse (Australian Bureau of Statistics 1998). The youth movement, Pony Club Australia, has just under 1000 clubs and 55 000 members (Pony Club Australia 2008). Equestrian Australia has 19 000 members and covers the disciplines of dressage, jumping, eventing, vaulting, show horse and carriage driving and is involved in the administration of international endurance riding and para-equestrian competitions. About 350 equestrian clubs and organisations with around 50 000 members are affiliated with Equestrian Australia. Some people may be both members of Equestrian Australia and other clubs and organisations but the extent of the overlap is unknown (F. Venhaus, Equestrian Australia, pers. comm. April 2009).

Measured by attendances, racing is Australia's second most popular spectator sport after Australian Rules Football (Australian Bureau of Statistics 2007). The Melbourne Cup attracts a global television audience of 700 million from 120 countries and territories (IER 2007). Many Australians also attend other horse sporting events such as polo, polocrosse, camp drafting, dressage, show jumping, eventing and rodeos.

2.1.1 Economic Data

Gross value of production

The major area of economic activity is the domestic market for horses and related goods and services. Estimates for quantifying the contribution of the horse industry to the Australian economy vary. There is a lack of reliable data relating to the size and structure of the Australian horse industry, particularly the non-racing sector.

The gross value of production (GVP) for the entire horse industry is difficult to assess because what constitutes 'final sales' is hard to define, and because of the industry's scope and complexity. Only a relatively small share of the horse industry economic activity is made up of final sales of horses, and much of the value of sales reflects the training component of production (Centre for International Economics 2007).

The GVP for the Australian horse industry in 2005–06 (Table 2.1) was estimated at A$3.6 billion per year (Centre for International Economics 2007). Betting, education and industry research and development were not included in this GVP estimate nor was flow-on expenditure on events such as transport costs for spectators, catering and accommodation.

Component	GVP ($ million)
Breeding	1 053.2
Racing	738.8
Equestrian	1 807.4
Horse related businesses	12.7
TOTAL	**3 612.1**

Table 2.1 Estimated annual GVP of the horse industry by sector in 2005–06 (Centre for International Economics 2007).

Thoroughbred racing

In August 2007, the Australian Racing Board (ARB) released a report on the size and scope of the thoroughbred racing industry in Australia using where possible data from the 2005–06 racing season (IER 2007).

Some of the major findings of the ARB report were:

- Economic activity generated by the Australian thoroughbred racing industry provides more than A$5.04 billion in value added to the national economy, representing about 0.58% of gross domestic product (GDP).
- The Australian thoroughbred racing industry directly employs more than 65 500 people. This is of similar size to the electricity, gas and water supply sector. A further 67 300 people are employed in down-the-line supplier and service industries.
- Significant employment and economic activity are generated in regional areas.
- The thoroughbred racing industry generates taxation revenue from a number of sources including betting taxes and Goods and Services Tax (GST). Each year, the Federal Government receives more than A$560 million and the state governments receive A$610 million in taxation revenue.

Economic activity in thoroughbred racing is significantly increased during major racing carnivals. For instance, it is estimated that the Victorian Spring Racing Carnival generates more than A$221.8 million in real gross value added (A$524 million in gross economic benefit) for Victoria, and a total of 730 000 interstate and international visits over 50 days (IER & Centre of Policy Studies, cited in Frontier Economics 2008). In 2007, the carnival suffered a 13.8% downturn in gross economic benefit as a consequence of the 2007 equine influenza outbreak in New South Wales and Queensland, mainly due to a downturn in interstate and overseas attendance (Anonymous 2008).

Harness racing

Estimated GDP of the Australian harness racing industry based on relevant racing and breeding variables in each state (R. Pollock, Australian Harness Racing Council, pers. comm. 2008) is presented in Table 2.2.

	Estimated GDP (A$ million)	Estimated GDP (A$ million)
Season	2005–06	2006–07
Australia	1 478	1 562
New South Wales	713	761
Victoria	359	379
South Australia	63	66
Western Australia	116	124
Queensland	190	192
Tasmania	36	38

Table 2.2 Estimated GDP of the Australian harness racing industry.

Non-racing sector

Obtaining a robust estimate of the value of the non-racing sector is very difficult (Frontier Economics 2008). However, using a number of approaches, including non-market valuation techniques and costs of horse ownership, the estimated total economic value of the Victorian non-racing sector is A$0.18–0.63 billion per year with the major economic impacts in regional Victoria.

Assuming a non-racing Australian domestic horse population of 435 000 (Gordon 2001) and applying the same approaches as Frontier Economics (Frontier Economics 2008), an estimate of the economic value of the national non-racing sector per year would be A$9.8–15.4 billion.

Australian horse industry as a whole

An earlier economic impact study published in 2001 by the Rural Industries Research and Development Corporation estimated that as a whole, the Australian horse industry contributed over A$6.3 billion to the GDP annually (Gordon 2001). The annual contribution of the racing sector was estimated to be A$3.4 billion and the non-racing

sector A$2.9 billion. These estimates did not include volunteer labour, estimated to be worth another A$1.7 billion (Gordon 2001).

Number and location of horses

Accurate horse numbers by Australian state and territory are not readily available and any information is acknowledged to be inaccurate and incomplete. The Australian Bureau of Statistics (ABS) records the number of horses on establishments with agricultural activity but these are known to underestimate total horse numbers.

A recent estimate is that there were a total of 932 000 domesticated horses in Australia (Centre for International Economics 2007). The majority of these (85%) are located in the south eastern portion of Australia in New South Wales, Queensland and Victoria (Gordon 2001) where most of the human population is also situated (Figure 2.1).

Figure 2.1 Density of domestic horses in Australia (Australian Bureau of Statistics 2006)[1].

There are also over 400 000 feral horses in Australia, mostly in remote arid and semi-arid regions of the Northern Territory, western and northern Queensland, South Australia, and the northern rangelands of Western Australia. There are also isolated populations in New South Wales and Victoria and occasional incursions into the Australian Capital Territory (Figure 2.2). Feral horses have potential to harbour and transmit exotic disease, though their remoteness may limit their ability to affect domesticated populations. Feral horses are controlled in all mainland states and territories, sometimes in conjunction with feral donkeys and camels (Dawson et al. 2006).

[1] Raw data on stud and other horses collected by the Australian Bureau of Statistics were interpolated to give a qualitative national coverage of the density of domestic horses.

Figure 2.2 The distribution of feral horses in Australia (adapted from Dobbie et al. 1993).

Australian horsemeat industry

Between 30 000 and 40 000 horses are processed for human and pet consumption each year. Meat processed for human consumption is exported and cannot be sold domestically. Horsemeat has been exported since the 1970s. Of the horses processed in Australia, about 20% are feral horses, harvested mainly from the northern regions. Horsemeat for export must be processed in export-accredited abattoirs.

A detailed review of the horsemeat industry has been published (Pilkington and Wilson 1993). According to the Australian Quarantine and Inspection Service (AQIS), the largest export volume was 6137 tonnes in 1998–99, valued at A$26.4 million, with more than half exported to Japan. In 2006–07, 2320 tonnes of horsemeat were exported to 14 countries with the majority going to Russia (48%), Switzerland (15%), Belgium (14%) and France (11%). The total value of exports in 2006–07 was A$10.3million (DAFF 2009). Other saleable by-products include horse hides, hair and bone meal.

Currently, there are two abattoirs that are export-accredited, one in Queensland and the other in South Australia. For the domestic market, there are numerous licensed knackeries that produce pet food from horsemeat.

Other equids

Zoo equids

Zoological gardens in Australia hold collections of the following equids, some of which are endangered species:

- onager (*Equus hemionus onager*)

- plains zebra (*Equus burchellii*)
- Przewalski's horse (*Equus ferus przewalskii*)

Donkeys

Donkeys were first imported to Australia in 1866 to serve as pack animals and in haulage teams. They were also bred with station horses to produce mules for heavy work. Australia now has an estimated five million feral donkeys in arid central Australia, the Kimberly in Western Australia and the Top End in the Northern Territory (DEH 2004). Some are harvested for pet meat when they can be obtained at low cost. Few donkeys are in domestic use today and mules are rarely bred.

No reliable estimate of the number of domestic donkeys is available.

2.2 Domestic trade in horses

The pattern of domestic trade is such that there is significant potential for rapid and extensive transmission of infectious horse diseases because nearly every activity undertaken with horses involves movement to and from a home property. Every day, large numbers of horses move rapidly and widely within Australia by road transport. Horses are frequently transported over long distances to and from points of congregation such as sales, race meetings and sporting events and then dispersed to various locations (Constable et al. 2000).

There are a few large commercial horse transport companies with extensive national networks and many smaller operators who service horse transport hubs on a local or regional basis. Significant numbers are also transported by owners or trainers in privately owned vehicles. Occasionally, racehorses are transported domestically by air, usually to and from Western Australia. Horses also are transported between Victoria and Tasmania across Bass Strait by ferry in horse floats and trucks. Depots are regularly used as transport hubs. Horses sometimes stay for only a few hours at these depots or change vehicles immediately after arrival and then depart for another local or interstate destination.

Horses move freely within and between most states and territories in Australia but there are some restrictions. Cattle tick, the most serious external parasite of cattle in Australia, can attach to horses and be spread via horse movements. For this reason, there are legislative restrictions in north-eastern New South Wales, the Northern Territory, Queensland and Western Australia for horses moving out of tick-infested areas into tick-free areas. Horses are required to report to designated crossing points for inspection, treatment and/or a permit. There are also legislative restrictions on the movement of horses into Western Australia to prevent the entry of liver fluke (*Fasciola hepatica*) and exotic noxious weeds.

Public auctions are common in the thoroughbred industry, however the majority of horses are sold privately.

Sales results for Australian thoroughbreds since 1996–97 have been summarised (Australian Racing Board 2008). Gross sales and median prices have increased steadily but recently levelled out. In the 2007–08 season, 4903 yearlings were auctioned for A$372 million, a decrease of 1% compared to the 2006–07 gross of

A$376 million. The median yearling price was A$25 000, a 4% increase over the A$24 000 median recorded in 2006–07.

Sales results and statistics for sales of other breeds are more difficult to obtain, but there is a robust market for elite horses in the non-racing sector. For instance, at the 2008 Dalby Stockhorse Sales, 279 horses sold for a gross of A$2.5 million at an average price of A$8940. The top price was for a mare that sold for A$70 000 (ASHS 2009).

Elite dressage and show jumping horses can command prices of several hundreds of thousands of dollars.

2.3 International trade in horses

Major discrepancies between sources relating to Australia's international trade in horses have been reported (Pilkington and Wilson 1993). Pilkington and Wilson (1993, p. 26) wrote:

> 'Definitions used by the stud books, Australian Quarantine and Inspection Service (AQIS) and Customs have differing purposes and lead to figures which cannot easily be compared or readily analysed to obtain a true indication of the size of the trade. Figures differ depending on sex and purpose of the export. Breeding animals attract different tariffs in destination countries to racing animals. Some destinations are not affiliated with the International Stud Book and so documentation from the Australian Stud Book [for thoroughbred horses] is not warranted.'

In 2009, these discrepancies remain.

2.3.1 Economic value

Horses are regularly exported from Australia to a wide variety of destinations for both competition and breeding purposes. Horse exports have traditionally contributed to the Australian economy since horses were first exported to India in the 1830s for use by the British Army as cavalry remounts. In the following 100 years, about 350 000 horses were exported to India (Pilkington and Wilson 1993). Freedom from significant equine diseases, such as African horse sickness, has underpinned Australia's status as a preferred supplier of healthy horses since the start of exports to India (Yarwood 1989) until today.

The annual economic value of international trade in horses since 2000 is summarised in Table 2.3.

Year	Value Imports (A$ million)	Value[2] Exports (A$ million)	Trade surplus (A$ million)
2000	96.6	91.9	- 4.7
2001	65.4	107.6	42.2
2002	84.2	122.0	37.9
2003	81.8	116.8	35.0
2004	66.1	107.7	41.6
2005	95.5	111.4	18.9
2006	123.3	117.7	- 5.6
2007	106.2	118.8	12.6
2008	138.5	134.1	- 4.4

Table 2.3 Estimated value of imports and exports of live horses by financial year (Australian Bureau of Statistics 2009).

The annual value of horse exports from Australia has steadily increased during the past decade. A trade surplus has been achieved in most years, rectifying the long term trade deficit in horses (Pilkington and Wilson 1993).

In 1993, the annual value of Australian horse exports had been static since 1984–85 at A$20–40 million, but compared favourably to the annual export income generated by sheep and cattle exports (Pilkington and Wilson 1993).

In 2008, the annual export income generated by cattle (A$534.2 million) and by sheep exports (A$330.3 million) far exceeded that by horse exports (A$134.1 million) (Australian Bureau of Statistics 2009).

The annual number of horses imported to, and exported from, Australia since 2000 is summarised in Table 2.4.

[2] Excludes value of horses exported for racing and breeding.

Year	Imports (number of horses)	Exports[3] (number of horses)
2000	5 022	2 976
2001	1 999	6 724
2002	1 915	3 217
2003	1 866	3 757
2004	2 133	5 309
2005	2 835	2 540
2006	3 616	2 791
2007	2 429	2 373
2008	2 704	2 308

Table 2.4 Estimated number of live horse imports and exports by financial year (Australian Bureau of Statistics 2009).

2.3.2 Exports: numbers and breeds

Currently, 2300–2500 horses are exported from Australia annually, primarily by air. Of these, the majority are thoroughbred racehorses. Some standardbreds are also exported for racing. A variety of other breeds, including warmbloods and Arabians, are also exported for equestrian sports or recreational use. In the past decade, a solid market has been developed in Middle Eastern countries for Arabian horses used for endurance riding.

Detailed export statistics by number and country of destination are only available for the thoroughbred breed and are kept by the Australian Stud Book. A summary is available (Australian Racing Board 2008) and the yearly summary by region (Figure 2.3) is presented on the Australian Stud Book website (Australian Jockey Club Limited and Victoria Racing Club Limited 2009)[4].

[3] Excludes horses exported for racing and breeding.

[4] Migration data can only be accessed online by Australian Stud Book subscribers.

Figure 2.3 **Horse exports by region in the 2008 season (Australian Jockey Club Limited and Victoria Racing Club Limited 2009).**

New Zealand is Australia's largest single trading partner, accounting for about 30% of thoroughbreds exported since 1996–97. The international market for Australian thoroughbred horses to Asian countries is also robust including to Hong Kong, Singapore, Malaysia, Korea, China and Macau which collectively accounted for over 60% of Australia's horse exports over the last decade.

In 2006–07, total thoroughbred exports increased significantly to 2362 horses, the best performance since 2000 when there were major exports to China and Malaysia. In the following season, the 2007 equine influenza outbreak in Australia had a profound impact on trade and exports decreased by 80% from 2362 to 469 horses (Australian Racing Board 2008).

2.3.3 Imports: numbers and breeds

Currently 2000–3000 horses are imported to Australia annually, primarily by air (Tables 2.4 and 2.5).

Year	New Zealand	All other countries	Total horses imported
1996	931	169	1 100
1997	1 358	131	1 489
1998	2 411	493	2 904
1999	5 330	549	5 879
2000	4 714	765	5 479
2001	1 972	489	2 461
2002	2 009	538	2 547
2003	1 622	548	2 170
2004	1 829	717	2 546
2005	2 194	628	2 822
2006	2 247	897	3 144
2007 (to October)	2 079	542	2 621

Table 2.5 Estimated number of horses imported into Australia, from 1996 to October 2007[5].

Air freight and quarantine costs tend to limit imports to horses of significant financial or sentimental value. The majority of horses imported from approved countries (other than New Zealand) arrive in Australia during the months of July and August.

Information from commercial sources indicates that 550–600 horses of all breeds are imported annually from approved countries (other than New Zealand) — 56% from Europe, 40% from the United States and 4% from Asia.

Between 2006 and 2008, the breed distribution of imported horses was: thoroughbreds (52%) — comprising breeding stallions (11%), racehorses in training (3%) and other breeding stock (38%); warmbloods (11%); standardbreds (9%); Arabians (5%); ponies and cobs (5%); miniatures (3%) and others (15%). 'Others' includes breeds such as Appaloosas, quarter horses, Hanoverians, shires, Percherons and polo ponies and event horses.

The Australian Stud Book has kept statistics of registered imported thoroughbreds by number and country for an extended period (Australian Racing Board 2008). The majority (77%) of registered thoroughbred horses imported to Australia since 1996–1997 have originated from New Zealand. Other sources include the United States (11.4 %), Ireland (5%), Great Britain (3.8%), France (0.7%) and Canada (0.5%).

Figure 2.4 presents an overview of imports by region in 2008 (Australian Jockey Club Limited and Victoria Racing Club Limited 2009). It should be noted that the country

[5] Figures from AQIS Live Animal Imports (2007).

of origin recorded by the Australian Stud Book may reflect the country of studbook registration not the country from which the horse was directly imported.

Figure 2.4 **Thoroughbred horse imports in 2008 by region (Australian Jockey Club Limited and Victoria Racing Club Limited 2009).**

Imports of thoroughbred horses were severely impacted by the equine influenza outbreak in Australia in 2007 (Australian Racing Board 2008) and imports decreased by 60% (see Figure 2.5).

Figure 2.5 **Exported and imported thoroughbred bloodstock[6] from 1996–97 to 2007–08 (Australian Jockey Club Limited and Victoria Racing Club Limited 2009).**

[6] These figures only include permanently imported and exported thoroughbred bloodstock.

2.4 Horse health in Australia

2.4.1 Equine diseases in Australia

OIE-listed diseases capable of affecting horses that occur in Australia include:

- anthrax (limited distribution)
- equine infectious anaemia (limited distribution)
- equine rhinopneumonitis (EHV-1 and EHV-4)
- equine viral arteritis
- Japanese encephalitis (Torres Strait)
- leptospirosis

Other infectious diseases that are present in Australia and capable of infecting horses include:

- botulism
- dermatophilosis
- equine coital exanthema (EHV-3)
- equine rotavirus
- Hendra virus (restricted distribution)
- melioidosis (restricted distribution)
- papillomatosis
- *Rhodococcus equi*
- Ross River virus
- strangles
- tetanus

Nationally notifiable diseases

The following diseases capable of affecting horses are included on Australia's National Notifiable Animal Disease List, April 2008 (DAFF 2008):

- African horse sickness
- anthrax
- Aujeszky's disease
- Borna disease
- brucellosis

- contagious equine metritis
- dourine
- encephalitides (tick-borne)
- epizootic lymphangitis
- equine encephalomyelitis (Eastern, Western and Venezuelan)
- equine encephalosis
- equine herpesvirus 1 (abortigenic and neurological strains)
- equine infectious anaemia
- equine influenza
- equine piroplasmosis (*Babesia caballi* and *Theileria equi*)
- equine viral arteritis
- Getah virus infection
- glanders
- Hendra virus infection
- Japanese encephalitis
- louping ill
- Nipah virus infection
- Potomac horse fever
- rabies
- salmonellosis (*Salmonella* Abortusequi)
- screw-worm-fly – New World (*Cochliomyia hominovorax*)
- screw-worm-fly – Old World (*Chrysomya bezziana*)
- surra (*Trypanosoma evansi*)
- trichinellosis
- tuberculosis (*Mycobacterium bovis*)
- vesicular stomatitis
- warble-fly myiasis
- West Nile virus infection – clinical

2.4.2 Australian Veterinary Emergency Plan

The Australian Veterinary Emergency Plan (AUSVETPLAN) (Animal Health Australia 2009) is Australia's national plan for responding in a consistent manner to

an outbreak, or suspected outbreak, of an emergency animal disease, anywhere in Australia.

Individual AUSVETPLAN Disease Strategy Manuals have been prepared for the following diseases capable of affecting horses:

- African horse sickness
- anthrax
- bovine brucellosis
- equine influenza
- Japanese encephalitis
- screw-worm-fly
- surra (*Trypanosoma evansi*)
- vesicular stomatitis

Response Policy Briefs are brief policy statements for an emergency animal disease that are subject to cost sharing between governments and livestock industries but which are not covered by full AUSVETPLAN Disease Strategy Manuals. Response Policy Briefs have been prepared for the following diseases capable of affecting horses:

- Borna disease
- brucellosis (due to *Brucella melitensis*)
- contagious equine metritis
- encephalitides (tick-borne) – includes louping ill
- epizootic lymphangitis
- equine babesiosis
- equine encephalomyelitis (Eastern, Western and Venezuelan)
- equine encephalosis
- Getah virus disease
- glanders
- Hendra virus
- Nipah virus
- Potomac horse fever
- trichinellosis
- West Nile virus infection – clinical

2.4.3 Emergency Animal Disease Response Agreement

The Emergency Animal Disease Response Agreement (EADRA) (Animal Health Australia 2001), ratified in March 2002, established new partnership arrangements between the Australian Government, state and territory governments, and peak livestock industry organisations. The Agreement is managed by Animal Health Australia and provides a framework for managing and funding responses to emergency animal disease incursions. It is regularly reviewed.

The signatories to the EADRA are committed to:

- minimising the risk of emergency animal disease incursions by developing and implementing biosecurity plans for their jurisdictions or industries
- maintaining an appropriate capacity to respond to an emergency animal disease by having available adequate numbers of trained personnel to fill roles specified in AUSVETPLAN
- participating in decision making relating to emergency animal disease responses, through representation on the Consultative Committee on Emergency Animal Diseases and a National Management Group
- sharing the eligible response costs of emergency animal disease incursions.

Currently, the Australian horse industry is not a signatory to the EADRA.

Diseases covered by the EADRA have been categorised according to the proportion of costs that will be shared between government and respective industries. Below is a list of scheduled diseases capable of affecting horses and the category assigned to them:

Category 1 (100% government funded response)

- Eastern, Western and Venezuelan equine encephalomyelitides
- Japanese encephalitis
- rabies

Category 2 (80% government; 20% industry funded response)

- Hendra virus infection
- glanders
- screw-worm-fly
- vesicular stomatitis

Category 3 (50% government; 50% industry funded response)

- African horse sickness
- anthrax
- trichinellosis

Category 4 (20% government; 80% industry funded response)

- Borna disease
- contagious equine metritis
- dourine
- epizootic lymphangitis
- equine babesiosis
- equine encephalosis
- equine influenza
- Getah virus infection
- Potomac horse fever
- surra

There are 11 significant diseases covered by the EADRA which affect horses only. For these, if the horse industry was a signatory to EADRA, the horse industry alone would share emergency response costs with government. There are also three other diseases — Borna disease, surra and vesicular stomatitis — for which the horse industry would share response costs with government and other livestock industries.

References

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

Animal Health Australia (2009) AUSVETPLAN. http://www.animalhealthaustralia.com.au/programs/eadp/ausvetplan/ausvetplan_home.cfm (Accessed 30 October 2009).

Anonymous (2008) 2007 Spring: an economic triumph despite EI. *Inside Racing* Mar.: 13-14.

ASHS (2009) News. Australian Stock Horse Society. http://www.ashs.com.au/news/default.asp (Accessed 20 November 2009).

Australian Bureau of Statistics (1998) *Safety in the home, Victoria*. No. 4387.2, Australian Bureau of Statistics, Canberra.

Australian Bureau of Statistics (2006) 7125.0- Agricultural commodities: small area data, Australia, 2005-06 (reissue). http://abs.gov.au/ausstats/abs@.nsf/Previousproducts/7125.0Main%20Features12005-06%20(Reissue)?opendocument&tabname=Summary&prodno=7125.0&issue=2005-06%20(Reissue)&num=&view= (Accessed 30 October 2009).

Australian Bureau of Statistics (2007) *Sport attendance, Australia*. Australian Bureau of Statistics, Canberra.

Australian Bureau of Statistics (2009) 5465.0- International trade, Australia: information consultancy subscription service, 2000. http://www.abs.gov.au/ausstats/abs@.nsf/productsbytitle/E0E5015B73608361CA25756D0013AF9F?OpenDocument (Accessed 30 October 2009).

Australian Jockey Club Limited, Victoria Racing Club Limited (2009) Australian stud book. http://www.studbook.org.au/ (Accessed 30 October 2009).

Australian Racing Board (2008) Australian racing fact book: a guide to the racing industry in Australia 2007-2008. Australian Racing Board. http://www.australianracingboard.com.au/factBook.html (Accessed 1 January 2009).

Centre for International Economics (2007) *Estimating Australian horse industry GVP and horse numbers: prepared for Animal Health Australia*. Centre for International Economics, Canberra and Sydney.

Constable FA, Tucak PB, Steel CM, Symons PJ (2000) Australian equine transport patterns and recommendations to prevent the dissemination of exotic disease. *Australian Equine Veterinarian* 18: 126-131.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

DAFF (2009) Horse industry background: Australian horsemeat industry. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/agriculture-food/meat-wool-dairy/ilg/industries/horses (Accessed 23 April 2009).

Dawson MJ, Lane C, Saunders G (2006) Proceedings of the national feral horse management workshop, Canberra, August 2006. In *Proceedings of the national feral horse management workshop, Canberra, August 2006,* (eds. Dawson MJ, Lane C, Saunders G) pp. 1-89, Invasive Animals Cooperative Research Centre, Canberra.

DEH (2004) Feral horse (*Equus caballus*) and feral donkey (*Equus asinus*) - invasive species fact sheet. Department of the Environment, Water and Heritage and the Arts. http://www.environment.gov.au/biodiversity/invasive/publications/pubs/feral-horse.pdf (Accessed 3 December 2008).

Dobbie WR, Berman DM, Braysher MI (1993) *Managing vertebrate pests: feral horses*. Australian Government Publishing Service, Canberra.

Frontier Economics (2008) *Benefits and costs related to Victorian management of equine influenza: a final report prepared for the Victorian Department of Primary Industries*. The Frontier Economics Network, Melbourne.

Gordon J (2001) *The horse industry - contributing to the Australian economy: a report for the Rural Industries Research and Development Corporation*. No. 01/083, Rural Industries and Development Corporation, Canberra.

IER (2007) *Economic impact of Australian racing*. Australian Racing Board Ltd, Sydney.

Pilkington M, Wilson G (1993) *Australian horses as a primary industry: numbers, organisation and research needs*. Australian Government Publishing Service, Canberra.

Pony Club Australia (2008) *Submission to the Senate Rural and Regional Affairs and Transport Committee Inquiry into the Horse Disease Response Levy Bill 2008: prepared by Pony Club Australia Inc*. Pony Club Australia Inc., Queensland.

Yarwood AT (1989) Origins: India and Australia. In *Walers: Australian horses abroad* p. 31. Melbourne University Press at the Miegunyah Press, Carlton.

3 Method for import risk analysis

The World Organisation for Animal Health (OIE) in its Terrestrial Animal Health Code (2009), hereafter referred to as 'the Code', refers to 'General Obligations related to Certification' in Chapter 5.1.

The Code states at Article 5.1.2. that:

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

Article 5.1.2. further states that:

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

The components of an import risk analysis (IRA), described in Chapter 2.1 of the Code, are:

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

While hazard identification, risk assessment and risk management occur consecutively within the context of a particular IRA, risk communication occurs in an ongoing and iterative manner throughout the process, and includes both formal and informal consultation with stakeholders. The release of this IRA report forms part of the risk communication process.

The method adopted by Biosecurity Australia for conducting IRAs conforms to that recommended by the OIE in Chapter 2.1 of the Code and is described in further detail in this chapter. Results of the hazard identification, including hazard refinement are detailed in chapter 4. Individual disease risk assessments and risk estimates are described in chapter 5. Proposals for risk management, for those disease agents for which the risk estimate exceeds Australia's appropriate level of protection (ALOP), are described in chapter 6.

This IRA considers the importation of horses from approved countries (see section 1.4.1 for details).

3.1 Hazard identification

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

In accordance with the Code, a disease agent was considered to be a potential hazard if it was assessed to be:

- appropriate to the species being imported
- present in the exporting country
- OIE-listed, or could potentially produce adverse consequences in Australia
- not present in Australia, or present in Australia and a notifiable disease or subject to control or eradication.

For this IRA, hazard identification was initiated by generating a comprehensive list of disease agents likely to be relevant to the importation of horses. The list includes disease agents listed by the OIE and known to affect horses, and any other diseases or disease agents considered relevant to horses. The list was subsequently refined by applying four criteria (above) for assessing a potential hazard. If reasons for the inclusion or exclusion of a particular disease agent were not clear cut, these agents were retained on the list and examined in the risk assessment.

3.2 Risk assessment

Risk assessment is defined in the Code as '… the evaluation of the likelihood and the biological and economic consequences of entry, establishment and spread of a hazard within the territory of an importing country'.

The Code notes that 'the principal aim of import risk analysis is to provide importing countries with an objective and defensible method of assessing the disease risks associated with the importation of animals … ' and further 'provides recommendations and principles for conducting transparent, objective and defensible risk analyses for international trade'.

Article 2.1.2 of the Code states that 'an importing country may decide to permit the importation using the appropriate sanitary standards recommended in the Terrestrial Code, thus eliminating the need for a risk assessment'. Each disease section in chapter 5 of the IRA states whether the Code recommendations exist for that disease agent and if the recommendations were used or a risk assessment was conducted.

In accordance with the Code, the 'release assessment describes the probability of the 'release' of each of the potential hazards (the pathogenic agents)' in an importing country and 'exposure assessment consists of describing the biological pathway(s) necessary for exposure of animals … and estimating the probability of the exposure(s) occurring'. The consequence assessment describes the potential consequences of a given exposure and estimates the probability of them occurring. The risk assessment for an identified disease agent concludes with risk estimation — the combination of the likelihood of release and exposure, and likely consequences of establishment and/or spread — and yields the unrestricted risk estimate.

Figure 3.1 illustrates the components of a risk assessment: the release, exposure and consequence assessments. Figure 3.2 provides an expanded schematic on the main components.

Figure 3.1 Components of risk assessment.

Chapter 2.1 of the Code provides recommendations for conducting IRAs, describing release, exposure and consequence assessments and their integration into a risk estimation, producing overall outcome of the risks associated with the hazards identified at the outset.

3.2.1 Evaluating and reporting likelihood

In this assessment, Biosecurity Australia used available data sources, including information on related disease agents and host species.

This assessment was conducted using a qualitative approach. The likelihood that an event will occur was evaluated and reported qualitatively, using qualitative likelihood descriptors for the release and exposure assessment, and the outbreak scenario (Table 3.1).

Likelihood	Descriptive definition
High	The event would be very likely to occur
Moderate	The event would occur with an even likelihood
Low	The event would be unlikely to occur
Very low	The event would be very unlikely to occur
Extremely low	The event would be extremely unlikely to occur
Negligible	The event would almost certainly not occur

Table 3.1 Nomenclature for qualitative likelihoods.

3.2.2 Risk assessment framework

Horses have been imported into Australia since European settlement. Importation by air began in the 1970s and became routine from the mid-1990s. The associated risks with shorter travel times were taken into account in developing subsequent import policies. The evaluation of disease risks involved estimating the likelihood of susceptible animals in Australia becoming exposed to a disease agent and the likely consequences of such exposure.

In evaluating the likelihood of susceptible animals in Australia becoming exposed to a disease agent, the following factors were considered:

- the likelihood of the disease agent being released into Australia via an imported horse (release assessment)
- the likelihood of a susceptible animal becoming exposed to the disease agent via an imported horse (exposure assessment).

The determination of likely consequences required:

- identification of the most likely outbreak scenario that could follow exposure to a disease agent. Possible outbreak scenarios can range from no infection occurring to the agent establishing and spreading in a local population with further spread to other susceptible populations. Only the most likely outbreak scenario relating to establishment and/or spread for each disease agent was assessed
- estimation of the likelihood of establishment and/or spread for that outbreak scenario
- effects (health, environmental and socioeconomic) associated with that outbreak scenario.

Likelihoods were assigned to release, exposure and establishment and/or spread (outbreak) scenarios.

The overall construct of this risk assessment, including the exposure groups identified, is illustrated in Figure 3.2. Key steps in the process, and references to figures and tables, are given in Table 3.2.

The risk assessment considered the likelihood of entry and exposure of a disease agent over a period of one year. The release and exposure assessments for each disease agent were based on the expected annual volume of trade in horses. The volume of trade was taken as the average number of horses imported each year over the previous ten years.

This IRA did not consider Australia's current risk management measures for imported horses when estimating risk. The IRA thus concluded with an unrestricted risk for each disease agent. If the unrestricted risk did not achieve Australia's ALOP, described in section 1.2.1, then risk management measures were recommended to reduce the risk in order to achieve the ALOP.

The outbreak scenario resulting from the exposure of susceptible animals was considered in a single pathway resulting in infection and establishment. Detailed disease considerations were discussed in the relevant disease chapter.

Figure 3.2 Overall construct of the risk assessment.

This pathway is deconstructed and reproduced in detail in later figures.

Likelihood / Risk factor	Estimation / description	Reference
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	Section 3.2.3, Table 3.1, Figure 3.3
Likelihood of *exposure*	Likelihood of *exposure*	Section 3.2.4, Table 3.1, Figure 3.4
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	Section 3.2.5, Table 3.3
Consequences assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	Section 3.2.6, Table 3.1
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	Section 3.2.6, Table 3.4, Figure 3.5
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	Section 3.2.6, Table 3.5
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	Section 3.2.7, Table 3.6

Table 3.2 Key steps in estimation of unrestricted risk.

3.2.3 Release assessment

The release assessment considered a single release scenario, in which horses were randomly sourced from the general horse population in the approved country.

Figure 3.3 Release pathway.

The release pathway considered the following relevant steps:

- presence of the disease agent at premises of origin of the horse
- whether the selected horse is infected
- whether the disease agent is detected at pre-export inspection (independent of any subsequently applied risk management measures)
- whether the horse is infected during transport
- whether the disease agent is detected at an Australian port of entry.

No risk management measures were considered in the unrestricted release scenario, except basic evaluation of health and fitness to travel by the certifying authority in the country of origin. With the exception of New Zealand, from where some horses are exported by sea, all horses exported to Australia are freighted by air in purpose built air stalls. The likelihood of release for each disease agent was based on the unrestricted release scenario.

3.2.4 Exposure assessment

For the purposes of this IRA, to determine the unrestricted risk estimate, the exposure assessment commenced at the point of arrival of horses in Australia. Figure 3.4 illustrates the potential for transmission by different pathways. For each disease agent, the most likely pathway was selected for analysis.

The exposure assessment considers multiple exposure groups, and potential exposure pathways of the disease agent by vectors, direct contact or fomites. The exposure group was considered to be horses and other susceptible species (referred to as 'susceptible animals'). The recognised exposure groups in this IRA were:

- equids (including feral equids)
- other domestic species (including other non-ruminants and ruminants, feral animals)
- wildlife (Australian native animals).

Non-susceptible animals were not considered. For each disease agent, the final outcome of the exposure assessment was an estimate of the likelihood that susceptible animals were exposed to the disease agent i.e. the likelihood of exposure.

The likelihood estimation of the exposure assessment did not consider Australia's current risk management measures for imported horses

Figure 3.4 **Potential exposure pathways (single exposure group).**

3.2.5 Estimation of the likelihood of release and exposure

The estimation of the likelihood of release and exposure involved consideration of the volume of trade in horses to be imported during a prescribed period. The period chosen by Biosecurity Australia is one year, which was considered a sufficient period to enable evaluation of seasonal effects. Data provided by the Australian Quarantine and Inspection Service (AQIS) show that for the last ten years, each year, Australia imported over 2000 horses from New Zealand and approximately 500 horses from elsewhere.

The likelihood of release and exposure was the estimated likelihood that there was at least one exposure event during an average year for the expected number of horses imported from countries where the disease being assessed was endemic.

The likelihood of release and exposure was estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix shown in Table 3.3.

The basis for combining qualitative likelihoods using a matrix is described by Standards Australia and Standards New Zealand (Standards Australia 2005).

	High	Moderate	Low	Very Low	Extremely low	Negligible
High	High	Moderate	Low	Very low	Extremely low	Negligible
Moderate	Moderate	Low	Low	Very low	Extremely low	Negligible
Low	Low	Low	Very low	Very low	Extremely low	Negligible
Very low	Very low	Very low	Very low	Extremely low	Extremely low	Negligible
Extremely Low	Extremely low	Extremely low	Extremely low	Extremely low	Negligible	Negligible
Negligible	Negligible	Negligible	Negligible	Negligible	Negligible	Negligible

Table 3.3 Matrix for combining qualitative likelihoods.

3.2.6 Consequence assessment

Criteria for assessing consequences associated with a pest or disease incursion are outlined in relevant Australian legislation and international agreements, and in the standards prepared by the OIE. In particular:

- the *Quarantine Act 1908* requires decision makers to take into account the probability of harm being caused (to humans, animals, plants, other aspects of the environment, or economic activities) and the probable extent of the harm (Section 5D)
- the SPS Agreement[7] states that 'Members shall take into account as relevant economic factors: the potential damage in terms of loss of production or sales in the event of the entry, establishment or spread of a pest or disease; the costs of control or eradication in the territory of the importing Member; and the relative cost-effectiveness of alternative approaches to limiting risks.'
- the Code expands the 'relevant economic factors' described in the SPS Agreement and provides examples of factors that will typically be relevant to an IRA. In each case, consequence assessments do not extend to considering the benefits or otherwise of trade in a given commodity, nor to the effect of import competition on industries or consumers in the importing country.

The Code also states that a consequence assessment 'describes the potential consequences of a given exposure and estimates the probability of them occurring'. This approach is reflected in the *Quarantine Proclamation 1998*, which requires that the 'level of quarantine risk' is considered in making quarantine decisions (Section 70).

In this IRA, likely consequences are considered for those attributable to the most likely outbreak scenario. These were addressed in terms of direct and indirect effects on animal and plant life and health on a national scale, including adverse health,

[7] SPS Agreement – World Trade Organization Agreement on the Application of Sanitary and Phytosanitary Measures.

environmental and socioeconomic effects (as detailed below), and separately in terms of consequences to human life or health. The latter is dealt with separately because primary responsibility for matters of human life or health rests with the Australian Government Department of Health and Ageing.

The following sequence of steps was taken in determining the likely consequences associated with an outbreak scenario:

1. identification of the most likely outbreak scenario (detailed in the relevant disease chapter) that may occur as a result of release of a disease agent and exposure to a susceptible animal
2. estimation of the likelihood of the outbreak scenario occurring to obtain a likelihood of establishment and/or spread
3. determination of the effects (health, environmental and socioeconomic) resulting from the outbreak scenario
4. combination of the likelihood of establishment and/or spread for the outbreak scenario with the corresponding overall effect to obtain an estimation of likely consequences.

Identification of an outbreak scenario

Once exposure of a susceptible animal has occurred, a number of possible outbreak scenarios could follow, representing a continuum ranging from no spread to widespread establishment of disease. For risk assessment purposes, outbreak scenarios were considered based on the epidemiology of each disease agent and described in each disease chapter. The outbreak scenario considered was dependent on detection of the disease agent in susceptible animals. The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. Consequences were assessed after the most likely outbreak scenario was selected.

For each disease agent, the likelihood of establishment and/or spread, and the associated overall effect for the outbreak scenario was determined. The likely consequences were determined using the matrix at Table 3.5.

Likelihood of establishment and/or spread associated with the outbreak scenario

When estimating the likelihood of establishment and/or spread associated with the outbreak scenario, qualitative descriptors such as ‘negligible’, ‘low’, and ‘moderate’ were used as detailed in Table 3.1.

Determination of the effects (health, environmental and socioeconomic) of establishment and/or spread

Potential effects of establishment and/or spread associated with the outbreak scenario may be direct or indirect. Consideration of effects was not limited to what might occur during one year, but covered a period as long as effects continued to be discernible. Adverse effects were evaluated in terms of seven (two direct and five indirect) criteria.

Direct effects

These describe effects on:

- life or health (including production effects) of susceptible animals
- the living environment, including life and health of wildlife, and any effects on the non-living environment.

Indirect effects

These describe effects on:

- new or modified eradication, control, monitoring or surveillance and compensation strategies or programs
- domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries
- international trade, including loss of markets, meeting new technical requirements to enter or maintain markets and changes in international consumer demand
- the environment, including biodiversity, endangered species and the integrity of ecosystems
- communities, including reduced tourism, reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures.

Effects were considered mutually exclusive, that is, an effect was not assessed more than once. Direct effects were considered separately from indirect ones.

Describing effects

Estimating the overall effect of establishment and/or spread associated with the outbreak scenario involved a two-step process where first, a qualitative descriptor of the effect of a disease agent was assigned to each of the identified direct and indirect criteria in terms of the *level of effect* and the *magnitude of effect*. The second step involved combining the effects for each of the seven criteria to obtain an estimation of the overall effect of establishment and/or spread.

Step 1: Assessing direct and indirect effects

Each direct and indirect effect was estimated at four levels — national, state or territory, district or regional, and local — and the values derived subsequently translated into a single qualitative score (A to G). In this context, the terms 'national', 'state or territory', 'district or region' and 'local', were defined as follows:

National	Australia-wide
State/ Territory	an Australian 'State' (New South Wales, Victoria, Queensland, Tasmania, South Australia or Western Australia) or 'Territory' (the Australian Capital Territory, the Northern Territory, the Australian Antarctic Territory and

other Australian Territories covered under the Quarantine Act)[8].

District/ Region	a geographically or geopolitically associated collection of aggregates — generally a recognised section of a State or Territory, such as the 'North West Slopes and Plains' of New South Wales or 'Far North Queensland'.
Local	an aggregate of households or enterprises — e.g. a rural community, a town or a local government area.

At each level, the magnitude of effect was described as 'unlikely to be discernible', of 'minor significance', 'significant' or 'highly significant':

- An 'unlikely to be discernible' effect is not usually distinguishable from normal day-to-day variation in the criterion.
- An effect of 'minor significance' is recognisable, but minor and reversible.
- A 'significant' effect is serious and substantive, but reversible and unlikely to disturb either economic viability or the intrinsic value of the criterion.
- A 'highly significant' effect is extremely serious and irreversible and likely to disturb either economic viability or the intrinsic value of the criterion.

When assessing effects, the frame of reference was the effect of each disease agent on the community as a whole and not just on directly affected parties. Related considerations were the disease agent, its persistence and geographic extent. In general, effects were considered greater if the effect was prolonged, as would be the case if the disease agent was expected to persist for several production cycles or if restocking following eradication programs was expected to take several generations. If an effect was not prolonged, consequences were considered likely to be less serious. Similarly, a disease agent with limited trade effects or pathogenicity but widespread at detection may have been considered to have less serious effects than a disease agent limited geographically but with major effects on trade.

Step 2: Combining direct and indirect effects

To estimate the overall effects of a disease outbreak on a national scale, it was necessary to combine the direct and indirect effects on the national economy or the Australian community. The effects were combined by first translating each individual direct or indirect effect to a national effect score (A–G) using the schema outlined in Table 3.4. This was done by determining which of the shaded cells with bold font in the Table corresponded to the level and magnitude of the particular effect. At each of the lower geographic levels, an effect more serious than 'minor' was understood to be discernible at the level above (e.g. a 'significant' effect at the state/territory level would be considered to be equivalent to at least a 'minor' effect at national level). In addition, the effect of a disease at a given level in more than one state/territory, district/region or local area was considered to represent at least the same magnitude of effect at the next highest geographic level.

[8] This excludes the Cocos (Keeling) Islands.

Once the appropriate shaded cell had been selected, the appropriate overall score for the outbreak scenario was assessed by reading the alphabetic (A–G) score from Table 3.4, starting at the national level and working down until the highest applicable combination of level and magnitude was reached. It is important to note that 'effect' at the national level is a different issue from 'spread of disease'. A disease may have serious consequences at the national level, despite only occurring in a small area.

National Impact Score				
G	**Highly significant**			
F	**Significant**			
E	**Minor** ←	*Greater than 'minor' at State level equals at least 'minor' at National level*		
D	Unlikely to be discernible	**Minor** ←	*Greater than 'minor' at district/region level equals at least 'minor' at State level*	
C	-	Unlikely to be discernible	**Minor** ←	*Greater than 'minor' at Local level equals at least 'minor' at district/region level*
B	-	-	Unlikely to be discernible	**Minor**
A	-	-	-	**Unlikely to be discernible**
	national	*State or Territory*	*district or region*	*local*
	Geographical Level			

Table 3.4 Assessment of direct or indirect effects on a national scale[9].

The measure of effect (A–G) obtained for each direct and indirect criterion was combined to give the overall effect of a disease agent.

[9] Shaded cells with bold font are those that dictate national effect scores. Effects greater than 'minor' at local, district/regional or state/territory level are considered to represent at least 'minor' effects at the next higher geographic level.

The following rules (Figure 3.5) were used for the combination of direct and indirect effects. They should not be considered as likelihoods of occurrence.

1. Where the effect of a disease with respect to any direct or indirect criterion is G, the overall effect is '*extreme*'.
2. Where the effect of a disease with respect to more than one criterion is F, the overall effect is '*extreme*'.
3. Where the effect of a disease with respect to a single criterion is F and the effect with respect to each remaining criterion is E, the overall effect is '*extreme*'.
4. Where the effect of a disease with respect to a single criterion is F and the effect with respect to remaining criteria is not unanimously E, the overall effect is '*high*'.
5. Where the effect of a disease with respect to all criteria is E, the overall effect is '*high*'.
6. Where the effect of a disease with respect to one or more criteria is E, the overall effect is '*moderate*'.
7. Where the effect of a disease with respect to all criteria is D, the overall effect is '*moderate*'.
8. Where the effect of a disease with respect to one or more criteria is D, the overall effect is '*low*'.
9. Where the effect of a disease with respect to all criteria is C, the overall effect is '*low*'.
10. Where the effect of a disease with respect to one or more criteria is C, the overall effect is '*very low*'.
11. Where the effect of a disease with respect to all criteria is B, the overall effect is '*very low*'.
12. Where the effect of a disease with respect to one or more criteria is B, the overall effect is '*negligible*'.
13. Where the effect of a disease with respect to all criteria is A, the overall effect is '*negligible*'.

Figure 3.5 Rules used for the combination of direct and indirect effects.

Note: These rules are mutually exclusive, and were addressed in the order that they appear in the list. For example, if the first set of conditions did not apply, the second set was considered. If the second set did not apply, the third set was considered, and so forth until one of the rules applied. No further rule was considered.

Derivation of likely consequences

The likely consequences were estimated by combining the likelihood of establishment and/or spread (associated with the outbreak scenario) with the overall effect of establishment and/or spread using the matrix shown in Table 3.5.

Likelihood of establishment and/or spread						
High	Negligible	Very low	Low	Moderate	High	Extreme
Moderate	Negligible	Very low	Low	Moderate	High	Extreme
Low	Negligible	Negligible	Very low	Low	Moderate	High
Very Low	Negligible	Negligible	Negligible	Very low	Low	Moderate
Extremely Low	Negligible	Negligible	Negligible	Negligible	Very low	Low
Negligible	Negligible	Negligible	Negligible	Negligible	Negligible	Very low
	Negligible	Very Low	Low	Moderate	High	Extreme
	Overall effect of establishment and/or spread					

Table 3.5 **Likely consequences: a combination of the likelihood and overall effect of establishment and/or spread.**

3.2.7 Risk estimation

Risk estimation is the integration of likelihood of release and exposure, and likely consequences of establishment and/or spread. This derives the risk associated with release, exposure, establishment and/or spread of a disease agent introduced by the importation of horses into Australia.

Estimation of risks of release, exposure, establishment and/or spread

The risk is estimated by:

- determining the likelihood of release and exposure and then
- combining the likelihood of release and exposure with the estimate of likely consequences of establishment and/or spread.

Combining the likelihood of release and exposure and likely consequences of establishment and/or spread was undertaken using the rules shown in the risk estimation matrix in Table 3.6.

Likelihood of release and exposure						
High likelihood	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Moderate likelihood	Negligible risk	Very low risk	Low risk	Moderate risk	High risk	Extreme risk
Low likelihood	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk	High risk
Very low likelihood	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk	Moderate risk
Extremely low likelihood	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk	Low risk
Negligible likelihood	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Negligible risk	Very low risk
	Negligible effect	*Very low effect*	*Low effect*	*Moderate effect*	*High effect*	*Extreme effect*

Likely consequences of establishment and/or spread

Table 3.6 Risk estimation matrix.

Estimation of unrestricted risk

Risk evaluation is described in the Code as the process of comparing the estimated risk with a country's ALOP. The result of this process was an estimate of the unrestricted risk of introducing a disease agent into Australia as a result of importing horses. Key steps in estimating the unrestricted risks are summarised in Table 3.2. To obtain the unrestricted risk of release, exposure, establishment and/or spread, the likelihood of release and exposure was combined with the likely consequences of establishment and/or spread using the risk estimation matrix shown in Table 3.6.

A risk estimation that was either 'very low' or 'negligible' was considered sufficient to achieve Australia's conservative ALOP. This provided a benchmark for evaluating risk and determining whether risk management was required.

The use of a benchmark for evaluating risks for each disease agent is illustrated in the process outlined below:

- if the unrestricted risk was 'negligible' or 'very low', then it achieved Australia's ALOP and further risk management was not required
- if the unrestricted risk was 'low', 'moderate', 'high' or 'extreme', risk management measures were required.

This was considered the final output of the risk assessment.

Worked examples (Figures 3.6A, 3.6B and Tables 3.7A, 3.7B) used two hypothetical scenarios to demonstrate the combination of likelihoods and effects to derive an estimation of unrestricted risk using the risk assessment method described

Figure 3.6A **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for Disease A.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Low*
Likelihood of *exposure*	Likelihood of *exposure*	*Moderate*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	***Low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**LOW**

Table 3.7A Worked example showing combined release, exposure and consequence assessments, resulting in an unrestricted risk.

The unrestricted risk associated with Disease A is determined to be 'low'. The unrestricted risk estimate exceeds Australia's ALOP and, therefore, risk management is considered necessary.

Figure 3.6B **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for Disease B.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of release	Likelihood of release	Very Low
Likelihood of exposure	Likelihood of exposure	Extremely low
Likelihood of release and exposure	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	**Extremely low**
Consequence assessment		
Likelihood of establishment and/or spread	Likelihood of establishment and/or spread associated with the identified outbreak scenario	Low
Overall effect of establishment and/or spread	Outbreak scenario effects (health, environmental and socioeconomic) of establishment and/or spread assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	Low
Likely consequences	Estimated by combining the likelihood of establishment and/or spread (associated with the outbreak scenario) with the overall effect of establishment and/or spread using the matrix shown in Table 3.5 to obtain the likely consequences	**Very Low**
Risk estimation		
The risk of release, exposure, establishment and/or spread	Estimated by combining the likelihood of release and exposure with the likely consequences of establishment and/or spread using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of release, exposure, establishment and/or spread	**NEGLIGIBLE**

Table 3.7B **Worked example showing combined release, exposure and consequence assessments, resulting in an unrestricted risk.**

The unrestricted risk associated with Disease B is determined to be ‘negligible’. As the unrestricted risk estimate achieves Australia’s ALOP, no risk management is considered necessary.

3.3 Risk management

Risk management options considered in this report aim to reduce the likelihood that the imported horses would lead to the release, exposure, establishment and/or spread of disease agents of quarantine concern in Australia. Risk management options included measures relevant to reducing the likelihood of release and/or exposure to achieve Australia’s ALOP. They are described in detail in the chapters for those diseases where the unrestricted risk did not achieve Australia’s ALOP.

If risk management measures were required, then the restricted risk was then derived using a particular risk management measure or a combination of measures. If the restricted risk is ‘very low’ or ‘negligible’, that measure or combination of measures was considered acceptable.

In general, risk management can be implemented by reducing the likelihood of:

- disease agents being released into Australia in imported horses by imposing risk management measures, such as pre-entry measures and post-arrival quarantine, that reduce the likelihood of release
- exposure of susceptible animals in Australia by an imported horse by imposing risk management measures that reduce the likelihood of exposure.

If a disease agent is already present in Australia, Article 2.1.2 of the Code states that import measures are not to be more trade restrictive than those applied within the country.

References

Standards Australia (2005) 'Risk management guidelines: companion to AS/NZS 4360:2004.' (Standards Australia International and Standards New Zealand: Sydney)

4 Hazard identification

The list of potential disease agents (hazards) was compiled from

- diseases listed by the OIE (OIE 2009) as equine diseases or multiple species diseases affecting equids
- causative agents for other diseases identified as occurring in equids.

The method of hazard identification and refinement is described in chapter 3 (section 3.1). The preliminary list of disease agents/diseases is shown in Table 4.1.

Table 4.1 summarises the results of the hazard refinement process, including the reason for removal or retention of each identified hazard. Additional technical information that was required for some disease agents in order to complete the hazard refinement is summarised in the Appendix.

Routine examination and treatment for external parasites are recommended for international movement of horses prior to travel (IFHA 2002; IFHA 2008; Ellis and Watkins 2004). Inclusion of general risk management measures of thorough examination and treatment for external parasites was considered appropriate. The Expert Panel similarly considered that anthelmintic treatment was an appropriate risk management measure for internal parasites. Australia therefore requires such measures for those parasites included in the hazard identification list (Table 4.1) – a risk assessment has not been conducted for every parasite. Parasite resistance to treatments was not considered in the IRA.

There are many potential disease agents of equids that are common commensals and may be present in Australia. There are others that are opportunistic, not reported to be pathogenic, or of uncertain relevance in equids due to limited or insufficient information. It is appropriate to list these agents here, not only to indicate that they were considered, but also in the event that evidence of disease is reported subsequent to the finalisation of this IRA.

Viruses: Akabane virus; Bunyaviridae – Californian group including Cache Valley, Jamestown Canyon, Main Drain and snowshoe hare viruses; equine papillomaviruses, equine reovirus, equine rotavirus, foamy viruses (spumaviruses), Kokobera virus, Kunjin virus, Molluscum contagiosum, Murray Valley encephalitis virus, Near Eastern equine encephalomyelitis virus, Nigerian encephalitis, Peruvian horse sickness, Powassan virus, Ross River virus, Salem virus, St Louis virus and viral papular dermatitis.

Bacteria: *Actinobacillus* spp., *Actinobaculum* spp., *Actinomyces* spp., *Arcanobacterium* spp., *Bacillus* spp., *Bacteroides* spp., *Clostridium* spp., *Corynebacterium* spp., *Dermatophilus* spp., *Escherichia coli*, *Francisella* spp., *Fusobacterium* spp., *Listeria monocytogenes*, *Mycobacterium* spp., *Mycoplasma* spp., *Nocardia* spp., *Pasturella* spp., *Pseudomonas* spp., *Rhodococcus equi*, *Salmonella* spp. and *Streptococcus* spp.

Helminths: *Anoplocephala* spp., Cyathostominae, *Dictyocaulis arnfeldi*, *Fasciola* spp., *Gasterophilus* spp., *Habronema* spp., *Oesophagodontus* spp., *Onchocerca* spp., *Oxyuris equi*, *Parascaris equorum*, *Pelodera strongyloides*,

Setaria spp., Strongylinae, *Strongyloides westeri, Thelazia* spp., *Trichophyton* spp. and *Triodontophorus* spp.

Protozoa: *Cryptosporidium parvum, Eimeria* spp., *Giardia* spp., *Isospora* spp., *Neospora caninum* and *Sarcocystis bertrami*.

Arthropods: *Chorioptes equi, Damalinia equi, Demodex equi, Gasterophilus* spp., *Haematopinus asini, Psoroptes* spp. and *Trombicula* spp.

Algae and fungi: *Absidia corymbifera, Aspergillus* spp., *Basidiobolus* spp., *Blastomyces dermatitidis, Brachycladium spiciferum, Candida* spp., *Coccidioides immitis, Conidiobolus* spp., *Cryptococcus neoformans, Curvularia geniculata, Helminthosporium spiciferum, Histoplasma* spp., *Malassezia* spp., *Microsporum* spp., *Monosporium apiospermum, Pithyium insidiosum, Rhinosporidium seeberi, Sporotrichum schenckii* and *Trichophyton* spp.

Chlamydia: *Chlamydophila* spp.

Table 4.1 Hazard identification and refinement.

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
OIE-LISTED DISEASES						
African horse sickness	All equids, exceptionally other species	Yes	*Australia*: absent	No	No	Retained: OIE-listed
Anthrax (*Bacillus anthracis*)	All mammals	Yes	*Australia*: present; control measures in place	Yes	Yes	Retained: present in Australia, control measures in place
Aujeszky's disease (Suid herpesvirus1)	Pigs, ruminants, dogs, rats and occasionally horses	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Bovine tuberculosis (*Mycobacterium bovis*)	Bovids, equids, other mammals	Yes	*Australia:* absent	Yes	Yes	Retained: not present in Australia
Brucellosis (*Brucella abortus*)	Bovids, occasionally horses	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Brucellosis (*Brucella suis*)	Pigs, rarely horses	Yes	*Australia*: present; control measures in place	Yes	Yes	Retained: present in Australia; control measures in place (considered with *Brucella abortus*)
Contagious equine metritis (*Taylorella equigenitalis*)	Equids	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Dourine (*Trypanosoma equiperdum*)	Equids	Yes	*Australia*: absent	No	No	Retained: OIE-listed
Eastern equine encephalomyelitis	Birds, equids, humans, pigs, other animals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Echinococcosis (*Echinococcus granulosus, E. multilocularis*)	Horses (intermediate host), carnivores (definitive host)	Yes	*Australia*: *E. granulosus* present; other species absent	Yes	Yes	Retained: species not present in Australia

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Equine infectious anaemia	Equids	Yes	*Australia*: present in limited areas; notifiable	Yes	Yes	Retained: OIE-listed;notifiable in Australia
Equine influenza	Equids	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Equine piroplasmosis (*Babesia caballi, Theileria equi*)	Equids	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Equine rhinopneumonitis (Equid herpesvirus 1 and 4)	Equids	Yes	*Australia*: strains present	Yes	Yes	Retained: abortigenic and neurological strains are notifiable in Australia
Equine viral arteritis	Equids	Yes	*Australia*: strains present; notifiable	Yes	Yes	Retained: strains not present in Australia
Glanders (*Burkholderia mallei*)	Equids, other mammals including humans	Yes	*Australia*: absent	No	No	Retained: OIE-listed
Japanese encephalitis	Some mammals, including equids; birds, reptiles	Yes	*Australia*: absent from mainland Australia	Yes	Yes	Retained: not present in mainland Australia
Leptospirosis (*Leptospira* spp.)	Vertebrates	Yes	*Australia:* multiple serovars present	Yes	Yes	Retained: serovars not present in Australia.
Nagana (*Trypanosoma brucei, T. congolense, T. vivax*)	Bovids, other livestock, equids, humans	Yes	*Australia*: absent	No	No	Removed: OIE list specifies tsetse transmitted trypanosomosis and therefore limited to Africa (except *T.* vivax); not present in approved countries (see Appendix)
New World screwworm (*Cochliomyia hominivorax*)	Mammals	Yes	*Australia*: absent	No	No	Retained: OIE-listed (considered with Old World screwworm)
Nipah virus	Pigs, dogs, cats humans, horses, bats	Yes	*Australia*: absent	No	No	Retained: OIE-listed

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Old World screwworm (*Chrysomya bezziana*)	Mammals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Q fever (*Coxiella burnetii*)	Multiple species	Yes	*Australia*: present	Yes	No	Removed: present in Australia, no official control
Rabies	Mammals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Rift Valley fever	Ruminants, horses, pigs, wildlife, humans	Yes	*Australia*: absent	No	No	Retained: OIE-listed
Surra (*Trypanosoma evansi*)	Some mammals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Trichinellosis (*Trichinella spiralis*)	Mammals, esp. carnivores	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Venezuelan equine encephalomyelitis	Birds, equids, humans, other animals	Yes	*Australia*: absent	No	No	Retained: OIE-listed
Vesicular stomatitis	Equids, bovids, pigs, humans	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Western equine encephalomyelitis	Birds, equids, humans, other animals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
West Nile fever	Birds, equids, humans, other animals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
DISEASES NOT LISTED BY OIE						
Viruses						
Borna disease	Horses, cats, cattle, sheep, rabbits, ostriches, humans	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Equid herpesvirus 2, 3, 5-9	Equids	Yes	*Australia*: some viruses present	Yes	Yes	Retained: viruses not present in Australia (considered with equine rhinopneumonitis, EHV 1,4)
Equine adenovirus 1 and 2	Horses	Yes	*Australia*: present	Yes	No	Removed: present in Australia
Equine coronavirus	Horses	Yes	*Australia*: present	Yes	No	Removed: present in Australia (see Appendix)
Equine encephalosis	Equids	Yes	*Australia*: absent	No	No	Retained: similar distribution to African horse sickness
Equine enterovirus	Horses	No[10]	*Australia:* not reported	Yes	No	Removed: not likely to produce adverse effects; possible worldwide occurrence
Equine parainfluenza virus	Horses	No	*Australia*: absent	No	No	Removed: doubtful significance (see Appendix)
Equine rhinitis A virus (formerly equine rhinovirus 1)	Horses, camels	Yes	*Australia*: present	Yes	No	Removed: present in Australia (see Appendix)
Equine rhinitis B virus (formerly equine rhinovirus 2 or 3)	Horses	Yes	*Australia*: present	Yes	No	Removed: present in Australia (see Appendix)

[10] Single isolation from oral cavity of clinically healthy horse in 1983 (Studdert 1996).

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Equine torovirus (Berne virus)	Horses	No	*Australia*: not reported	Yes	No	Removed: not likely to produce adverse effects; possible worldwide occurrence (see Appendix)
Getah virus	Horses, pigs	Yes	*Australia*: absent	Yes	No	Removed: Not transmitted by horses (see Appendix)
Hendra virus	Bats, humans, horses	Yes	*Australia*: present	No	No	Removed: present in Australia
Horse pox	Equids	Yes	*Australia*: absent	No[11]	Yes	Retained: Code recommendations
Louping ill virus	Sheep, horses, other animals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Bacteria						
Equine paratyphoid (*Salmonella* Abortusequi)	Equids	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Melioidosis (*Burkholderia pseudomallei*)	Mammals	Yes	*Australia*: present	Yes	No	Removed: present in Australia
Proliferative enteropathy (*Lawsonia intracellularis*)	Horses, pigs, other mammals	Yes	*Australia*: present	Yes	No	Removed: present in Australia
Taylorella asinigenitalis	Equids	Yes	Australia: absent	Yes	Yes	Retained: not present in Australia

[11] Historical references to horse pox exist but there are no recent reports of this infection worldwide; not OIE-listed; Code chapter.

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Rickettsias						
Equine granulocytic anaplasmosis (formerly equine ehrlichiosis) (*Anaplasma phagocytophilum*) (formerly *Ehrlicha equi*)	Ruminants, horses, dogs	Yes	*Australia*: not reported	Yes	Yes	Retained: not present in Australia
Lyme disease (*Borrelia burgdorferi*)	Humans, wild animals, other mammals	Yes (human)	*Australia*: not isolated	Yes	Yes	Retained: not present in Australia
Spirochaetosis (*Borrelia theileri)*	Cattle, horses, other ruminants	No	*Australia*: present	Yes	No	Removed: present in Australia
Potomac horse fever (*Neorickettsia risticii*) (formerly *Ehrlichia risticii*)	Horses, possibly other animals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Fungi						
Epizootic lymphangitis (*Histoplasma farciminosum*)	Equids, other mammals	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Protozoa						
Besnoitiosis (*Besnoitia bennetti*)	Equids	Yes	*Australia*: absent	Yes	No	Removed: not transmitted by live horses (see Appendix)
Equine protozoal myeloencephalitis (*Sarcocystis neurona*)	American opossum, horses	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia
Arthropods: Insecta						
Nasal bot (*Rhinoestrus purpureus*)	Equids	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated and inspected for ectoparasites

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Warble-fly (*Hypoderma bovis, H. lineata*)	Cattle, rarely equids, humans	Yes	*Australia*: absent	Yes	No	Removed: Horses are dead-end host (see Appendix)
Arthropods: Arachnida mites						
Horse mange (*Sarcoptes scabei* var *equi*)	Equids, other mammals	Yes	*Australia*: absent[12]	Yes	Yes	All imported horses to be treated and inspected for ectoparasites[13]
Psoroptic mange (*Psoroptes equi*)	Equids	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated and inspected for ectoparasites
Arthropods: Arachnida ticks						
Amblyomma spp., *Ornithodorus* spp.	Mammals, reptiles birds	Yes	*Australia*: some species present	Yes	Yes	All imported horses to be treated and inspected for ectoparasites
Hyalomma spp.	Cattle, horses and other mammals	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated and inspected for ectoparasites
Ixodes spp. not present in Australia	Humans, dogs equids, others	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated and inspected for ectoparasites
Ear tick (*Otobius megnini*)	Dogs, sheep, equids, cattle, others	Yes	*Australia*: present	Yes	No	Removed: present in Australia

[12] *Sarcoptes scabei* affects other species in Australia; evidence for host specificity is equivocal.
[13] Internationally accepted to treat and inspect horses for ectoparasites.

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Rhipicephalus spp. not present in Australia	Cattle, horses, dogs, other mammals	Yes	*Australia*: absent	Yes Some species	Yes	All imported horses to be treated and inspected for ectoparasites
Helminths: nematodes						
Stomach tumour worm (*Draschia megastoma*)	Equids	Yes	*Australia*: present	Yes	No	Removed: present in Australia
Arterial worm (*Elaeophora boehmi*)	Horses	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated for endoparasites[14]
Gullet worm (*Gongylonema pulchrum*)	Equids, cattle, sheep, humans, other mammals	Yes	*Australia*: present	Yes	No	Removed: present in Australia
Bloody sweat worm (*Parafilaria multipapillosa*)	Equids	Yes	*Australia*: absent	No	No	Removed: not reported in approved countries
Rhabditis gingivalis	Equids	Yes	*Australia*: absent	Yes	Yes	Saprophitic. All imported horses to be treated for endoparasites
Peritoneal worm (*Setaria equina*)	Equids	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated for endoparasites
Eye worm (*Thelazia lacrymalis*)	Horse, cattle, buffalo, camel, dog	Yes	*Australia*: absent	Yes	Yes	All imported horses to be treated for endoparasites

[14] Internationally accepted to treat horses for endoparasites.

Disease (disease agent)	Susceptible species	Adverse consequences in Australia?	Occurrence in Australia	Present in approved countries?	Potential hazard?	Reasons for removal/retention
Helminths: cestodes						
Echinococcosus (*Echinococcus equinus*)	Equids	Yes	*Australia*: absent	Yes	Yes	Retained: not present in Australia (considered with other *Echinococcus* spp.)
Helminths: trematodes						
Giant liver fluke (*Fasciola gigantica*)	Sheep, goat, cattle horses, others	Yes	*Australia:* absent	Yes	Yes	Retained: not present in Australia
Liver fluke (*Fasciola hepatica*)	Sheep, goat, cattle horses, others	Yes	*Australia*: present	Yes	No	Retained: considered with *Fasciola gigantica*
Stomach fluke (*Gastrodiscus aegyptaeicus*)	Equids, pig, warthog	Yes	*Australia:* absent	No	Yes	Removed: not reported in approved countries (see Appendix)
Schistosomiasis (*Schistosoma indicum, S. Intercalatum, S. Japonicum, S. mattheei, S. nasale, S. spindale)*	Equids, ruminants, rodents	Yes	*Australia:* absent	Yes	Yes	Retained: not present in Australia

Conclusion

The following diseases were retained for risk assessment (chapter 5) on the basis of information provided in Table 4.1.

OIE-listed diseases

- African horse sickness
- anthrax (*Bacillus anthracis*)
- Aujeszky's disease (Suid herpesvirus 1)
- bovine tuberculosis (*Mycobacterium bovis*)
- brucellosis (*Brucella abortus, B. suis*)
- contagious equine metritis (*Taylorella equigenitalis*)
- dourine (*Trypanosoma equiperdum*)
- Eastern equine encephalomyelitis
- echinococcosis (*Echinococcus granulosus, E. multilocularis*)
- equine infectious anaemia
- equine influenza
- equine piroplasmosis (*Babesia caballi, Theileria equi*)
- equine rhinopneumonitis (Equid herpesvirus 1 and 4)
- equine viral arteritis
- glanders (*Burkholderia mallei*)
- Japanese encephalitis
- leptospirosis
- New World screwworm (*Cochliomyia hominivorax*)
- Nipah virus
- Old World screwworm (*Chrysomya bezziana*)
- rabies
- Rift Valley fever
- surra (*Trypanosoma evansi*)
- trichinellosis (*Trichinella spiralis*)
- Venezuelan equine encephalomyelitis
- vesicular stomatitis

- Western equine encephalomyelitis
- West Nile fever

Other diseases

Viruses

- Borna disease
- equid herpesvirus 2, 3, 5–9
- equine encephalosis
- horse pox
- louping ill

Bacteria

- equine paratyphoid (*Salmonella* Abortusequi)
- *Taylorella asinigenitalis*

Rickettsias

- equine granulocytic anaplasmosis (formerly equine ehrlichiosis) (*Anaplasma phagocytophilum,* formerly *Ehrlicha equi*)
- Lyme disease (*Borrelia burgdorferi*)
- Potomac horse fever (formerly equine ehrlichiosis) (*Neorickettsia risticii,* formerly *Ehrlichia risticii*)

Fungi

- epizootic lymphangitis

Protozoa

- equine protozoal myeloencephalitis (*Sarcocystis neurona*)

Cestodes

- echinococcosis (*Echinococcus equinus*)

Trematodes

- fascioliasis (*Fasciola gigantica*, *F. hepatica*)
- schistosomiasis (*Schistosoma indicum*, *S. intercalatum*, *S. japonicum*, *S. mattheei*, *S. nasale* and *S. spindale*)

References

Ellis P, Watkins KL (2004) International movement of athletic horses - quarantine and regulatory controls. In *Equine sports medicine and surgery* (eds. Hinchcliff KW, Geor RJ, Kaneps AJ) pp. 1227-1238. Elsevier, Amsterdam.

IFHA (2002) Guidelines to facilitate the temporary movement of registered racehorses for international races by the Permanent Liaison Committee on the International Movement of Horses. The International Federation of Horseracing Authorities, Boulogne.

IFHA (2008) *International agreement on breeding, racing and wagering*. The International Federation of Horseracing Authorities, Boulogne.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Studdert MJ (1996) Picornaviridae. In *Virus infections of equines* (ed. Studdert MJ) pp. 211-212. Elsevier Science Publishers, Amsterdam.

5 Risk assessments

5.1 African horse sickness

5.1.1 Technical information

Background

African horse sickness (AHS) is caused by a virus belonging to the Orbivirus genus of the family Reoviridae (Mertens et al. 2005). Infection results in high mortality in domestic horses and mules, and less severe disease in donkeys. Zebras are recognised as the natural reservoir host (Mellor and Hamblin 2004).

AHS is predominantly a disease of Africa and the nine serotypes have varying temporal and spatial occurrences (Mellor and Boorman 1995; Calisher and Mertens 1998). AHS is endemic to sub-Saharan Africa and probably Yemen (Sailleau et al. 2000), although there have been outbreaks in northern Africa, the Iberian Peninsula, the Indian Subcontinent and the Middle East (Mellor and Hamblin 2004). AHS has never been reported in Australia and has not been reported in any of the approved countries for at least the past ten years.

AHS is an OIE-listed disease (OIE 2009b).

Epidemiology

AHS virus is transmitted by biting arthropods. Species of the genus *Culicoides* are the principal vectors (Mellor and Hamblin 2004). In Australia, several species of *Culicoides* are vectors of bluetongue virus and are potential vectors for AHS virus.

AHS can clinically affect dogs (van Rensburg et al. 1981) and has been reported to subclinically infect camels (Wernery and Kaaden 2002). However, non-equids are not considered to be involved in the maintenance and spread of AHS virus (Mellor and Hamblin 2004).

Clinical signs

In experimental cases, the incubation period is usually 5–7 days, but can be as short as two days and as long as ten days. The duration of the incubation period depends on the virulence of the virus and the dose of the virus received (Guthrie 2007). Four clinical syndromes have been described, according to the range and severity of clinical signs (Brown and Mebus 1992). These include the ‘pulmonary’ (or ‘dunkop’) form, the ‘cardiac’ (or ‘Dikkop’) form, the ‘mixed’ form (has features of both pulmonary and cardiac forms) and finally, a mild form referred to as horse sickness fever.

Mortality in horses can be as high as 95% in susceptible populations (Coetzer and Guthrie 2004). Mules generally develop a milder form of the disease and donkeys can be subclinically infected. Zebras generally do not show clinical signs of disease.

There is no specific treatment for AHS.

Diagnosis

Diagnosis of AHS is virtually impossible during the early pyrexic phase of the disease. A presumptive diagnosis should be possible once the characteristic clinical signs develop. Typical macroscopic lesions on post mortem are usually sufficient to allow a provisional diagnosis of AHS. AHS can be definitively diagnosed by isolating the virus or detecting its nucleic acids or antigens (Guthrie 2007).

AHS virus can be isolated, by intracerebral inoculation of mice, or in cell cultures. Virus isolation in mice is the preferred technique for primary isolation (OIE 2008). The isolated virus can be identified by complement fixation or immunofluorescence. The isolate should be serotyped using virus neutralisation or other methods.

AHS viral antigens can be detected with enzyme-linked immunosorbent assays (ELISAs). A reverse-transcription polymerase chain reaction technique is used to detect viral RNA (Sailleau et al. 1997).

Serology can also be used to diagnose AHS. Antibodies can be detected within 8–14 days after infection, and may persist for 1–4 years. Available serologic tests include complement fixation, ELISAs, immunoblotting and virus neutralisation (Hamblin et al. 1990; Hamblin et al. 1992). The indirect ELISA and complement fixation tests are the prescribed tests for international trade (OIE 2008). The virus neutralisation test is used for serotyping. Immunodiffusion and haemagglutination inhibition tests have also been described (Guthrie 2007).

Conclusion

AHS is not present in any approved country. While this remains the case, certification of country freedom, in accordance with the Code recommendations (OIE 2009a), will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Brown CC, Mebus CA (1992) The pathology of African horse sickness. In *Proceedings of the second symposium on bluetongue, African horse sickness and related orbiviruses, June 17-21, 1991, Paris, France,* (eds. Walton TE, Osburn BI) pp. 832-834, CRC Press, Boca Raton.

Calisher CH, Mertens PPC (1998) Taxonomy of African horse sickness viruses. *Archives of Virology* 14: 3-11.

Coetzer JAW, Guthrie AJ (2004) African horse sickness. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 1231-1246. Oxford University Press, Oxford.

Guthrie AJ (2007) African horse sickness. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 164-171. Saunders Elsevier, St. Louis.

Hamblin C, Anderson EC, Mellor PS, Graham SD, Mertens PPC, Burroughs JN (1992) The detection of African horse sickness virus antigens and antibodies in young Equidae. *Epidemiology and Infection* 108: 193-201.

Hamblin C, Graham SD, Anderson EC, Crowther JR (1990) A competitive ELISA for the detection of group-specific antibodies to African horse sickness virus. *Epidemiology and Infection* 104: 303-312.

Mellor PS, Boorman J (1995) The transmission and geographical spread of African horse sickness and bluetongue viruses. *Annals of Tropical Medicine and Parasitology* 89: 1-15.

Mellor PS, Hamblin C (2004) African horse sickness. *Veterinary Research* 35: 445-466.

Mertens PPC, Maan S, Samuel A, Attoui H (2005) Orbivirus. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 466-483. Elsevier, San Diego.

OIE (2008) African horse sickness. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.01_AHS.pdf (Accessed 1 September 2009).

OIE (2009a) African horse sickness. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/en_chapitre_1.12.1.pdf (Accessed 19 August 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Sailleau C, Hamblin C, Paweska JT, Zientara S (2000) Identification and differentiation of the nine African horse sickness virus serotypes by RT-PCR amplification of the sertype-specific genome segment 2. *Journal of General Virology* 81: 831-837.

Sailleau C, Moulay S, Cruciere C, Laegreid WW, Zientara S (1997) Detection of African horse sickness virus in the blood of experimentally infected horses: comparison of virus isolation and a PCR assay. *Research in Veterinary Science* 62: 229-232.

van Rensburg IBJ, de Clerk J, Groenewald HB, Botha WS (1981) An outbreak of African horsesickness in dogs. *Journal of the South African Veterinary Association* 52: 323-325.

Wernery U, Kaaden O-R (2002) Nonpathogenic viral infections: African horse sickness. In *Infectious diseases in camelids* pp. 212-214. Blackwell Wissenschafts-Verlag, Berlin.

5.2 Anthrax

5.2.1 Technical information

Background

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

Domesticated and wild ruminants are most susceptible, horses less susceptible and omnivores and carnivores relatively resistant. Although *B. anthracis* occurs worldwide, outbreaks are most common in parts of Africa, Asia and the Middle East, with sporadic cases in Australia, Europe and the United States (CFSPH 2007; OIE 2008).

Over the past 150 years, outbreaks in Australia have been recorded in Gippsland and the 'anthrax belt' — which extends from northern Victoria to the central pastoral areas of New South Wales. In January 2008, an outbreak occurred outside the anthrax belt in New South Wales, in an area that had not had a case of anthrax since the early 1900s. Prevailing climatic conditions and soil disturbance may have allowed cattle to be exposed to anthrax spores buried in the soil (DAFF 2008).

Anthrax is a notifiable disease in Australia and control measures include vaccination, premises quarantine, movement controls and surveillance (Animal Health Australia 2005).

Anthrax is a multiple species OIE-listed disease (OIE 2009b).

Epidemiology

B. anthracis is thought to multiply almost exclusively inside the body and exists in the environment as dormant spores. Spores are not found in host tissues unless they are exposed to air. Bacteria are present in the carcass and body discharges; however, spores from carcasses are the only source of infection in animals. Vegetative organisms are thought to be destroyed within a few days during the decomposition of unopened carcasses. Spores can remain viable in the soil or animal products for decades. Once the soil has been contaminated by spores, it is very difficult to decontaminate. Carnivores, rain and other agents can disperse the spores to other locations. Transmission occurs by ingestion or inhalation of spores in soil or on plants, although entry through skin lesions has not been ruled out. Contaminated bone meal and other feed can also spread anthrax, and flies can disseminate anthrax mechanically. Outbreaks are often associated with heavy rainfall, flooding, or drought (CFSPH 2007).

Clinical signs

The incubation period is generally 1–7 days, but spores can germinate in the lungs up to six weeks post-infection (CFSPH 2007). Clinical signs of disease include pyrexia, anorexia, depression, severe colic, dyspnoea and bloody diarrhoea. Swellings may be seen in the neck, sternum, lower abdomen and external genitalia. Affected animals

usually die within 1–3 days, but some animals can survive for up to a week. Rigor mortis is usually absent or incomplete, the carcass is typically bloated and dark, and tarry blood may ooze from the orifices. Decomposition is rapid and post-mortem examinations should be avoided to prevent human exposure and contamination of the environment with spores (de Vos and Turnbull 2004).

Diagnosis

The history, including clinical presentation, is the first step in the diagnosis of anthrax. Demonstration of *B. anthracis* in blood or tissue smears is confirmatory; however, their absence does not exclude the possibility of anthrax (de Vos and Turnbull 2004). Bacterial culture can be used for diagnosis and polymerase chain reaction can be used to identify *B. anthracis* and to detect bacterial toxin and capsule genes. Antibodies develop late in the course of disease, and serology is only useful in retrospective studies. A skin hypersensitivity test using AnthraxinT is widely used in some countries for the retrospective diagnosis of anthrax in animals and humans (CFSPH 2007). More recently, hand-held immunochromatographic assay kits have been evaluated and used in Australia to provide a rapid field diagnosis in livestock.

Conclusion

Anthrax is present in approved countries and in Australia. The disease is notifiable and control measures are in place in Australia. The Code recommendations (OIE 2009a) include premises freedom or vaccination and that anthrax is notifiable. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Animal Health Australia (2005) *Disease strategy: anthrax (version 3.2)*. Primary Industries Ministerial Council, Canberra.

CFSPH (2007) Anthrax. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 24 February 2009).

DAFF (2008) Anthrax. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/anthrax (Accessed 24 February 2009).

de Vos V, Turnbull PCB (2004) Anthrax. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 1788-1818. Oxford University Press, Oxford.

OIE (2008) Disease timelines: anthrax. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_timelines&disease_type=Terrestrial&disease_id=17&empty=999999 (Accessed 24 February 2009).

OIE (2009a) Anthrax. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.1.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

5.3 Aujeszky's disease

5.3.1 Technical information

Background

Aujeszky's disease (pseudorabies or 'mad itch') is predominately a disease of pigs but was first identified in cattle in the United States in 1813 (Kluge et al. 1999) Aujeszky's disease is caused by suid herpesvirus 1 (SHV-1), a member of the alphavirus subfamily of the family Herpesviridae (Davison et al. 2005). Strains of varying pathogenicity have been reported; however, there is only one serotype of SHV-1.

Aujeszky's disease has had an almost worldwide distribution including North and South America, Europe and Asia. However, many countries, have either eradicated the disease, including several European countries, New Zealand and Singapore, or are in the process of doing so. The disease has never been reported in Australia.

Aujeszky's disease is a multiple species OIE-listed disease (OIE 2009) and the Code does not have recommendations for species other than pigs.

Epidemiology

Pigs are the primary host and reservoir of SHV-1 making them the principal source of infection and transmission of disease to other animals, such as cattle, sheep, goats, dogs, cats, rats and mice (Studdert 1996). Horses, birds and humans are considered resistant to SHV-1 infection (Kluge et al. 1999). On rare occasions horses have become infected with SHV-1, but only when housed in close proximity to infected pigs (Kimman et al. 1991).

Aujeszky's disease in species other than pigs is only reported to occur when the disease is endemic in the pig population (Vandevelde 2006).

Clinical signs

Horses have been shown to be susceptible to experimental infection with high doses of SHV-1 but rarely become infected under natural conditions. In experimentally infected ponies the incubation period was 7–8 days (Kimman et al. 1991). The incubation period for natural infection in horses is unknown. Infected horses show depression, which may lead to excitation, sweating, muscular tremors, mania and death.

Conclusion

Aujeszky's disease is present in pigs in approved countries but horses are very unlikely to become infected with, or to transmit SHV-1.

Aujeszky's disease was not considered further in the IRA.

References

Davison AJ, Eberle R, Haywood GS, McGeoch DJ, Mihnson AC, Pellett PE, Roizman B, Studdert MJ, Thiry E (2005) Herpesviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 193-212. Elsevier, San Diego.

Kimman TG, Binkhorst GJ, van den Ingh TS, Pol JM, Gielkens AL, Roelvink ME (1991) Aujeszky's disease in horses fulfils Koch's postulates. *The Veterinary Record* 128: 103-106.

Kluge JP, Beran GW, Hill HT, Platt KB (1999) Pseudorabies (Aujeszky's disease). In *Diseases of swine* (eds. Straw BE, D'Allaire S, Mengeling WL, Taylor DJ) pp. 233-246. Blackwell Science, Oxford.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Studdert MJ (1996) Pseudorabies (Aujeszky's disease). In *Virus infections of equines* (ed. Studdert MJ) pp. 61-63. Elsevier, Amsterdam.

Vandevelde M (2006) Pseudorabies. In *Infectious diseases of the dog and cat* (ed. Greene CE) pp. 183-186. Saunders Elsevier, St. Louis.

5.4 Borna Disease

5.4.1 Technical information

Background

Borna disease, an infectious encephalomyelitis, is caused by a virus belonging to the genus Bornavirus in the family, Bornaviridae (Schwemmle et al. 2005). Natural infection with Borna disease virus (BDV) mainly affects horses and sheep and may be associated with neurological disease in cats (Reeves et al. 1998). Other species found to be serologically positive to BDV include dogs, cattle, new world camelids, ostriches and rabbits (Ludwig and Bode 2000; Kamhieh et al. 2008). Many other species of mammals are susceptible to experimental infection with BDV, but the virus' ability to infect humans remains unclear (Lipkin and Briese 2007).

BDV is thought to occur in many parts of the world; however, the exact geographical distribution is unknown (Ludwig and Bode 2000). The disease is endemic in horses and sheep in certain parts of Europe (Austria, Germany and Switzerland). BDV-specific antibodies have been detected in an increasing number of countries although clinical disease has not been reported (Richt et al. 2000).

Australia is considered to be free from Borna disease (Geering et al. 1995; Kamhieh et al. 2006). While there is some serological evidence of exposure to BDV or Borna disease-like virus in Australia, there have been no confirmed clinical cases and the virus has never been isolated (Kamhieh et al. 2006; Kamhieh et al. 2008).

Borna disease is not an OIE-listed disease (OIE 2009).

Epidemiology

The mode of transmission and possible reservoir hosts of infection are unknown (Staeheli et al. 2000).

Borna disease recurs in specific areas or individual farms during spring and summer, a phenomenon that remains unexplained.

The disease occurs sporadically in sheep and horses, with only one or a few horses in a stable affected. In sheep, up to 12% of a flock can be affected (Lipkin and Briese 2007). Morbidity rates of only 0.006–0.23% have been reported in horses from endemic areas of Germany, but the disease is usually fatal (Radostits et al. 2007).

In countries where clinical disease is not reported, 3–42% of horses have shown evidence of antibodies or nucleic acid (Radostits et al. 2007). In endemic areas in Europe, 12–20 % of horses show serological evidence of exposure (Radostits et al. 2007). However, clinically affected animals may have very low or undetectable levels of antibody (Radostits et al. 2007).

Clinical Signs

The incubation period of Borna disease is estimated to be from one to six months (Radostits et al. 2007). Natural BDV infection can result in peracute, acute or subacute infection (Richt et al. 2000). Clinical signs of Borna disease in horses vary, and include pyrexia, pharyngeal paralysis, muscle tremor, propioceptive deficits and

hyperaesthesia (Radostits et al. 2007). Lethargy, somnolence and paralysis occur in terminal stages, sometimes accompanied by blindness. Death occurs one to three weeks after the appearance of clinical signs. Subclinical infection in horses may occur as viral antigen has been detected in clinically normal horses (Ludwig and Bode 2000; Richt et al. 2000). However, the reliability of viral antigen in confirming the presence of BDV or Borna disease may be questionable (Herzog et al. 2008). There is no effective treatment.

Diagnosis

Reliable ante mortem diagnosis of Borna disease is difficult and clinical signs of disease are not specific. BDV infection in live animals may be indicated by evidence of specific antibodies in serum or cerebrospinal fluid. The presence of viral nucleic acids in saliva, nasal or conjunctival fluid may confirm the diagnosis. However, antibodies or viral nucleic acids may not be present in all cases of infection (Lipkin and Briese 2007). Data also suggest that detection of specific antibodies or viral nucleic acids do not always support a diagnosis of Borna disease (Herzog et al. 2008).

A definitive diagnosis in dead animals is based on neuropathological examinations which show distinctive intranuclear antigen, the presence of viral nucleic acid in brain tissue or the isolation of virus (Lipkin and Briese 2007).

The Dresden strain used to vaccinate horses and sheep against Borna disease was discontinued in 1992 due to concerns over post-vaccinal shedding of virus (Lipkin and Briese 2007) and lack of efficacy (Radostits et al. 2007). There are currently no vaccines available for use in horses.

Conclusion

Borna disease is present in some approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.4.2 Risk assessment

For details of the method used in this risk assessment, see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of Borna disease being present in an imported horse:

- Borna disease occurs sporadically and seasonally in limited regions of Europe although serological evidence suggests that subclinical infection is widespread (Ludwig and Bode 2000).
- The incubation period of Borna disease is estimated to be from one to six months (Radostits et al. 2007).
- Subclinical infections with BDV may occur, although this is difficult to confirm as infectious virus has not been isolated from healthy animals.
- Horses affected by Borna disease can show severe clinical signs (Richt et al. 2000).

- Studies suggest that there is viral persistence without apparent disease in naturally infected horses (Ludwig and Bode 2000), although this is not well substantiated.
- The epidemiology of BDV in countries where the disease occurs is not well understood and a conservative approach is warranted.

Based on these considerations, the likelihood of release of Borna disease associated with horses from a country where the disease is present is estimated to be '*very low*'.

Exposure assessment

The mode of transmission of Borna disease from horses is not clearly understood. It has been suggested that the disease may only be spread via reservoir hosts (Staeheli et al. 2000). However, direct and indirect contact are considered the most likely exposure pathways (Richt et al. 2000).

The exposure groups would include equids (including feral equids) and other domestic species (primarily sheep as they are considered more susceptible than other species).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to Borna disease via an imported horse:

- Contact with body secretions of infected horses could result in exposure of susceptible animals (Richt et al. 1997).
- Neither reservoirs nor modes for transmission of natural infection are known.
- Spread of BDV may be reliant on suitable reservoir hosts (Staeheli et al. 2000). It is unknown if Australia would have suitable reservoir hosts.
- Borna disease is more common in stables with poor hygiene (Staeheli et al. 2000).

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with BDV was estimated to be *'extremely low'*.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be *'very low'* combined with the likelihood of exposure estimated to be *'extremely low'*, the likelihood of release and exposure for Borna disease was estimated to be ***'extremely low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak

scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to Borna disease is considered to be limited further establishment and/or spread to populations of susceptible animals through direct contact with infected horses or fomites.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to Borna disease.

- The disease has remained confined to limited endemic regions despite widespread serological evidence of potential exposure to virus around the world.
- There is no direct evidence of transmission from horses to any other animals or to humans (Staeheli et al. 2000).
- The absence of species-specific mutations in BDV strains in horses and sheep or other livestock suggests a common source of virus in an as yet unknown animal reservoir (Staeheli et al. 2000).

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of Borna disease was estimated to be '*very low*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) effect criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of Borna disease for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Borna disease is responsible for loss of productivity in sheep due to increased culling and mortality.
- Morbidity in susceptible species may be low but most affected animals will die or be euthanased.
- Borna disease may be a zoonosis and has been linked to psychiatric disorders in humans (Ludwig and Bode 2000).

Based on these considerations, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- In areas where Borna disease is endemic, it is not known if vertebrate wildlife have serological evidence of infection with the virus. Clinical signs of disease are not reported. It is not known if Australian native fauna and insects are susceptible to infection with the virus, and it is considered that clinical disease is unlikely to be discernible in wildlife.

Based on this consideration, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Borna disease is nationally notifiable in Australia (DAFF 2008).
- There is no AUSVETPLAN Disease Strategy Manual for Borna disease; however, the disease is scheduled as Category 4 under Australia's Emergency Animal Disease Response Agreement (EADRA) for cost-sharing arrangements (Animal Health Australia 2001). Should it be activated, EADRA states that costs of the response would be covered by government and relevant industries by contributions of 20% and 80%, respectively (Animal Health Australia 2001). However, currently the horse industry is not a signatory to this Agreement. Other animal industries, such as those associated with sheep, are signatories to the agreement.
- If Borna disease were to be identified in Australia, a combination of strategies would be employed, including slaughter and disposal of clinically affected animals, quarantine and movement controls, tracing and surveillance, vector control, decontamination, epidemiological investigations, and a public awareness campaign.
- In this outbreak scenario where Borna disease has only limited spread, eradication of the disease would be possible. However, if the disease were to become established in vertebrate and/or invertebrate reservoir hosts, periodic outbreaks could occur.

Based on these considerations, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- Borna disease is a nationally notifiable disease in Australia and if it was detected in any state, movement restrictions would be imposed and other states/territories may close their borders to all susceptible animals.

- As a result of the detection of Borna disease, movement restrictions would be imposed on all susceptible species and other potentially infected fomites.
- Movements of animals to sale and slaughter would be affected. Clinically affected sheep would not be accepted for slaughter for human consumption. Horse racing and other equestrian events may be prohibited.
- Following detection of Borna disease in one state or territory of Australia, other states may close their borders to all susceptible animals and products until the extent of the outbreak was ascertained.
- Public health perceptions and market fluctuations may reduce the value of the sheep industry.
- Supporting industries such as stockfeed manufacturers, veterinarians and farriers could also be affected.

Based on these considerations, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- The effects on international trade of a confirmed outbreak of Borna disease in Australia would result in national disruption to exports of live animals, including horses and sheep and possibly markets for meat.
- If eradication were delayed, possibly because of establishment in feral or wild animals, the effect on live animal trade may be prolonged. Zoning to assist in the international marketing of these animals would need to be adopted.
- If Borna disease were to become established, recurrent outbreaks would result in periodic disruption to international trade.

Based on these considerations, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Borna disease is not considered to lead to any indirect effects on the environment.

Based on this consideration, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Disruption of horse events would have social consequences for people involved in horse events.

- Slaughter of clinically affected animals would have emotional effects for people in communities.
- Public concerns of a potential zoonotic disease might have a detrimental effect on tourism in affected rural and regional communities.
- Where susceptible species were important to the local economy, the economic viability of communities within affected regions may be threatened due to loss of associated industries.

Based on these considerations, the effect of the establishment and/or spread of Borna disease in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is D, the overall effect associated with the outbreak scenario is considered to be *'low'*.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread (*'very low'*) is combined with the estimate of the overall effect of establishment and/or spread (*'low'*) which results in ***'negligible'*** likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of Borna disease introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure (*'extremely low'*) is combined with the likely consequences of establishment and/or spread (*'negligible*), resulting in a risk estimation of **NEGLIGIBLE**.

Conclusion

The unrestricted risk associated with Borna disease is determined to be **NEGLIGIBLE**. As the unrestricted risk estimate achieves Australia's ALOP, no specific risk management is considered necessary.

As described in section 6.1.1, horses should not be sourced from premises where an emerging disease agent of potential quarantine concern is known to occur. This would apply to premises where clinical cases of Borna disease have occurred.

A summary of the risk assessment for Borna disease is shown in Figure 5.1 and Table 5.1.

Figure 5.1 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for Borna disease.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Very low*
Likelihood of *exposure*	Likelihood of *exposure*	*Extremely low*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	***Extremely low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Very low*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Low*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Negligible***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**NEGLIGIBLE**

Table 5.1 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for Borna disease.

References

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

Geering WA, Forman AJ, Nunn MJ (1995) Borna disease. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 71-75. Australian Government Publishing Service, Canberra.

Herzog S, Herden C, Frese K, Lange-Herbst H, Grabner A (2008) Borna disease virus infection of horses: contradictory results between antemortem and postmortem investigations. *Pferdeheilkunde* 24: 766-774.

Kamhieh S, Hodgson J, Bode L, Ludwig H, Ward C, Flower RLP (2006) No evidence of endemic Borna disease virus infection in Australian horses in contrast with endemic infection in other continents. *Archives of Virology* 151: 709-719.

Kamhieh S, Hodgson JI, Bode L, Ludwig H, Flower RLP (2008) Borna disease virus: evidence of naturally-occurring infection in cats in Australia. *Acta Pathologica, Microbiologica et Immunologica Scandinavica* 116: 50-52.

Lipkin WI, Briese T (2007) Bornaviridae. In *Fields virology* (eds. Knipe DM, Howley PM) pp. 1829-1851. Lippincott Williams & Wilkins, Philadelphia.

Ludwig H, Bode L (2000) Borna disease virus: new aspects on infection, disease, diagnosis and epidemiology. *Revue Scientifique et Technique de l'Office International des Epizooties* 19: 259-288.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

Reeves NA, Helps CR, Gunn-Moore DA, Blundell C, Finnemore PL, Pearson GR, Harbour DA (1998) Natural borna disease virus infection in cats in the United Kingdom. *The Veterinary Record* 143: 523-526.

Richt JA, Grabner A, Herzog S (2000) Borna disease in horses. *Veterinary Clinics of North America: Equine Practice* 16: 579-595.

Richt JA, Pfeuffer I, Christ M, Frese K, Bechter K, Herzog S (1997) Borna disease virus infection in animals and humans. *Emerging Infectious Diseases* 3: 343-352.

Schwemmle M, Carbone KM, Tomonago K, Nowothny N, Garten W (2005) Family Bornaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on Taxonomy of Viruses* (eds. Fauquet M, Mayo A, Maniloff J, Desselberger U, Ball LA) pp. 615-622. Elsevier, San Diego.

Staeheli P, Sauder C, Hausmann J, Ehrensperfer F, Schwemmle M (2000) Epidemiology of Borna disease virus. *Journal of General Virology* 81: 2123-2135.

5.5 Brucellosis

5.5.1 Technical information

Background

Brucellosis in horses is caused by small gram negative coccobacilli of the *Brucella* genus (Corbel and MacMillan 1998). There are six *Brucella* species that produce characteristic infections depending on host and species (*B. melitensis, B. abortus, B. suis, B. neotomae, B. canis,* and *B. ovis*) (Moreno et al. 2002). Clinical cases of brucellosis in horses are rare, and most often caused by *B. abortus* and occasionally *B. suis* (McCaughey and Kerr 1967; Cooke and Kingston 1988; Cvetnic et al. 2005; Nicoletti 2007). *B. abortus* is divided into eight biovars on culture and serological properties. However, field strains of the biovars do not differ in their pathogenicity (Godfroid et al. 2004).

Bovine brucellosis, caused by *B. abortus*, is widespread but Australia has been free from the disease since 1989 (Animal Health Australia 2006). Other countries reporting eradication of bovine brucellosis include Austria, Belgium, Canada, Denmark, Finland, Germany, Japan, the Netherlands, New Zealand, Norway, Sweden, Switzerland and the United Kingdom. Australia, unlike some countries which have *B. abortus* in wildlife, is biologically free of this disease agent.

Porcine brucellosis, *B. suis*, is present in Australia but control programs are in place (herd accreditation and pig movement restrictions) and the disease is nationally notifiable.

This chapter will only consider *B. abortus* infection further as it is exotic to Australia and more common in horses. Any conclusions relating to *B. abortus* would be equally applicable to *B. suis*.

Brucellosis (*B. abortus*) is a multiple species OIE-listed disease (OIE 2009b).

Epidemiology

B. abortus is transmitted by ingestion, inhalation, through skin abrasions and mucous membranes from contact with infected cattle and discharges, or contaminated pasture and feed (Denny 1972; Cohen et al. 1992; Corbel and MacMillan 1998).

The major reservoir of *B. abortus* is domestic cattle, though some wild ruminants (elk and bison) are known to harbour infection and can reinfect bovine herds. Other wild and domestic species such as camelids, dogs, horses, moose, and racoons are susceptible to infection by *B. abortus,* but transmission to other animals is considered rare and horse to horse transmission is unlikely (Cohen et al. 1992).

Although *B. abortus* has been isolated from equine faeces (Karlson and Boyd, 1940), urine and aborted foetuses (McNutt and Murray 1924; McCaughey and Kerr 1967; Shortridge 1967; Robertson et al. 1973; Hinton et al. 1977), transmission of infection by horses to cattle or other species has been suggested but not demonstrated (Shortridge 1967; Corbel and MacMillan 1998). Transmission of *Brucellae* from horses to cattle was implied in an early report (White and Swett 1935), and two later reports suggested that aborting mares infected with *B. abortus* were a source of infection to cattle grazing the same pasture (McCaughey and Kerr 1967; Robertson et

al. 1973). Experimentally infected mares developed intermittent bacteraemia for two months after infection but foaled normally and did not shed sufficient bacteria to infect in-contact cattle (MacMillan et al. 1982; MacMillan and Cockrem 1986). It is conceivable that an infected suppurating wound could result in exposure of susceptible animals but *B. abortus* is difficult to isolate from material containing pus (Nicoletti 2007).

In countries with endemic brucellosis affecting cattle and/or small ruminants, 0.2% to 40% of horses have serological evidence of exposure (Hutchins and Lepherd 1968; Denny 1973; Refai 2002; Thakur et al. 2003; Acosta-Gonzalez et al. 2006). The prevalence of brucellosis in horses is higher in animals grazing *Brucella*-contaminated pastures or sharing pasture with infected cattle (Cohen et al. 1992). The incubation period for *B. abortus* in horses is not defined and infection is thought to remain dormant unless the animal is stressed and overt disease develops. Most cases of brucellosis in horses are reported in animals older than three years; however, there is no demonstrated association with age, breed or gender.

Brucellosis is an important zoonosis. Horses with open lesions containing *Brucellae* are a potential source of infection (Acha and Szyfres 2001) but reports of cases in humans through contact with horses are rare, possibly because of poor survival of the organism in material containing pus (Nicoletti 2007).

Clinical signs

Most horses infected with *B. abortus* do not show clinical signs. Others may show pyrexia, stiffness of gait and lethargy. *B. abortus* infection in horses is associated with bursitis, tenosynovitis, arthritis and osteomyelitis (MacMillan et al. 1982) and with septic bursitis over the second and third dorsal vertebral spinous processes (fistulous withers) or the first and second cervical vertebra (poll evil). Up to 80% of horses with fistulous withers or poll evil were seropositive for *B. abortus* (Nicoletti 2007). Other organisms can cause fistulous withers and the isolation of *B. abortus* is less common with the eradication of the disease in cattle (Gaughan et al. 1988). Abortion in horses due to *B. abortus* (and *B. suis*) infection is rare (Shortridge 1967; Robertson et al. 1973; Hinton et al. 1977).

Diagnosis

Brucella spp. are intracellular organisms that require special media and conditions for culture. As a result, they may be difficult to detect in mixed infections. *B. abortus* can be isolated from clinical cases of poll evil and fistulous withers, but it is clearly not the only cause of these syndromes.

Diagnosis of *Brucella* spp. infection relies on isolation of the organism from infected material, or on serological evidence of specific antibody consistent with infection. The OIE Manual (OIE 2009a) outlines diagnostic techniques that are general for all species.

Treatment of brucellosis in horses with antibiotics is rarely effective because of insufficient blood flow to affected tissues.

Conclusion

Brucellosis is present in approved countries but horses are very unlikely to become infected with or to transmit *B. abortus*.

Brucellosis was not considered further in the IRA.

References

Acha PN, Szyfres B (2001) Bacterioses listed B (Botulism, Brucellosis). In *Zoonoses and communicable diseases common to man and animals* (eds. Acha PN, Szyfres B) pp. 28-67. Pan American Health Organization, Washington D.C.

Acosta-Gonzalez RI, Gonzalez-Reyes I, Flores-Gutierrez GH (2006) Prevalence of *Brucella abortus* antibodies in equines of a tropical region of Mexico. *Canadian Journal of Veterinary Research* 70: 302-304.

Animal Health Australia (2006) Brucellosis (*Brucella abortus*). National Animal Health Information System (NAHIS). http://www.animalhealthaustralia.com.au/nahis/pmwiki/pmwiki.php?n=Factsheet.62-1 (Accessed 13 February 2009).

Cohen ND, Carter GK, McMullan WC (1992) Fistulous withers in horses: 24 cases (1984-1990). *Journal of the American Veterinary Medical Association* 201: 121-124.

Cooke DR, Kingston GC (1988) Isolation of *Brucella suis* biotype 1 from a horse. *Australian Veterinary Journal* 65: 162-163.

Corbel MJ, MacMillan AP (1998) Brucellosis. In *Topley & Wilson's microbiology and microbial infections* (eds. Collier L, Balows A, Sussman M) pp. 821-847. Oxford University Press, London.

Cvetnic Z, Spicic S, Curic S, Jukic B, Lojkic M, Thiebaud M, Garin-Bastuji B (2005) Isolation of *Brucella suis* biovar 3 from horses in Croatia. *The Veterinary Record* 156: 584-585.

Denny HR (1972) Brucellosis in the horse. *The Veterinary Record* 90: 86-91.

Denny HR (1973) A review of brucellosis in the horse. *Equine Veterinary Journal* 5: 121-125.

Gaughan EM, Fubini SL, Dietze A (1988) Fistulous withers in horses: 14 cases (1978-1987). *Journal of the American Veterinary Medical Association* 193: 964-966.

Godfroid J, Bosman PP, Herr S, Bishop GC (2004) Bovine brucellosis. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 1510-1527. Oxford University Press, Oxford.

Hinton M, Barker GL, Morgan TL (1977) Abortion in a mare associated with *Brucella abortus* infection and twins. *The Veterinary Record* 101: 526.

Hutchins DR, Lepherd EE (1968) The occurrence of agglutinins to *Brucella abortus* in horses. *Australian Veterinary Journal* 44: 323-325.

MacMillan AP, Baskerville A, Hambleton P, Corbel MJ (1982) Experimental *Brucella abortus* infection in the horse: observations during the three months following inoculation. *Research in Veterinary Science* 33: 351-359.

MacMillan AP, Cockrem DS (1986) Observations on the long term effects of *Brucella abortus* infection in the horse, including effects during pregnancy and lactation. *Equine Veterinary Journal* 18: 388-390.

McCaughey WJ, Kerr WR (1967) Abortion due to brucellosis in a thoroughbred mare. *The Veterinary Record* 80: 186-187.

McNutt SH, Murray C (1924) *Bacterium abortum* (Bang) isolated from the fetus of an aborting mare. *Journal of the American Veterinary Medical Association* 65: 215-216.

Moreno E, Cloeckaert A, Moriyón I (2002) *Brucella* evolution and taxonomy. *Veterinary Microbiology* 90: 209-227.

Nicoletti PL (2007) Brucellosis. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 348-350. Saunders Elsevier, St. Louis.

OIE (2009a) Bovine brucellosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.04.03_BOVINE_BRUCELL.pdf (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Refai M (2002) Incidence and control of brucellosis in the Near East region. *Veterinary Microbiology* 90: 81-110.

Robertson FJ, Milne J, Silver CL, Clark H (1973) Abortion associated with *Brucella abortus* (Biotype 1) in the TB mare. *The Veterinary Record* 92: 480-481.

Shortridge EH (1967) Two cases of suspected *Brucella abortus* abortion in mares. *New Zealand Veterinary Journal* 15: 33-34.

Thakur SD, Vaid RK, Kumar M (2003) Equine brucellosis: a review. *Centaur* 19: 46-52.

White GC, Swett PP (1935) Bang's disease infection transmitted to a dairy herd by horses. *Journal of the American Veterinary Medical Association* 87: 146-150.

5.6 Contagious equine metritis

5.6.1 Technical information

Background

Taylorella equigenitalis is the causal agent of contagious equine metritis (CEM), a venereal disease of equids. *T. equigenitalis* is the type species of the genus *Taylorella* (Swerczek 1981). Two strains are usually recognised, based on the sensitivity of isolates to streptomycin. However, 32 strains have been characterised and these are further divided into five distinct groups (Bleumink-Pluym et al. 1990).

CEM was first described in the United Kingdom (Crowhurst et al. 1977) and Ireland (O'Driscoll 1977) in their spring of 1977, and in Australia later that year (Hughes et al. 1978). Further cases occurred in Australia in the breeding seasons of 1978 and 1979 but no additional cases have been recorded since 1980. The estimated cost of the outbreak to industry exceeded A$14 million (Pascoe pers. comm. 1998). From 1996 to 2007, cases of CEM were reported in Belgium (7), the Czech Republic (8), Denmark (3), Finland (9), France (69), Germany (40), Japan (18), Slovenia (45), Sweden (19), Switzerland (1) and the United Kingdom (13) (OIE 2009b). On 15 December 2008, a horse in Kentucky tested positive to CEM. Since then 21 positive stallions and five positive mares from hundreds of horses exposed in over 46 states in the United States have been reported, showing widespread dissemination of infection during an outbreak (USDA 2009). There may be an underestimation of the distribution of CEM worldwide as it not notifiable in some countries.

The United States has detected a small number of CEM cases in post-arrival quarantine. A total of 28 stallions and mares imported into the United States from Europe from 1997 to 2007 were carriers of *T. equigenitalis*. All of these horses were certified negative for CEM on pre-export bacteriological screening for the presence of *T. equigenitalis* (Timoney 2007).

Twenty of the 28 carriers of *T. equigenitalis* detected in the United States were stallions. Of 16 stallions test-mated to two mares, 13 were culture-negative on initial bacteriological examination. In 11 of the 16 cases, both test mares were infected; the other five stallions transmitted infection to only one of the two mares with which they were test-mated.

CEM is an OIE-listed disease (OIE 2009c).

Epidemiology

T. equigenitalis affects horses and donkeys, but infection in donkeys appears to be self-limiting with minimal clinical signs (Timoney et al. 1984). Although undomesticated equids (Przewalski's horses, onagers and zebras) may be susceptible to *T. equigenitalis* infection, these animals are unlikely to play a significant role in the epidemiology of CEM.

The existence of carrier stallions and mares is the major factor in the epidemiology of CEM. Stallions and clinically recovered mares may harbour *T. equigenitalis* for extended periods — sometimes years after initial infection — whether or not clinical signs of disease or reduced fertility are apparent. *T. equigenitalis* may persist on mucous membranes of the clitoral sinus or fossa, and in the uterus even during

pregnancy. In stallions, the organism localises in the urethral fossa and associated sinus, as well as in the distal urethra and on the external surface of the penis and prepuce. Bacteria can also colonise the external genitalia of newborn foals of infected dams at parturition, forming a potential source of infection years later as the foal reaches sexual maturity (Timoney and Powell 1982).

Stallions pose the greatest risk as they are subclinically infected or latent carriers and more likely to spread the disease, especially when used extensively during breeding seasons. Mares are also a risk, particularly latent carriers. During mating, stallions can become infected from a mare carrier and then transmit disease to other mares.

Geldings are unlikely to harbour the bacteria as the usual form of transmission is through coitus. However, infected geldings have been reported despite no history of mating (Burger and Dobretsberger 2007). Geldings are not considered to be involved in the epidemiology of CEM.

Studies indicate there is widespread presence of *T. equigenitalis* in a variety of breeds, including Icelandic horses (Parlevliet et al. 1997). Presence in an isolated population over a long period without apparent clinical signs was attributed to non-pathogenic strains that might be regarded as commensal organisms.

CEM is spread primarily through natural service or artificial insemination (Timoney 1996). In the latter case, infection can occur through the use of contaminated semen, and mechanical spread by contamination of equipment or unhygienic breeding practices.

Clinical signs

Clinical signs of disease are only seen in mares, with the incubation period ranging from 2 to 12 days after breeding.

In mares, there are two states of infection: acute disease and chronic carrier states. Acute disease results in endometritis with a grey/mucoid vulval discharge that ranges from very mild to profuse. At this stage of infection, the organism can be detected in 84% of cervical swabs and 69% of clitoral swabs (Wood et al. 2005). Endometritis and associated cervicitis and vaginitis last for about two weeks , with a return to oestrus after a shortened dioestrus and failure to conceive (Timoney 1996). In the chronic carrier state there are no outward signs of infection or long-term adverse effects on fertility. However, the organism establishes itself in the clitoral region and can be transmitted to susceptible animals. In one study in chronically infected mares, the organism was detected in 93% of clitoral swabs but in only 31% of cervical swabs (Wood et al. 2005).

Infected stallions are mechanical carriers with no clinical signs of infection.

Diagnosis

Identification of the organism by culture is the prescribed test for international trade (OIE 2008).

Culture, isolation and identification of *T. equigenitalis* forms the basis of a definitive diagnosis but false negative cultures are not uncommon (Bleumink-Pluym et al. 1994; Parlevliet et al. 1997). Specific swabbing, transport methods and culture techniques are needed for isolation to be successful (Ricketts 1996).

T. equigenitalis is a gram-negative nonmotile coccobacillus. It is a fastidious organism requiring 5–10% CO_2 and 35–37 °C for culture. Growth can vary from 72 hours to 14 days (Ward et al. 1984), but an incubation period of six days (HBLB 2007) or seven days (OIE 2008) is recommended before certifying cultures as negative for *T. equigenitalis*.

Colonies are small, smooth, glossy and yellow–grey (OIE 2008).

Suitable transport media are critical to bacterial survival and refrigeration extends viability during transport. Swabs in transport media survived 10 days at −70 °C (Sahu et al. 1979). Dried cultures of *T. equigenitalis* are killed on exposure to common disinfectants (2% chlorhexidine diacetate, 10% benzalkonium chloride) for ten minutes (Swaney and Kislow 1981). In mares, swabs are collected from the clitoris, including the fossa and sinuses, and the cervix or endometrium. In chronically infected mares, the organism was detected in 93% of clitoral swabs and in 31% of cervical swabs. However, in acutely infected mares, the organism was detected in 69% of clitoral swabs and in 84% of cervical swabs (Wood et al. 2005).

For stallions, swabs are collected from the penile sheath, urethral fossa or sinus and urethra. Previously, a sample of pre-ejaculatory fluid was obtained and cultured; however this is not considered as reliable as culture from other sample sites (Böse et al. 2007). Diagnostic techniques recommended in the OIE manual (OIE 2008) for sampling both stallions and mares are those outlined in the British Horserace Betting Levy Board Code of Practice (HBLB 2007).

Swabs should be transported to a laboratory in Amies charcoal medium, kept cool and plated within 24–48 hours of collection (HBLB 2007; OIE 2008).

In 2006, an international ring trial of various laboratories showed discrepancies in results from different laboratories. Five samples containing pure *Taylorella* cultures, mixed cultures (*Taylorella* spp. and contaminants), or non-*Taylorella* species (contaminants only) gave false positive and false negative results (Heath et al. 2007). This highlights the need for consistency in laboratory culture methods to address variance in test results.

Serology

Serological testing for diagnosis and control of CEM is not reliable on its own for detecting infection with *Taylorella* species (OIE 2008). Carrier animals may be infectious in the absence of humoral antibody (Rogerson 1993). Stallions do not show serological evidence of infection and the antibody response in mares occurs early in infection declining as the organism is eliminated, even if they remain carriers (Timoney 1996).

Complement fixation and microtitration serum agglutination tests are effective in distinguishing between positive and negative sera. However, serological tests do not reliably detect carriers (Gummow et al. 1986).

The complement fixation test can be useful in confirming recent cases of CEM infection in mares (Powell 1981). In an outbreak, serology may aid in epidemiological investigations (Rogerson 1993). The number of *T. equigenitalis* organisms mechanically carried by subclinically infected or latent carrier stallions is very low and they may not be detected by culture methods alone. However, serology can be used as an adjunct to culture for *T. equigenitalis* in screening mares between 21 and 45 days after being mated to a suspect carrier stallion (OIE 2008).

Molecular tests

Molecular techniques have been used in diagnosis, both in swab material and on isolates. Molecular genotyping has identified strain differences in isolates from different geographical areas (Bleumink-Pluym et al. 1990; Matsuda et al. 1998).

When comparing polymerase chain reaction (PCR) to conventional culture methods, the PCR-based techniques are more sensitive and faster in confirming diagnosis (Bleumink-Pluym et al. 1993; Anzai et al. 1999; Bleumink-Pluym et al. 1994; Chanter et al. 1998; Premanandh et al. 2003). PCR can also differentiate between *T. equigenitalis* and *T. asinigenitalis* without the need for any prior bacteriological DNA extraction or bacterial isolation (Duquesne et al. 2007; Wakeley et al. 2006).

Despite the existence of a number of PCR assays, none has been validated for use as a routine diagnostic test.

Treatment

Several treatment protocols have been described.

Stallions

The United States Department of Agriculture (USDA) treatment protocol for positive stallions states that the external genitalia (prepuce, penis, fossa glandis and urethral sinus) must be thoroughly cleaned with no less than 2% chlorhexidine scrub while the stallion's penis is in full erection. After cleaning, the entire penis is coated with an antibiotic ointment with activity against *T. equigenitalis,* such as silver sulfadiazine or 0.2% nitrofurazone. This must be repeated daily for five consecutive days. Following treatment, the stallion will then be retested by collecting three sets of cultures on days one, four and seven of a one week period, beginning no less than 21 days after the last day of treatment. If all cultures are negative, the stallion can then be test mated to two mares (USDA:APHIS 2009).

Mares

The USDA treatment protocol for positive mares states that on day one smegma should be manually expressed from the central sinus of the clitoris. Clitoral sinuses are then infused with a ceruminolytic agent, and then flushed with saline. Clitoral sinuses and clitoral fossa are scrubbed and cleaned with no less than 2% chlorhexidine scrub. Sinuses and the entire clitoral area are infused with an antibiotic ointment with activity against *T. equigenitalis,* such as silver sufladiazine or 0.2% nitrofurazone. Cleaning, scrubbing and antibiotic infusion are continued daily from days two to five. Following treatment, the mare will be retested by collecting three sets of culture on days one, four and seven of a one week period, being no less than 21 days after the last day of treatment. After day seven the distal portion of the vaginal tract is cleaned and disinfected. A distal cervix or endometrial swab is then collected (USDA:APHIS 2009).

Recesses of the clitoral fossa and sinuses of mares are frequent sites of colonisation by *T. equigenitalis* in carrier animals and thorough local treatment is needed to eliminate the pathogen (OIE 2008). Treatment may take several weeks and may need to be repeated before intensive swabbing consistently fails to recover *T. equigenitalis*.

A number of carrier mares have been reported to be refractive to several courses of treatment (OIE 2008). Treatment of CEM in fillies has also been ineffective on occasions and persistence of the organism has been demonstrated on bacterial culture

and/or a number of test matings (Timoney 1996). However, in most cases, a single treatment course is successful in eliminating *T. equigenitalis* from both colts and fillies (Powell 1978).

Despite cases of protracted treatment of CEM in stallions, all infected stallions have responded to treatment eventually (Timoney pers. comm. 2008).

Immunology

Effective vaccines that protect against CEM infection or prevent colonisation by *T. equigenitalis* are not available (OIE 2008).

Conclusion

CEM is present in approved countries. The Code recommendations (OIE 2009a) include premises freedom and diagnostic testing. Certification requirements, in accordance with the Code recommendations, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Anzai T, Eguchi M, Sekizaki T, Kamada M, Yamamoto K, Okuda T (1999) Development of a PCR test for rapid diagnosis of contagious equine metritis. *The Journal of Veterinary Medical Science* 61: 1287-1292.

Bleumink-Pluym N, ter Laak EA, van der Zeijst BA (1990) Epidemiologic study of *Taylorella equigenitalis* strains by field inversion gel electrophoresis of genomic restriction endonuclease fragments. *Journal of Clinical Microbiology* 28: 2012-2016.

Bleumink-Pluym NM, Houwers DJ, Parlevliet JM, Colenbrander B (1993) PCR-based detection of CEM agent. *The Veterinary Record* 133: 375-376.

Bleumink-Pluym NMC, Werdler ME, Houwers DJ, Parlevliet JM, Colenbrander B, van der Zeijst BA (1994) Development and evaluation of PCR test for detection of *Taylorella equigenitalis*. *Journal of Clinical Microbiology* 32: 893-896.

Böse R, Kache C, Wonnemann H, Nurnus U (2007) Current situation with CEM in Germany. *First international CEM conference 2007*

Burger D, Dobretsberger M (2007) CEM in stallions at the Spanish riding school in Vienna. *First international CEM conference 2007*

Chanter C, Vigano F, Collin NC, Mumford JA (1998) Use of a PCR assay for *Taylorella equigenitalis* applied to samples from the United Kingdom. *The Veterinary Record* 143: 225-227.

Crowhurst RC, Simpson DJ, Greenwood RE, Ellis DR (1977) Swabbing for contagious metritis tests. *The Veterinary Record* 101: 413.

Duquesne F, Pronost S, Laugier C, Petry S (2007) Identification of *Taylorella equigenitalis* responsible for contagious equine metritis in equine genital swabs by direct polymerase chain reaction. *Research in Veterinary Science* 82: 47-49.

Gummow B, Herr S, Brett OL (1986) A short, reliable, highly reproducible complement fixation test for the serological diagnosis of contagious equine metritis. *Onderstepoort Journal of Veterinary Research* 53: 241-243.

HBLB (2007) Codes of Practice on equine diseases. Horserace Betting Levy Board. http://www.hblb.org.uk/ (Accessed 3 August 2007).

Heath P, Markey G, Saywell W (2007) Results of two CEM ring trials. *First international conference on contagious equine metritis, July 11-12, 2007, Lelystad, The Netherlands.*

Hughes KL, Bryden JD, MacDonald F (1978) Equine contagious metritis. *Australian Veterinary Journal* 54: 101.

Matsuda M, Miyazawa T, Moore JE, Buckley TC, Thomas LA (1998) Molecular genotyping by pulsed field gel electrophoresis of restricted genomic DNA of strains of *Taylorella equigenitalis* isolated in Ireland and the United States. *Veterinary Research Communications* 22: 217-224.

O'Driscoll J (1977) Venereal infection in thoroughbreds with *Bacillus proteus mirabilis*. *The Veterinary Record* 100: 534.

OIE (2008) Contagious equine metritis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.02_CEM.pdf (Accessed 1 September 2009).

OIE (2009a) Contagious equine metritis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.2.htm (Accessed 1 September 2009a).

OIE (2009b) List of countries by disease situation: contagious equine metritis. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_status_lists&disease_type=Terrestrial&disease_id=55&empty=999999 (Accessed 18 June 2009b).

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009c).

Parlevliet JM, Bleumink-Pluym NM, Houwers DJ, Remmen JL, Sluijter FJ, Colenbrander B (1997) Epidemiologic aspects of *Taylorella equigenitalis*. *Theriogenology* 47: 1169-1177.

Powell DG (1978) Contagious equine metritis. *Equine Veterinary Journal* 10: 1-4.

Powell DG (1981) Contagious equine metritis. *Advances in Veterinary Science and Comparative Medicine* 25: 161-184.

Premanandh J, George LV, Wernery U, Sasse J (2003) Evaluation of a newly developed real-time PCR for the detection of *Taylorella equigenitalis* and discrimination from *T. asinigenitalis*. *Veterinary Microbiology* 95: 229-237.

Ricketts SW (1996) Contagious equine metritis (CEM). *Equine Veterinary Education* 8: 166-170.

Rogerson B (1993) Contagious equine metritis. In *Australian standard diagnostic techniques for animal diseases* (eds. Corner LA, Bagust TJ) pp. 1-10. Commonwealth Scientific and Industrial Research Organisation for the Standing Committee on Agriculture and Resource Management, Melbourne.

Sahu SP, Dardiri AH, Rommel FA, Pierson RE (1979) Survival of contagious equine metritis bacteria in transport media. *American Journal of Veterinary Research* 40: 1040-1042.

Swaney LM, Kislow HM (1981) Disinfection of the causative agent of contagious equine metritis by Nolvasan and Roccal II. *Veterinary Microbiology* 6: 59-68.

Swerczek TW (1981) Contagious equine metritis: test for suspect carriers. *The Veterinary Record* 108: 420-421.

Timoney P (2007) Risk of spread of contagious equine metritis through international trade. In *First international conference on contagious equine metritis, July 11-12, 2007, Lelystad, The Netherlands,* pp. 10-11, Central Institute for Animal Disease Control -Lelystad of Wageningen University and Research Centre, Wageningen.

Timoney PJ (1996) Contagious equine metritis. *Comparative Immunology, Microbiology and Infectious Diseases* 19: 199-204.

Timoney PJ, O'Reilly PJ, McArdle JF, Ward J, Harrington AM (1984) Contagious equine metritis: experimental infection in the donkey. *Veterinary Microbiology* 10: 259-268.

Timoney PJ, Powell DG (1982) Isolation of the contagious equine metritis organism from colts and fillies in the United Kingdom and Ireland. *The Veterinary Record* 111: 478-482.

USDA (2009) Contagious equine metritis. United States Department of Agriculture, Animal and Plant Health Inspection Agency. http://www.aphis.usda.gov/newsroom/hot_issues/cem/index.shtml (Accessed 26 August 2009).

USDA:APHIS (2009) *USDA APHIS veterinary services FY 2009 CEM incident testing protocol for CEM investigations*. United States Department of Agriculture, Animal and Plant Health Inspection Service, United States of America.

Wakeley PR, Errington J, Hannon S, Roest HIJ, Carson T, Hunt B, Sawyer J, Heath P (2006) Development of a real time PCR for the detection of *Taylorella equigenitalis* directly from genital swabs and discrimination from *Taylorella asinigenitalis*. *Veterinary Microbiology* 118: 247-254.

Ward J, Hourigan M, McGuirk J, Gogarty A (1984) Incubation times for primary isolation of the contagious equine metritis organism. *The Veterinary Record* 114: 298.

Wood JL, Kelly L, Cardwell JM, Park AW (2005) Quantitative assessment of the risks of reducing the routine swabbing requirements for the detection of *Taylorella equigenitalis*. *The Veterinary Record* 157: 41-46.

5.7 Dourine

5.7.1 Technical information

Background

Trypanosoma equiperdum is classically recognised as the cause of dourine, a sexually transmitted disease of horses and donkeys. Dourine is mostly reported in Africa, eastern Europe and the Russian Federation and has been eradicated from Canada, western Europe and the United States. Dourine has not been reported in Australia.

A single case of dourine was reported in Germany in a mare in 2002 (OIE 2002). The horse was subclinically infected and diagnosed serologically as a result of an export test. The previous report of dourine in Germany was in 1953. The mare had been imported from eastern Europe several years before and had never foaled. This horse was euthanased. There are no other recent reports of dourine in approved countries.

T. equiperdum is morphologically identical to *T. evansi,* the cause of surra, and to the slender form of *T. brucei brucei*, a cause of nagana, a tsetse-transmitted trypanosome of livestock in Africa (Soulsby 1982; Stephen 1986). However, some authors consider that *T. equiperdum* is synonymous with *T. evansi* (Monzón and Russo 1997) and the distinction of these trypanosomes at the species level remains uncertain (Claes et al. 2005). Phylogenetic studies of trypanosomes (Brun et al. 1998; Claes et al. 2003; Haag et al. 1998) have been unable to establish the relationships between these species.

Dourine is an OIE-listed disease (OIE 2009b).

Epidemiology

T. equiperdum is transmitted in seminal fluid and mucous genital exudates at mating. Foals can be infected by genital tract discharges from infected mares or from milk contaminated with discharges from lesions on the udder (Hoare 1972). Transmission by needles and by arthropod vectors may occur but, due to the transient and low grade parasitaemia transmission by these methods is unlikely.

Although evolution and phylogeny of *T. equiperdum* are not established, its direct mode of transmission differentiates this parasite from other trypanosomes and does not limit its distribution to regions that have suitable vectors.

Clinical signs

The incubation period varies from one week to several months. Descriptions of clinical signs of dourine include anaemia, vaginal or urethral discharge, genital oedema, urticarial plaques and sometimes neurological signs with recovery and cycles of periodic relapse (Geering et al. 1995).

Infection may be so mild that clinical signs of disease are not observed. About half of infected animals die (Stephen 1986) although spontaneous recovery and latent carriers may occur (OIE 2008). Horses are considered more susceptible than donkeys (OIE 2008).

Diagnosis

The transient presence of trypanosomes in the blood makes direct microscopy unreliable. Concentration techniques such as centrifugation and examination of the buffy coat have been used, as has examination of vaginal and preputial washings.

Testing for humoral antibody can be done by complement fixation, agar gel immunodiffusion, enzyme-linked immunosorbent assay, card agglutination and by indirect fluorescent antibody. Cross-reactions with other trypanosomes (Zablotskij et al. 2003) can confuse results, as can anticomplementary effects of some sera (OIE 2008).

There are no vaccines available and the only effective means of controlling spread is euthanasia of infected animals.

Conclusion

Dourine is not present in any approved country. While this remains the case, certification of country freedom, in accordance with the Code recommendations (OIE 2009a), will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Brun R, Hecker H, Lun ZR (1998) *Trypanosoma evansi* and *T. equiperdum*: distribution, biology, treatment and phylogenetic relationship (a review). *Veterinary Parasitology* 79: 95-107.

Claes F, Agbo EC, Radwanski M, te Pas MFW, Baltz T, de Waal DT, Goddeeris BM, Claasen E, Büscher P (2003) How does *Trypanosoma equiperdum* fit into the *Trypanozoon* group? a cluster analysis by RAPD and multiplex-endonuclease genotyping approach. *Parasitology* 126: 425-431.

Claes F, Büscher P, Touratier L, Goddeeris BM (2005) *Trypanosoma equiperdum*: master of disguise or historical mistake? *Trends in Parasitology* 21: 316-321.

Geering WA, Forman AJ, Nunn MJ (1995) Dourine. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 368-371. Australian Government Publishing Service, Canberra.

Haag J, O'huigin C, Overath P (1998) The molecular phylogeny of trypanosomes: evidence for an early divergence of the Salivaria. *Molecular and Biochemical Parasitology* 91: 37-49.

Hoare CA (1972) Subgenus *Trypanozoon*. In *The trypanosomes of mammals: a zoological monograph* pp. 554-604. Blackwell Scientific Publications, Oxford.

Monzón CM, Russo AM (1997) Epidemiological review of equine trypanosomosis in Argentina. Proceedings of the first internet conference on Salivarian trypanosomes. http://www.fao.org/docrep/W5781E/w5781e03.htm#epidemiological%20review%20of%20equine%20trypanosomosis%20in%20argentina (Accessed 24 December 2003).

OIE (2002) Dourine in Germany. *Disease Information* 15: 220.

OIE (2008) Dourine. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.03_DOURINE.pdf (Accessed 1 September 2009).

OIE (2009a) Dourine. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.3.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Soulsby EJL (1982) Protozoa. In *Helminths, arthropods and protozoa of domesticated animals* pp. 507-759. Baillière Tindall, London.

Stephen LE (1986) *Trypanosoma (Trypanozoon) Equiperdum* Dolfein, 1901. In *Trypanosomiasis: a veterinary perspective* pp. 216-225. Pergamon Press, Oxford.

Zablotskij VT, Georgiu C, de Waal T, Clausen PH, Claes F, Touratier L (2003) The current challenges of dourine: difficulties in differentiating *Trypanosoma equiperdum* within the subgenus *Trypanozoon*. *Revue Scientifique et Technique de l'Office International des Epizooties* 22: 1087-1096.

5.8 Echinococcosis

5.8.1 Technical information

Background

Echinococcosis is a zoonotic disease caused by different species of the cestode *Echinococcus* in the family Taeniidae. The taxonomy of the genus *Echinococcus* is being revised. *E. granulosus* is now believed to exist as a complex of distinct species, differing in a variety of criteria which affect the epidemiology, pathology and control of cystic hydatid disease (Romig et al. 2006; Busi et al. 2007). The most significant zoonotic species are *E. granulosus* and *E. multilocularis*.

E. granulosus has a worldwide distribution including Australia. *E. multilocularis* is found in Africa, Asia, Canada, Europe and the United States, and can cause disease in humans, although the disease is rare (Sréter et al. 2003). *E. equinus* is found in Africa, Europe and the Middle East (Torgerson and Budke 2003) and appears to be non-pathogenic to humans.

There are no reports of *E. multilocularis* or *E. equinus* in Australia (Thompson and McManus 2001; Animal Health Australia 2007).

Echinococcosis is a multiple species OIE-listed disease (OIE 2009b). There are no recommendations in the Code for the purpose of international trade for live animals other than carnivores (OIE 2009a).

Epidemiology

Carnivores are the definitive hosts for *Echinococcus* spp., with mammals (including humans and horses) acting as intermediate hosts (Torgerson and Budke 2003). The infective stage, or proglottid, of *Echincoccus* spp. is shed in faeces of the definitive hosts. Tissue invasion, in the form of hydatid cysts, occurs in intermediate hosts after ingestion of proglottids. In horses, cysts grow slowly in the liver and occasionally occur in the lungs. Cystic stages in intermediate hosts cannot be transmitted unless tissues containing mature cysts are ingested by the definitive hosts (Sellon 2007).

Equids are intermediate hosts for *E. equinus* and dogs the definitive host (Torgerson and Budke 2003; Romig et al. 2006). The cycle is maintained by feeding dogs raw or undercooked horse offal (Torgerson and Budke 2003).

The United States has reported disease due to *E. equinus* in four horses, which originated from the United Kingdom and Ireland, where the disease is endemic. In the United Kingdom, prevalence of *E. equinus* is higher in horses used for hunting. In Ireland, prevalence of *E. equinus* in slaughtered horses ranges from 10% to 62% (Sellon 2007). Disease is found in all age groups (Thompson and Smyth 1975).

A number of carnivores act as the definitive hosts for *E. multilocularis*. Intermediate hosts are small mammals, usually rodents. In rare cases, domestic animals (including horses) and humans can also become infected (CFSPH 2005).

Clinical signs

Clinical manifestation of echinococcosis in horses is rare, and the disease is usually diagnosed at slaughter (Thompson and Smyth 1975). If clinical signs do occur, they

are related to pressure of the growing cyst on surrounding organs and tissue (Sellon 2007).

Diagnosis

There are no definitive tests that can be performed ante mortem in horses, and diagnosis is most commonly made at post mortem examination (Sellon 2007).

Treatment

Long-term treatment with anthelmintics can suppress some of the cysts caused by *E. granulosus* and *E. multilocularis.* Surgical removal of cysts is the definitive treatment.

Conclusion

E. equinus and *E. multilocularis* are present in some approved countries and there are no recommendations in the Code for horses. There are no reports of significant disease in horses and the disease cannot be transmitted by live horses.

Echinococcosis was not considered further in the IRA.

References

Animal Health Australia (2007) *Animal health in Australia 2006*. Animal Health Australia, Canberra.

Busi M, Snábel V, Varcasia A, Garippa G, Perrone V, De Liberato C, D'Amelio S (2007) Genetic variation within and between G1 and G3 genotypes of *Echinococcus granulosus* in Italy revealed by multilocus DNA sequencing. *Veterinary Parasitology* 150: 75-83.

CFSPH (2005) Echinococcosis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 22 April 2009).

OIE (2009a) Echinococcosis/hydatidosis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.4.htm (Accessed 8 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Romig T, Dinkel A, Mackenstedt U (2006) The present situation of echinococcosis in Europe. *Parasitology International* 55: S187-S191.

Sellon DC (2007) Miscellaneous parasitic diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 473-480. Saunders Elsevier, St. Louis.

Sréter T, Széll Z, Egyed Z, Varga I (2003) *Echinococcus multilocularis*: an emerging pathogen in Hungary and central eastern Europe. *Emerging Infectious Diseases* 9: 384-386.

Thompson RCA, McManus DP (2001) Aetiology: parasites and life-cycles. In *WHO/OIE manual on echinococcosis in humans and animals: a public health problem of global concern* pp. 1-16. Office International des Épizooties, Paris.

Thompson RCA, Smyth JD (1975) Equine hydatidosis: a review of the current status in Great Britain and the results of an epidemiological survey. *Veterinary Parasitology* 1: 107-127.

Torgerson PR, Budke CM (2003) Echinococcosis - an international public health challenge. *Research in Veterinary Science* 74: 191-202.

5.9 Epizootic lymphangitis

5.9.1 Technical information

Background

Epizootic lymphangitis is a contagious, chronic disease of horses, mules and donkeys caused by the saprophytic soil fungus, *Histoplasma capsulatum* var. *farciminosum* (also known as *Histoplasma farciminosum, Cryptococcus farciminosis, Zymonema farciminosa* and *Saccharomyces farciminosus*). Infection in other species is rare, but has been reported to occur in camels, dogs and humans (Ueda et al. 2003; CFSPH 2005). Epizootic lymphangitis — also known as pseudofarcy, pseudoglanders or equine histoplasmosis — is characterised by a spreading, suppurative dermatitis and lymphangitis, ulcerating conjunctivitis or multifocal pneumonia.

The disease is more common in the tropics and subtropics and is endemic in northern Africa and parts of Asia, including China, India and Pakistan (Picard and Vismer 2004; Kohn 2007). Epizootic lymphangitis is reported to have occurred in Japan before World War II (Chandler et al. 1980, cited in Ueda et al. 2003) and one autochthonous case was described in 2001 (Katayama et al. 2001).

Epizootic lymphangitis is not an OIE-listed disease (OIE 2009b), however, the Code includes recommendations for the importation of horses with respect to epizootic lymphangitis (OIE 2009a).

Epidemiology

H. capsulatum var. *farciminosum* exists as a yeast in tissues and a mycelium in the environment. In its saprophytic soil phase, *H. capsulatum* var. *farciminosum* is relatively resistant to environmental conditions and can survive for many months in a warm, moist environment. The disease is contagious between horses and is a zoonosis. Transmission can occur via fomites, biting flies of the *Musca* or *Stomoxys* genera, contact of infected material with traumatised skin, venereally and inhalation (Picard and Vismer 2004; Kohn 2007). Morbidity is higher when large numbers of animals are gathered together and epizootic lymphangitis was a serious concern during the early 20th century. The disease is economically important in areas of the world where large numbers of horses, donkeys, or mules are assembled (CFSPH 2005).

Clinical signs

The incubation period varies from several weeks to six months (Kohn 2007) and the organism, once established, spreads locally by invasion and then via the lymphatics. Epizootic lymphangitis sometimes spreads to the underlying joints. Occasionally, conjunctivitis, keratoconjunctivitis, a nasal discharge or pneumonia can also occur. The lymph nodes may be enlarged, but pyrexia is uncommon. Lesions usually heal spontaneously after 2–3 months. Mortality is 10–15%; however, extensive lesions with high mortality rates can occur in areas where there is poor veterinary care and nutrition (Picard and Vismer 2004).

Diagnosis

Epizootic lymphangitis is diagnosed by detecting *H. capsulatum* var. *farciminosum* in tissue sections or smears of lesions. *H. capsulatum* var. *farciminosum* can be cultured from lesions in about half of the cases. Antibodies have been detected by indirect and direct fluorescent antibody tests, enzyme-linked immunosorbent assay, passive haemagglutination and skin hypersensitivity tests. Inoculation of samples into immunosuppressed mice can also be used for diagnosis (OIE 2008).

Conclusion

Epizootic lymphangitis is present in some approved countries. The Code recommendations (OIE 2009a) include premises freedom. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

CFSPH (2005) Epizootic lymphangitis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 12 March 2009).

Katayama Y, Kuwano A, Yoshihara T (2001) Histoplasmosis in the lung of a race horse with yersiniosis. *The Journal of Veterinary Medical Science* 63: 1229-1231.

Kohn C (2007) Miscellaneous fungal diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 431-445. Saunders Elsevier, St. Louis.

OIE (2008) Epizootic lymphangitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.04_EPIZ_LYMPHANGITIS.pdf (Accessed 1 September 2009).

OIE (2009a) Epizootic lymphangitis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.4.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Picard JA, Vismer HF (2004) Mycoses. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 2095-2136. Oxford University Press, Oxford.

Ueda Y, Sano A, Tamura M, Inomata T, Kamei K, Yokoyama K, Kishi F, Ito J, Mikami Y, Miyaji M, Nishimura K (2003) Diagnosis of histoplasmosis by detection of the internal transcribed spacer region of fungal rRNA gene from a paraffin-embedded skin sample from a dog in Japan. *Veterinary Microbiology* 94: 219-224.

5.10 Equid herpesviruses

5.10.1 Technical information

Background

Equid herpesviruses (EHVs) are members of the family Herpesviridae, order Herpesvirales, and are present in equid populations worldwide (Davison et al. 2005). There are nine EHVs, six in the subfamily Alphaherpesvirinae (EHV-1, EHV-3, EHV-4, EHV-6, EHV-8, EHV-9) and three in the subfamily Gammaherpesvirinae (EHV-2, EHV-5, EHV-7) (Davison et al. 2009). Of the nine EHVs characterised, only EHV-1 to EHV-5 infect domestic horses (Slater 2007). EHV-6, EHV-7 and EHV-8 have been reported in donkeys (Browning et al. 1988). Serological evidence of EHV-9 is reported in captive zoo animals (Fukushi et al. 1997; Kasem et al. 2008; Schrenzel et al. 2008) and zebras (Borchers et al. 2008). Zebras may serve as a source of infection for other animals (Borchers et al. 2008).

EHV-1 is also known as equine abortion virus, EHV-2 as equine cytomegalovirus, EHV-3 as equine coital exanthema virus, EHV-4 as equine rhinopneumonitis, EHV-6 as asinine herpesvirus 1, EHV-7 as asinine herpesvirus 2 and EHV-8 as asinine herpesvirus 3 (Pellett and Roizman 2007). EHV-6 and EHV-9 have not been reported in Australia.

Equine rhinopneumonitis is a collective term for any of several extremely contagious disease entities of equids, caused by two closely related herpesviruses, EHV-1 and EHV-4 (OIE 2009a). EHV-1 and EHV-4 are closely related but can be distinguished antigenically and genetically (Slater 2007). Both EHV-1 and EHV-4 are respiratory pathogens that initially establish infection in the upper respiratory tract. EHV-1 can establish a lymphocyte associated viraemia, which is the means of systemic spread to other organs.

EHV-1 infects a variety of cells (e.g. endothelial, epithelial, lymphoid, neuronal and respiratory) which distinguishes it from EHV-4, which is restricted mainly to epithelial and neuronal cells. The disease manifestations of EHV-1 systemic infection include abortion and neurological disease. Most EHV-1 abortions occur as sporadic single abortions late in gestation, but poor management of the index case may result in outbreaks of abortion. EHV-1 neurological disease has been reported with increased frequency in the last decade, particularly in the United States and Europe (Slater 2007). Nucleotide sequence analysis of EHV-1 isolates from cases of neurological disease found that a high proportion of these isolates contained a mutation in the polymerase gene (Nugent et al. 2006) that has subsequently been shown to be associated with high virus titres in circulating blood (Allen and Breathnach 2006).

There are no movement controls or official eradication or control programs for EHVs in Australia. However, EHV-1 (abortigenic and neurological disease) is nationally notifiable. Some breeding establishments voluntarily comply with the recommendations of Equine Veterinarians Australia (formerly the Australian Equine Veterinary Association) on procedures including prevention, abortion management, movement controls and vaccination (Anonymous 2005).

EHV-2 and EHV-5 mainly infect lymphoid cells where they have the capacity to establish latency. The clinical importance of these two viruses has not been clarified.

Diseases caused by these viruses are different to those caused by EHV-1 and EHV-4 and they do not share cross-protective antigens. EHV-3 is considered an uncommon venereal disease (Slater 2007). EHV-6 causes venereal disease in donkeys similar to EHV-3 (Browning et al. 1988) and is not considered to be of quarantine concern.

Equine rhinopneumonitis (EHV-1 and EHV-4) is an OIE-listed disease (OIE 2009b).

Epidemiology

Latently infected horses present the main reservoir of infection. The fragility of herpesviruses ensures that close contact is required for transmission. Transmission is through direct contact and contaminated fomites. Most adult horses show evidence of exposure to EHV-1, EHV-2, EHV-4 and EHV-5 (Slater 2007).

Herpesviruses commonly establish persistent latent infections which can recrudesce at times of stress (e.g. parturition, lactation) resulting in groups of mares and foals acting as reservoirs of virus for uninfected young horses (Gilkerson et al. 1999). These young horses later form a virus reservoir for the infection of subsequent generations.

Given the latent nature of herpesviruses, serological surveys of prevalence may be a poor indicator of true prevalence of infected animals. Various serological surveys have estimated prevalence in weaned foals at around 30% (Gilkerson et al. 1998), in brood mares at 26% and in unweaned foals at 11% (Gilkerson et al. 1999).

Clinical signs

EHV-1 is a respiratory pathogen that establishes a viraemia and systemic infection, manifested by abortion and neurological disease. EHV-4 is associated mainly with respiratory disease, and neurological disease due to this virus is rare. Both viruses usually cause self-limiting upper respiratory tract infections. However, immunocompromised or young animals can suffer from pneumonitis, complicated by secondary bacterial infection (Slater 2007). The incubation period for both viruses varies, and can be up to ten days before the appearance of respiratory signs. Infection in horses with previous exposure to virus is either subclinical or in the form of a mild respiratory illness (Slater 2007). Some horses develop a ‘poor performance syndrome’ on recovery, which can be associated with nonspecific bronchial hypersensitivity and a syndrome similar to recurrent airway obstruction (Slater 2007).

Pregnant mares and older horses show no apparent illness before abortion or the onset of neurological signs. Pregnant mares abort if infected in the last trimester of gestation — abortion does not occur if infection occurs before this. Neurological disease is uncommon, with clinical signs assumed to occur during or at the end of the viraemic phase. The prognosis is poor if the horse is recumbent as these animals progress to develop complications that require euthanasia (Slater 2007).

It is not clear what roles EHV-2 and EHV-5 play in clinical disease. Horses and foals testing positive to EHV-2 and EHV-5 do not usually show clinical signs of disease, and these viruses are ubiquitous in horse populations (Slater 2007). EHV-3 and EHV-6 cause a venereal disease and are the only EHVs that do not affect the respiratory tract.

Diagnosis

It is usually not possible to diagnose any of the EHV diseases based on clinical signs alone (Slater 2007). Isolation of virus, detection of viral antigens or nucleic acid, or

detection of antibody is required to confirm EHV disease (Slater 2007). Shedding of virus is short-lived and is most reliable for up to five days after infection. Direct immunofluorescent tests are used for detecting viral antigens and polymerase chain reaction tests for the detection of nucleic acid, which can distinguish between the various EHVs. Serological tests to detect antibody, which persist for more than nine months, include complement fixation, virus neutralisation, and enzyme-linked immunosorbent assays (Slater 2007).

Vaccines (live attenuated and inactivated) are available commercially (OIE 2008). However, vaccine-derived immunity is short-lived and revaccination at regular intervals is recommended (OIE 2008).

Conclusion

EHV-2 to EHV-9 inclusive are present in approved countries. EHV-6 and EHV-9 are not present in Australia. The viruses are not subject to official control in Australia. These equid herpesviruses were not considered further in the IRA.

EHV-1 is present in approved countries and in Australia. EHV-1 (abortigenic and neurological strains) is notifiable in Australia. The Code recommendations (OIE 2009a) include premises freedom from EHV-1. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Allen GP, Breathnach CC (2006) Quantification by real-time PCR of the magnitude and duration of leucocyte-associated viraemia in horses infected with neuropathogenic vs. non-neuropathogenic strains of EHV-1. *Equine Veterinary Journal* 38: 252-257.

Anonymous (2005) AEVA guide to management of EHV1 abortion. *Australian Equine Veterinarian* 15: 37-40.

Borchers K, Lieckfeldt D, Ludwig A, Fukushi H, Allen G, Fyumagwa R, Hoare R (2008) Detection of equid herpesvirus 9 DNA in the trigeminal ganglia of a Burchell's zebra from the Serengeti ecosystem. *Journal of Veterinary Medical Science* 70: 1377-1381.

Browning GF, Ficorilli N, Studdert MJ (1988) Asinine herpesvirus genomes: comparison with those of the equine herpesviruses. *Archives of Virology* 101: 183-190.

Davison AJ, Eberle R, Ehlers B, Hayward GS, McGeoch DJ, Minson AC, Pellett PE, Roizman B, Studdert MJ, Thiry E (2009) The order *Herpesvirales*. *Archives of Virology* 154: 171-177.

Davison AJ, Eberle R, Haywood GS, McGeoch DJ, Mihnson AC, Pellett PE, Roizman B, Studdert MJ, Thiry E (2005) Herpesviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 193-212. Elsevier, San Diego.

Fukushi H, Tomita T, Taniguchi A, Ochiai Y, Kirisawa R, Matsumura T, Yanai T, Masegi T, Yamaguchi T, Hirai K (1997) Gazelle herpesvirus 1: a new neurotropic herpesvirus immunologically related to equine herpesvirus 1 . *Virology* 227: 34-44.

Gilkerson JR, Love DN, Drummer HE, Studdert MJ, Whalley JM (1998) Seroprevalence of equine herpesvirus 1 in thoroughbred foals before and after weaning. *Australian Veterinary Journal* 76: 677-682.

Gilkerson JR, Whalley JM, Drummer HE, Studdert MJ, Love DN (1999) Epidemiology of EHV-1 and EHV-4 in the mare and foal populations on a Hunter Valley stud farm: are mares the source of EHV-1 for unweaned foals. *Veterinary Microbiology* 68: 27-34.

Kasem S, Yamada S, Kiupel M, Woodruff M, Ohya K, Fukushi H (2008) Equine herpesvirus type 9 in giraffe with encephalitis. *Emerging Infectious Diseases* 14: 1948-1949.

Nugent J, Birch-Machin I, Smith KC, Mumford JA, Swann Z, Newton JR, Bowden RJ, Allen GP, Davis-Poynter N (2006) Analysis of equid herpesvirus 1 strain variation reveals a point mutation of the DNA polymerase strongly associated with neuropathogenic versus nonneuropathogenic disease outbreaks. *The Journal of Virology* 80: 4047-4060.

OIE (2008) Equine rhinopneumonitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.09_EQUINE_RHINO.pdf (Accessed 1 September 2009).

OIE (2009a) Equine rhinopneumonitis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.9.htm (Accessed 19 August 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pellett PE, Roizman B (2007) The family Herpesviridae: a brief introduction. In *Fields virology* (eds. Knipe DM, Howley PM) pp. 2479-2499. Lippincott Williams & Wilkins, Philadelphia.

Schrenzel MD, Tucker TA, Donovan TA, Busch MDM, Wise AG, Maes RK, Kiupel M (2008) New hosts for equine herpesvirus 9. *Emerging Infectious Diseases* 14: 1616-1619.

Slater J (2007) Equine herpesvirus. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 135-151. Saunders Elsevier, St. Louis.

5.11 Equine encephalosis

5.11.1 Technical information

Background

Equine encephalosis, a mild or subclinical disease of equids in southern Africa, is caused by a virus belonging to the Orbivirus genus of the family Reoviridae (Mertens et al. 2005). Equine encephalosis virus (EEV) has seven known serotypes (1–7) and is closely related to African horse sickness (AHS) and bluetongue viruses.

First isolated from horses exhibiting neurological signs in the Republic of South Africa in 1967, EEV is endemic in South Africa, Zimbabwe and Kenya. The distribution of EEV is not clear because EEV produces mild, if any, clinical signs in endemic countries, and rarely death. Reports of its activity are due mainly to surveillance and laboratory capability to conduct tests. The host and vector distribution and environmental conditions suitable for transmission suggest the virus is widespread in sub-Saharan Africa. Most reports of EEV infection have been in southern Africa, and distribution north of the Sahara is not well documented. There was an outbreak in Israel in 2008 (Promed Mail 2009b) and serological evidence of the virus in horses in Ethiopia (Promed Mail 2009a).

Equine encephalosis is not an OIE-listed disease (OIE 2009).

Epidemiology

Epidemiological investigations implicate *Culicoides* spp. midges as vectors of EEV. Transmission in horses is seasonal in temperate parts of southern Africa with virus recovered during late summer and early autumn, when midges are active. Horses stabled at night did not become infected, when nocturnal insects were active (Erasmus et al. 1970). EEV has been shown experimentally to replicate in *C. imicola* (Venter et al. 1999) and *C. bolitinos* (Venter et al. 2002), and the epidemiological characteristics of EEV are very similar to that of AHS virus (Howell et al. 2004). Prevalence of antibody to EEV in endemic areas can be as high as 75–100% in adult horses, to 85% in donkeys and to 60% in zebras. Outbreaks of disease can occur, as in Israel in 2008, where climatic conditions favour vector activity and there is a population of naïve horses. Reports of vector spread into southern Europe (Purse et al. 2008) may affect the distribution of EEV.

Viraemia and pathogenesis have not been studied but are likely to be similar to other orbivirus infections such as AHS (Howell et al. 2004).

A vaccine has not been developed for equine encephalosis. No attempts have been made to control the disease except by stabling horses at night.

Clinical signs

Of the equids, horses are the only species known to show clinical signs of infection with EEV, with virus being recovered from horses of all breeds and ages. The incubation period is 3–6 days. Over 90% develop either no or very mild signs of infection that is marked by a slight rise in body temperature for 1–2 days. Obvious signs of infection include pyrexia, listlessness and inappetence for 1–5 days. Similar clinical signs were reported in 42 outbreaks in previously unexposed horses in Israel (OIE 2010). Severe cases may show severe swelling of the head, central nervous

system involvement, respiratory distress and heart failure. Death rarely follows, usually 6–8 days after neurological signs (Erasmus et al. 1978; Howell et al. 2004).

Diagnosis

As most infections are subclinical, active cases are identified by isolating virus from heparinised blood, while historical cases can be identified by antibody tests, such as the complement fixation test, indirect enzyme-linked immunosorbent assay (ELISA) or competitive ELISA (Erasmus et al. 1978; Williams et al. 1993; Howell et al. 2004). The OIE Reference Laboratory for AHS and bluetongue in Onderstepoort, South Africa has developed an indirect ELISA for the detection of EEV antibodies based on recombinant EEV VP7 and a reverse transcriptase polymerase chain reaction technique for identification of EEV (Promed Mail 2009a).

Conclusion

Equine encephalosis has not been reported in horses in any approved country. Accordingly, no further analysis was necessary.

However, equine encephalosis is an emerging disease and there is potential for spread to countries where the disease is not endemic but competent vectors are present. In the event that equine encephalosis is detected in equids in an approved country, Australia may implement provisional quarantine measures pending further investigation and assessment.

References

Erasmus BJ, Adelaar TF, Smit JD, Lecatsas G, Toms T (1970) The isolation and characterization of equine encephalosis virus. *Bulletin de l'Office International des Epizooties* 74: 781-789.

Erasmus BJ, Boshoff ST, Pieterse LM (1978) The isolation and characterization of equine encephalosis and serologically related orbiviruses from horses. In *Equine infectious IV* (eds. Bryans JT, Gerber H) pp. 447-450. Veterinary Publications, Princeton.

Howell PG, Guthrie AJ, Coetzer JAW (2004) Equine encephalosis. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 1247-1251. Oxford University Press, Oxford.

Mertens PPC, Maan S, Samuel A, Attoui H (2005) Orbivirus. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 466-483. Elsevier, San Diego.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2010) Equine encephalosis, Israel. World Animal Health Information System (WAHIS). http://www.oie.int/wahis/public.php?page=single_report&pop=1&reportid=7957 (Accessed 15 February 2010).

Promed Mail (2009a) Equine encephalosis - Ethiopia: serological evidence, request for information. ProMED Mail. http://www.promedmail.org (Accessed 16 July 2009).

Promed Mail (2009b) Equine encephalosis - Israel: 2008. ProMED Mail. http://www.promedmail.org (Accessed 16 July 2009).

Purse BV, Brown HE, Harrup L, Mertens PPC, Rogers DJ (2008) Invasion of bluetongue and other orbivirus infections into Europe: the role of biological and climatic processes. *Revue Scientifique et Technique de l'Office International des Epizooties* 27: 427-442.

Venter GJ, Groenewald D, Venter E, Hermanides KG, Howell PG (2002) A comparison of the vector competence of the biting midges, *Culicoides (Avaritia) bolitinos* and *C. (A.) imicola*, for the Bryanston serotype of equine encephalosis virus. *Medical and Veterinary Entomology* 16: 372-377.

Venter GJ, Groenewald DM, Paweska JT, Venter EH, Howell PG (1999) Vector competence of selected South African *Culicoides* species for the Bryanston serotype of equine encephalosis virus. *Medical and Veterinary Entomology* 13: 393-400.

Williams R, Du Plessis DH, Van Wyngaardt W (1993) Group-reactive ELISAs for detecting antibodies to African horsesickness and equine encephalosis in horse, donkey and zebra sera. *Journal of Veterinary Diagnostic Investigation* 5: 3-7.

5.12 Equine granulocytic anaplasmosis

5.12.1 Technical information

Background

Equine granulocytic anaplasmosis (EGA) (formerly equine granulocytic ehrlichiosis) is caused by *Anaplasma phagocytophilum* (formerly *Ehrlichia equi*). Molecular evidence demonstrated that the former species *Ehrlichia phagocytophila* (=*Cytoecetes phagocytophila*), *E. equi* and the agent causing human granulocytic ehrlichiosis belonged to the same species (Dumler et al. 2001). They were grouped as one species — *A. phagocytophila*. This was later changed in corrigendum to *A. phagocytophilum* (Judicial Commission 2002), which is currently the accepted taxonomic name (Dumler et al. 2005; Euzeby 2009).

A. phagocytophilum is an obligate intracellular organism within the order Rickettsiales. It is transmitted by ticks and causes disease in horses, humans (human granulocytic anaplasmosis), ruminants (tick-borne fever), dogs, cats, rodents, deer and other mammalian wildlife species (Dumler et al. 2001; Dumler et al. 2005). Infection with *A. phagocytophilum* has been reported in donkeys in Italy (de la Fuente et al. 2005). Given the wide mammalian host range, it is likely that infection of other equids does occur; however, there are few reports in the literature.

A. phagocytophilum is endemic in regions of Asia, Europe, North America, Russia and South America. Disease incidence is seasonal and corresponds with the host-seeking activity of the tick vector (Dumler et al. 2001; Dumler et al. 2005; Stuen 2007). The disease agent has not been reported in Australia, Belgium, Finland, Hong Kong, Ireland, Luxembourg, Macau, New Zealand, Singapore and United Arab Emirates.

EGA is not an OIE-listed disease (OIE 2009).

Epidemiology

EGA occurs seasonally where reservoirs of infection exist and susceptible horses are exposed to tick vectors infected with *A. phagocytophilum*. Reservoir hosts are primarily rodents and ruminants. Other mammals such as cats, dogs, horses, humans and white-tailed deer are sentinels for the presence of infection (Vredevoe et al. 1999; Liz et al. 2000; Bown et al. 2003; Dumler et al. 2005). Horses are considered aberrant hosts due to the absence of persistent infection (Pusterla and Madigan 2007).

Transmission to mammals occurs via the bite of an infected tick vector. *Ixodes* spp. are the principal biological vectors, the species of which varies depending on geographical location (Bown et al. 2003; Dumler et al. 2005; Rikihisa 2006). *Ixodes* spp. are present in Australia, mainly in coastal regions, and are likely to be capable of transmitting the disease.

A. phagocytophilum propagates within mammalian leukocytes and tick salivary glands. *Ixodes* spp. are three-host ticks and infection is maintained trans-stadially from one developmental stage of the tick to the next. This contributes to the wide mammalian host range of *A. phagocytophilum* because at each developmental stage the tick feeds on a different animal. Transovarial transmission between ticks does not

occur. Emergent larval ticks are not infectious until they have fed on an infected animal (Dumler et al. 2001; Bown et al. 2003; Hotopp et al. 2006; Stuen 2007).

Birds are recognised as potential reservoirs of ticks infected with *A. phagocytophilum*, especially in endemic regions. Migratory birds can potentially introduce infected ticks to areas free of *A. phagocytophilum* (Alekseev et al. 2001; Bjoersdorff et al. 2001; EFSA 2007; Ogden et al. 2008).

Natural direct transmission between mammals does not occur. Transmission is possible via inoculation of a susceptible horse with 20 ml blood from a horse with an active infection. Therefore, there is potential for iatrogenic transmission via blood transfusion or the use of blood-contaminated equipment (Pusterla and Madigan 2007; Radostits et al. 2007).

Prevalence is seasonal correlating directly with the peak period of mammalian blood-feeding activity of nymph and adult ticks. Seroprevalence in horses in California has been reported to be 3–18%. One farm in northern California recorded a seroprevalence of 50% in healthy horses (Madigan et al. 1990; Pusterla and Madigan 2007). In southern France (Camargue), a seroprevalence of 11.3% was reported in 424 horses. In this study, the prevalence of infection was greater in horses stabled in close proximity to wild birds (Leblond et al. 2005).

Genetic diversity exists between isolates of *A. phagocytophilum* from different geographical regions and host species (Radostits et al. 2007). Isolates also vary in mammalian host specificity and pathogenicity. The incidence of subclinical infection in horses is greater in areas where tick-borne fever of ruminants is endemic. Experimental infection of horses with an isolate that is pathogenic to ruminants resulted in seroconversion in horses in the absence of disease (Pusterla et al. 1998). When naïve horses are introduced to an endemic region, they are more likely to develop EGA than horses native to endemic regions. Immunity persists in horses for at least two years, and a carrier status does not occur (Pusterla and Madigan 2007).

Clinical signs

The incubation period and severity of clinical signs varies with age, immune status and species of infected animal, as well as infective dose and isolate. The incubation period ranges from 10 to 20 days and signs of disease vary from subclinical to severe.

In horses greater than four years of age, the disease usually presents with progressive onset of pyrexia, depression, partial anorexia, distal limb oedema, petechiation, icterus, ataxia and a reluctance to move. Horses less than four years of age display less severe signs of disease, and pyrexia may be the only sign in horses less than one year of age. Clinical pathology includes leukopaenia, thrombocytopaenia, and the presence of inclusion bodies (morulae) within neutrophils. Morulae are occasionally seen in eosinophils and macrophages (Pusterla and Madigan 2007).

The duration of disease is 3–16 days (Pusterla and Madigan 2007). Immune suppression occurs in infected animals, often resulting in increased susceptibility to secondary infection by opportunistic pathogens. Provided there is no concurrent infection, the disease in untreated horses resolves in 2–3 weeks (Pusterla and Madigan 2007). Mortalities are rare and usually due to secondary infection or injury sustained as a result of ataxia (Madigan and Pusterla 2000).

Diagnosis

Diagnosis of EGA is based on history of exposure to an endemic area, typical clinical signs of disease, haematological findings, and identification of typical morulae in neutrophils in a peripheral blood smear (Dumler et al. 2007; Radostits et al. 2007).

Detection of morulae can be increased by preparing smears using the buffy coat, or examining serial blood smears collected over several days after the onset of clinical disease (Franzen et al. 2005; Dumler et al. 2007; Pusterla and Madigan 2007). Morulae are usually detectable after the onset of pyrexia and remain in blood smears for 1–2 weeks. They have been seen in up to 50% of neutrophils by five days post-infection (Pusterla and Madigan 2007).

Clinical signs of EGA are not pathognomonic, and in some regions, infection of horses with *A. phagocytophilum* occurs without causing overt disease (Radostits et al. 2007). Several polymerase chain reaction (PCR) assays have been developed for *A. phagocytophilum*, some of which are considered highly sensitive and specific (Massung and Slater 2003; Pusterla and Madigan 2007). Molecular analysis using PCR assays is most useful during the early or later stages of infection, when the number of organisms in the blood is too low for detection of morulae in smears (Pusterla and Madigan 2007). The results of PCR analysis from horses experimentally infected with the European strain of *A. phagocytophilum* were positive from day five to day 21 post-infection (end of the study period). Results were positive in all horses 2–3 days before the onset of pyrexia and 4–7 days before morulae were detected in blood smears (Franzen et al. 2005). PCR analysis can also be used to provide molecular confirmation of the identity of the organisms within the morulae.

A. phagocytophilum can be cultured from whole (unclotted) blood or the buffy coat. Antibiotic-free cell culture methods are required with 2–6 weeks incubation before infected cells can be detected. Cultures are monitored 2–3 times per week by examining cells for the presence of morulae in intracytoplasmic inclusions (Dumler et al. 2007). Due to the specialised methods required for culture and identification, it is rarely attempted for infections in horses (Pusterla and Madigan 2007).

Serological tests are useful to indicate possible exposure to *A. phagocytophilum*. An indirect fluorescent antibody test using antigen prepared from an infected horse has been described (Nyindo et al. 1978). All strains of *A. phagocytophilum* cross-react serologically and share antigens with *A. marginale* (endemic in Australia), *Ehrlichia canis*, *E. chaffeensis*, and *E. ruminantium* (Strik et al. 2007).

Antibody titres in naturally infected horses peak 19–81 days after the onset of clinical signs (Van Andel et al. 1998). Paired-titre testing with a fourfold (or greater) increase in antibody titre to *A. phagocytophilum* suggests recent exposure (Madigan et al. 1990). During the acute phase of disease, demonstration of morulae in blood smears is a more reliable method of detection than serology (Artursson et al. 1999).

Treatment

Tetracyclines are effective against *A. phagocytophilum*. Daily intravenous administration of oxytetracycline for 5–7 days is recommended for horses. Improvement in clinical condition is usually seen within 12–24 hours after the onset of treatment (Radostits et al. 2007). In rare cases, horses treated for less than seven days can have a relapse of disease within 30 days after completion of treatment (Pusterla and Madigan 2007). *A. phagocytophilum* has also been shown to be

susceptible to rifamycins and some fluoroquinolones (Klein et al. 1997; Dumler et al. 2005).

Conclusion

EGA is present in some approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.12.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of EGA being present in an imported horse.

- *A. phagocytophilum* has a wide distribution and occurs seasonally when tick vectors feed on horses.
- Seroprevalence of up to 50% has been reported in horses in endemic areas (Madigan et al. 1990; Pusterla and Madigan 2007).
- Subclinical infections are common and milder signs of disease occur in horses less than four years old.
- The incubation period is 10–20 days.

Based on these considerations, the likelihood of release of EGA associated with horses from a country where the disease is present is estimated to be 'moderate'**.**

Exposure assessment

The most likely pathway for exposure is importation of an infected horse into a region where *Ixodes* spp. are endemic and able to feed on the imported horse, then transmit infection to other exposure groups.

Exposure groups considered are equids (including feral equids), other domestic species (including other non-ruminants and ruminants, feral animals) and wildlife.

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to *A. phagocytophilum* via an imported horse.

- Transmission to mammals occurs via the bite of *Ixodes* spp. ticks. Australia has potential vectors located mainly in coastal regions.
- Trans-stadial transmission occurs in ticks. Once infected, ticks transmit infection to other susceptible animals on which they feed during their lifetime.
- Horses are infectious for a period of 1–2 weeks when *A. phagocytophilum* is present in peripheral blood.
- *A. phagocytophilum* has a wide mammalian host range.

Based on these considerations, the likelihood of susceptible animals being exposed to EGA via an infected imported horse was estimated to be '*low*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure involves consideration of the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*moderate*' combined with the likelihood of exposure estimated to be '*low*', the likelihood of release and exposure for EGA was estimated to be ***'low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of a susceptible animal has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to EGA is considered to be establishment and/or spread to populations of susceptible animals within a region and potentially spread to different regions and more than one state, through the movement of infected horses to regions where *Ixodes* spp. tick vectors are endemic and actively feeding.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to EGA.

- Susceptible animals include domestic and feral animals (cats, dogs, equids, rodents and ruminants), wildlife and humans.
- Transmission and spread may occur in regions where *Ixodes* spp. are endemic and feed on susceptible animals. Movement of infected animals (domestic, feral and wild) between these regions could contribute to the spread of infection.
- Trans-stadial transmission occurs in ticks, and *Ixodes* spp. are three-host ticks. Once infected, the tick transmits infection to each susceptible animal it bites during its lifetime.
- Subclinical infection is common, especially in reservoir hosts. Clinical signs of disease are not pathognomonic, especially in horses less than four years old. Also as increased susceptibility to secondary infections can mask signs of infection, detection is likely to be delayed.

- Given the widespread movement of animals within Australia, infection is most likely to spread to more than one region where *Ixodes* spp. are endemic, and potentially to more than one state before detection.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of EGA was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of EGA for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Infection with *A. phagocytophilum* causes clinical disease and/or loss of productivity in susceptible animals.
- Morbidity is variable depending on the isolate and infected species. Naïve animals are more likely to develop clinical signs of disease than animals from endemic areas. Isolates from different geographical regions differ in species specificity and pathogenicity.
- Immune suppression occurs in infected animals, often resulting in increased susceptibility to secondary infection by opportunistic pathogens.
- Case fatalities associated with EGA are rare, usually from secondary infection or injury sustained as a result of ataxia.
- EGA is a zoonotic disease.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- It is not known if Australian wildlife would be susceptible to disease. In areas where *A. phagocytophilum* is endemic, reservoir hosts include wild rodents and ruminants, in which subclinical infection is common.
- EGA is not considered to have any direct effects on the non-living environment.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to *minor at the local level* (national effect score B in Table 3.4).

Indirect effects

The effects on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Disease caused by *A. phagocytophilum* is not notifiable in Australia, there is no AUSVETPLAN Disease Strategy Manual for EGA and the disease is not scheduled in Australia's Emergency Animal Disease Response Agreement; however, a combination of strategies is used to control exotic disease incursions and minimise the effects on animal and human health, trade and the environment.
- Control measures could include tracing and surveillance, movement controls on animals, tick control, treatment of infected animals (including humans) and a public awareness campaign to encourage cooperation from industry.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion were estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- *A. phagocytophilum* is multi-species zoonotic pathogen and would have an effect on domestic trade and industries associated with susceptible animals.
- There would be productivity losses, increased costs and operational procedures associated with implementing control measures.
- An incursion of a zoonotic tick-borne pathogen could also have a detrimental effect on tourism in affected rural and regional communities. Resources would be required to manage the public health issues.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- If *A. phagocytophilum* were detected in Australia, there may be some disruption to exports of susceptible animals to countries in which *A. phagocytophilum* is exotic, and suitable tick vectors are endemic.
- If the disease could not be eradicated, zoning may be an option to maintain export market access for affected industry sectors.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- In areas where *A. phagocytophilum* is endemic, reservoirs include wild rodents and ruminants, in which subclinical infection is common.
- EGA has a wide mammalian host range however it is not known if Australian wildlife would be susceptible to disease.
- Increased use of acaricides to control ticks could have an effect on a range of arthropod species and disrupt the food source of wildlife, lead to environmental contamination (including water sources) and resistance to acaricides.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to *minor at the local level* (national effect score B in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- There may be some minor disruption to horse, dog and ruminant events, for example movement restrictions and treatment of animals for ticks.
- Where susceptible animals are important to the local economy, the viability of communities within affected regions may be affected.
- Public concerns of a zoonotic disease may have a detrimental effect on tourism in affected rural and regional communities.

Based on these considerations, the effect of the establishment and/or spread of EGA in Australia for this criterion was estimated to significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is 'D', the overall effect associated with the outbreak scenario is considered to be '*low*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*low*') which results in '***low***' likely consequences.

Risk estimation

Risk estimation is the integration of likelihood of release and exposure and likely consequences of establishment or spread to derive the risk associated with release, exposure, establishment and/or spread of EGA introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure (‘***low***’**)** is combined with the likely consequences of establishment and/or spread (‘***low***’) which results in a risk estimation of **VERY LOW**.

Conclusion

The unrestricted risk associated with EGA is determined to be **VERY LOW**. As the unrestricted risk estimate achieves Australia’s ALOP, no risk management is considered necessary.

A summary of the risk assessment for EGA is shown in Figure 5.2 and Table 5.2.

Figure 5.2 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for EGA.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Moderate*
Likelihood of *exposure*	Likelihood of *exposure*	*Low*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Low*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Low***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**VERY LOW**

Table 5.2 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for EGA.

References

Alekseev AN, Dubinina HV, Semenov AV, Bolshakov CV (2001) Evidence of ehrlichiosis agents found in ticks (Acari: Ixodidae) collected from migratory birds. *Journal of Medical Entomology* 38: 471-474.

Artursson K, Gunnarsson A, Wikstrom U-B, Engvall EO (1999) A serological and clinical follow-up in horses with confirmed equine granulocytic ehrlichiosis. *Equine Veterinary Journal* 31: 473-477.

Bjoersdorff A, Bergstrom S, Massung RF, Haemig PD, Olsen B (2001) *Ehrlichia*-infected ticks on migrating birds. *Emerging Infectious Diseases* 7: 877-879.

Bown KJ, Begon M, Bennett M, Woldehiwet Z, Ogden NH (2003) Seasonal dynamics of *Anaplasma phagocytophila* in a rodent-tick (*Ixodes trianguliceps*) system, United Kingdom. *Emerging Infectious Diseases* 9: 63-70.

de la Fuente J, Torina A, Caracappa S, Tumino G, Furla R, Almazan C, Kocan KM (2005) Serologic and molecular characterization of *Anaplasma* species infection in farm animals and ticks from Sicily. *Veterinary Parasitology* 133: 357-362.

Dumler JS, Barbet AF, Bekker CPJ, Dasch GA, Palmer GH, Ray SC, Rikihisa Y, Rurangirwa FR (2001) Reorganization of genera in the families *Rickettsiaceae* and

Anaplasmataceae in the order *Rickettsiales*: unification of some species of *Ehrlichia* with *Anaplasma*, *Cowdria* with *Ehrlichia* and *Ehrlichia* with *Neorickettsia*, descriptions of six new species combinations and designation of *Ehrlichia equi* and 'HGE agent' as subjective synonyms of *Ehrlichia phagocytophila*. *International Journal of Systematic and Evolutionary Microbiology* 51: 2145-2165.

Dumler JS, Madigan JE, Pusterla N, Bakken JS (2007) Ehrlichiosis in humans: epidemiology, clinical presentation, diagnosis, and treatment. *Clinical Infectious Diseases* 45: S45-S51.

Dumler JS, Rikihisa Y, Dasch GA (2005) Genus I. *Anaplasma* Theiler 1910, 7^{AL} emend. Dumler, Barbet, Bekker, Dasch, Palmer, Ray, Rikihisa and Rurangirwa 2001, 2157. In *Bergey's manual of systematic bacteriology* (ed. Garrity GM) pp. 117-124. Springer-Verlag, Wien, New York.

EFSA (2007) Scientific opinion on an assessment of the risk of tick introduction into UK, Republic of Ireland, and Malta as a consequence of abandoning national rules. *The EFSA Journal* 469: 1-102.

Euzeby JP (2009) List of prokaryotic names with standing in nomenclature (LPSN). International Journal of Systematic Bacteriology. http://www.bacterio.cict.fr/ (Accessed 16 January 2009).

Franzen P, Aspan A, Egenvall A, Gunnarsson A, Aberg L, Pringle J (2005) Acute clinical, hematologic, serologic, and polymerase chain reaction findings in horses experimentally infected with a European strain of Anaplasma phagocytophilum. *Journal of Veterinary Internal Medicine* 19: 232-239.

Hotopp JCD, Mingqun L, Madupu R, Crabtree J, Angiuoli SV, Eisen J, Seshadri R, Ren Q, Wu M, Utterback TR, Smith S, Lewis M, Khouri H, Zhang C, Niu H, Lin Q, Ohashi N, Zhi N, Nelson W, Brinkac LM, Dodson RJ, Rosovitz MJ, Sundaram J, Daughtery SC, Davidsen T, Durkin AS, Gwinn M, Haft DH, Selengut JD, Sullivan SA, Zafar N, Zhou L, Benahmed F, Forberger H, Halpin R, Mulligan S, Robinson J, White O, Rikihisa Y, Tettelin H (2006) Comparative genomics of emerging human ehrlichiosis agents. *PLoS Genetics* 2: 208-223.

Judicial Commission (2002) Notification that new names and new combinations have appeared in volume 51, part 6, of the IJSEM. *International Journal of Systematic and Evolutionary Microbiology* 52: 5-6.

Klein MB, Nelson CM, Goodman JL (1997) Antibiotic susceptibility of the newly cultivated of human granulocytic ehrlichiosis: promising activity of quinolones and rifamycins. *Antimicrobial Agents and Chemotherapy* 41: 76-79.

Leblond A, Pradier S, Pitel PH, Fortier G, Boireau P, Chadoeuf J, Sabatier P (2005) An epidemiological survey of equine anaplasmosis (*Anaplasma phagocytophilum*) in southern France. *Revue Scientifique et Technique de l'Office International des Epizooties* 24: 899-908.

Liz JS, Anderes L, Sumner JW, Massung RF, Gern L, Rutti B, Brossard M (2000) PCR detection of granulocytic ehrlichiae in *Ixodes ricinus* ticks and wild small mammals in western Switzerland. *Journal of Clinical Microbiology* 38: 1002-1007.

Madigan JE, Hietala S, Chalmers S, DeRock E (1990) Seroepidemiologic survey of antibodies to *Erhlichia equi* in horses of northern California. *Journal of the American Veterinary Medical Association* 196: 1962-1964.

Madigan JE, Pusterla N (2000) Ehrlichial diseases. *Veterinary Clinics of North America: Equine Practice* 16: 487-499.

Massung RF, Slater KG (2003) Comparison of PCR assays for detection of the agent of human granulocytic ehrlichiosis, *Anaplasma phagocytophilum*. *Journal of Clinical Microbiology* 41: 717-722.

Nyindo MB, Ristic M, Lewis GE, Huxsoll DL, Stephenson EH (1978) Immune response of ponies to experimental infection with *Ehrlichia equi*. *American Journal of Veterinary Research* 39: 15-18.

Ogden NH, Lindsay LR, Hanincova K, Barker IK, Bigras-Poulin M, Charron DF, Heagy A, Francis CM, O'Callaghan CJ, Schwartz I, Thompson RA (2008) Role of migratory birds in introduction and range expansion of *Ixodes scapularis* ticks and of *Borrelia burgdorferi* and *Anaplasma phagocytophilum* in Canada. *Applied and Environmental Microbiology* 74: 1780-1790.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pusterla N, Lutz H, Braun U (1998) Experimental infection of four horses with *Ehrlichia phagocytophila*. *The Veterinary Record* 143: 303-305.

Pusterla N, Madigan JE (2007) *Anaplasma phagocytophila*. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 354-357. Saunders Elsevier, St. Louis.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with Rickettsiales. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1455-1470. Saunders Elsevier, Edinburgh.

Rikihisa Y (2006) New findings on members of the family *Anaplasmataceae* of veterinary importance. *Annals of the New York Academy of Sciences* 1078: 438-445.

Strik NI, Alleman AR, Barbet AF, Sorenson HL, Wamsley HL, Gaschen FP, Luckschander N, Wong S, Chu F, Foley JE, Bjoersdorff A, Stuen S, Knowles DP (2007) Characterization of *Anaplasma phagocytophilum* major surface protein 5 and the extent of its cross-reactivity with *A. marginale*. *Clinical and Vaccine Immunology* 14: 262-268.

Stuen S (2007) *Anaplasma phagocytophilum*: the most widespread tick-borne infection in animals in Europe. *Veterinary Research Communications* 31: 79-84.

Van Andel AE, Magnarelli LA, Heimer R, Wilson ML (1998) Development and duration of antibody response against *Ehrlichia equi* in horses. *Journal of the American Medical Association* 212: 1910-1914.

Vredevoe LK, Richter PJ, Madigan JE, Kimsey RB (1999) Association of *Ixodes pacificus* (Acari: Ixodidae) with the spatial and temporal distribution of equine granulocytic ehrlichiosis in California. *Journal of Medical Entomology* 36: 551-561.

5.13 Equine infectious anaemia

5.13.1 Technical information

Background

Equine infectious anaemia (EIA), a debilitating disease of equids worldwide, is caused by a virus belonging to the lentivirus genus of the family Retroviridae (Linial et al. 2005).

EIA, first described in France in 1843, is also called 'swamp fever' due to its prevalence in horses in low-lying swampy areas of warm wet regions such as the Gulf Coast states of the United States and along the inland river systems of central and western Queensland. Serological surveys and clinical surveillance have indicated that EIA is extremely uncommon in other parts of Australia. In Australia, there were 12 761 routine blood sample submissions to State laboratories testing for EIA between January 2004 and December 2008. Of these, only 48 (3.7%) were positive and all were from Queensland. The disease has never been identified in South Australia, Tasmania or Western Australia (Animal Health Australia 2009). EIA is widespread around the world. In Europe, EIA is endemic in Romania; although sporadic outbreaks have been reported in Croatia, Italy, France and Greece (OIE 2008). An outbreak, since eradicated, was also reported in Ireland in 2006 (More et al. 2008a). Many countries implement control and eradication programs for EIA.

Although endemic in parts of Queensland, EIA is a notifiable disease in Australia (DAFF 2008).

EIA is an OIE-listed disease (OIE 2009b).

Epidemiology

Horses exposed to EIA virus generally develop detectable immune responses to the viral antigen within 37 days, although sometimes this may take more than 60 days, following infection (Cullinane et al. 2007). Despite developing an immune response, horses infected with EIA become inapparent carriers that remain infective for life.

EIA is a typical blood-borne infection and the recognised routes of transmission are by the mechanical transfer of blood by biting flies, veterinary instruments or plasma (Leroux et al. 2004; More et al. 2008a). However, finding virus in body fluids other than blood (Quinlivan et al. 2007) suggests other forms of transmission are possible (More et al. 2008b).

There is no vaccine for EIA. Control involves preventing direct or indirect contact with secretions, excretions and blood or plasma of infected horses.

Clinical signs

The incubation period is usually 1–3 weeks but can be as long as three months (Cheevers and McGuire 1985). The clinical course of EIA is variable, depending on the dose and virulence of the virus strain and the susceptibility of the horse. Although distinctions are not absolute, three characteristic clinical stages of EIA have been described (Mealy 2007).

On initial infection, horses may develop severe acute non-specific clinical disease and die within 2–3 weeks. Clinical signs of EIA include severe pyrexia, anaemia, swelling of the abdomen and legs, but are rarely seen as blood-feeding flies generally transmit low doses of virus. Thrombocytopaenia is often the first abnormality observed in pyrexic horses. Most cases are mild with pyrexia lasting only 24 hours and can be missed by horse owners and veterinarians. A chronic form, caused by new mutant EIA virus strains generated within the infected horse, can be characterised by recurrent bouts of mild pyrexia, anaemia, weight loss and lethargy (Leroux et al. 2004).

As EIA virus is characterised by rapid viral replication and antigenic variation, most horses progress from a chronic stage with recurring peaks of viraemia and pyrexia to a subclinical infection (Leroux et al. 2004).

Diagnosis

Early diagnosis may be difficult because serologic tests can be negative 10–14 days after infection (Coggins et al. 1972). Diagnostic tests such as the agar gel immunodiffusion test (also known as the Coggins test), enzyme-linked immunosorbent assay and polymerase chain reaction assay, used alone or in combination, identify infected horses which are then either isolated or euthanased in efforts to control the spread of disease (Brangan et al. 2008).

Conclusion

EIA is present in some approved countries and in parts of Australia. The disease is nationally notifiable in Australia. The Code recommendations (OIE 2009a) include premises freedom and diagnostic testing. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Animal Health Australia (2009) Equine infectious anaemia: serological testing for equine infectious anaemia from routine submission to state laboratories (summary). National Animal Health Information System (NAHIS). http://www.animalhealthaustralia.com.au/nahis/public.php?page=out_showtable&outputid=32&sm=1&sy=2004&em=9&ey=2008&date_submit=OK (Accessed 10 March 2009).

Brangan P, Bailey DC, Larkin JF, Myers T, More SJ (2008) Management of the national programme to eradicate equine infectious anaemia from Ireland during 2006: a review. *Equine Veterinary Journal* 40: 702-704.

Cheevers WP, McGuire TC (1985) Equine infectious anemia virus: immunopathogenesis and persistence. *Reviews of Infectious Diseases* 7: 83-88.

Coggins L, Norcross NL, Nusbaum SR (1972) Diagnosis of equine infectious anemia by immunodiffusion test. *American Journal of Veterinary Research* 33: 11-18.

Cullinane A, Quinlivan M, Nelly M, Patterson H, Kenna R, Garvey M, Gildea S, Lyons P, Flynn M, Galvin P, Neylon M, Jankowska K (2007) Diagnosis of equine infectious anaemia during the 2006 outbreak in Ireland. *The Veterinary Record* 161: 647-652.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

Leroux C, Cadore J-L, Montelaro RC (2004) Equine infectious anemia virus (EIAV): what has HIV's country cousin got to tell us? *Veterinary Research* 35: 485-512.

Linial ML, Fan H, Hahn B, Lwer R, Neil J, Quackenbush S, Rethwilm A, Sonigo P, Stoye J, Tristem M (2005) Retroviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 421-440. Elsevier, San Diego.

Mealy RH (2007) Equine infectious anemia. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 213-219. Saunders Elsevier, St. Louis.

More SJ, Aznar I, Bailey DC, Larkin JF, Leadon DP, Lenihan P, Flaherty B, Fogarty U, Brangan P (2008a) An outbreak of equine infectious anaemia in Ireland during 2006: investigation methodology, initial source of infection, diagnosis and clinical presentation, modes of transmission and spread in the Meath cluster. *Equine Veterinary Journal* 40: 706-708.

More SJ, Aznar I, Myers T, Leadon DP, Clegg TA (2008b) An outbreak of equine infectious anaemia in Ireland during 2006: the modes of transmission and spread in the Kildare cluster. *Equine Veterinary Journal* 40: 709-711.

OIE (2008) WAHID interface. World Animal Health Information Database: version 1.2. http://www.oie.int/wahid-prod/public.php?page=home (Accessed 14 November 2008).

OIE (2009a) Equine infectious anaemia. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.6.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Quinlivan M, Cook RF, Cullinane A (2007) Real-time quantitative RT-PCR and PCR assays for a novel European field isolate of equine infectious anaemia virus based on sequence determination of the gag gene. *The Veterinary Record* 160: 611-618.

5.14 Equine influenza

5.14.1 Technical information

Background

Equine influenza (EI) is an acute respiratory disease of equids caused by equine influenza virus (EIV).

EIV is an influenza A virus of the family Orthomyxoviridae (genus Influenzavirus A). Influenza A viruses are further subtyped by haemagglutinin (H) and neuraminidase (N) envelope glycoproteins. Subtypes H7N7 and H3N8 (formerly designated equine 1 and equine 2 viruses, respectively) cause outbreaks of respiratory disease in horses (Kawaoka et al. 2005). The H7N7 subtype (A/eq/Prague/56) has not been isolated since 1980 (Webster 1993) though serological evidence of virus presence was recorded in eastern Europe in 1996 (Madic et al. 1996), but this antibody may be entirely of vaccine origin (Guo et al. 1995).

EIV is not considered zoonotic. Experimental infection with equine H3N8 viruses has produced mild influenza-like illness and seroconversion in humans (Kasel et al. 1965; Kasel and Couch 1969). However, transmission of EIV to humans under natural conditions of exposure has not occurred during outbreaks in horses (McQueen et al. 1966; Davenport et al. 1967).

Serious outbreaks of equine H3N8 have occurred in equine populations previously free of disease. Importation of horses infected with EI was believed to cause an outbreak in South Africa in 1986 and India in 1987. Outbreaks also occurred in Hong Kong in 1992, Dubai in 1995 and the Philippines in 1997 (Daly et al. 2004a).

Australia was free of EI until August 2007, when the disease was introduced with imported horses. The causative virus, called A/equine/Sydney/07 H3N8, was almost identical to viruses causing an outbreak in Japan in August 2007 and in Pennsylvania in late August 2007 (Newton 2008). EI was subsequently eradicated from Australia, with the last case on 25 December 2007 (DAFF 2008). Iceland and New Zealand are the only countries with substantial equine populations never to have reported EI (OIE 2008a).

EI is an OIE-listed disease (OIE 2009c).

Agent properties

Influenza A viruses have a lipid envelope, are 80–120 nm in diameter and contain a genome of eight segments of single-stranded, negative-sense RNA (Landolt et al. 2007).

Influenza viruses can persist and remain infective on skin, fabrics and contaminated equipment. At 35–40% relative humidity and a temperature of 28 °C, influenza A viruses persist on hard, nonporous surfaces such as stainless steel and plastic for 24–48 hours, but for less than 8–12 hours on porous surfaces such as cloth and paper (Bean et al. 1982). Influenza A virus was transferred from stainless steel surfaces to hands for up to 24 hours and from paper tissues to hands for up to 15 minutes, with virus persisting on hands for up to five minutes after transfer from environmental surfaces (Bean et al. 1982). Influenza virus remains infective for longer periods at

lower relative humidity than at higher humidity (Loosli et al. 1943; Hemmes et al. 1960).

EIV remains viable in tap water (pH 7.0) for 14 days at 4 °C and up to two days at 37 °C, canal water (pH 6.9) up to 18 days at 22 °C and for 14 days at 37 °C, horse urine (pH 8.0) for 5–6 days at 4 °C, 15 °C or 37 °C, horse blood for 18 hours at 37 °C, soil under dark storage at 18 °C for 24 hours, and in soil under sunlight at 15 °C for eight hours (Yadav et al. 1993).

Influenza viruses are inactivated by a range of disinfectants and chemicals, including alcohol, chloroxylenol, phenolics, quaternary ammonium compounds, oxidising agents (e.g. Virkon®) and detergents (Yadav et al. 1993; Prince and Prince 2001; Animal Health Australia 2007). EIV is sensitive to heat (50 °C, 30 minutes), exposure to sunlight at 15 °C for 15 minutes, ether and acid (pH 3), although 2% sodium carbonate was ineffective after 60 minutes exposure at 32 °C (Yadav et al. 1993).

Influenza viruses are protected in the presence of organic matter which enhances resistance to physical and chemical inactivation. Organic material should be removed so disinfectants can work optimally (Swayne and Halvorson 2003).

The surfactant action of soaps and detergents is an effective decontaminant for EIV because it destroys the outer lipid envelope of the virus. Soap and water or alcohol-based hand rubs are satisfactory for personal disinfection against influenza viruses (Grayson et al. 2009).

Inactivation trials on avian influenza virus using chlorine levels typical of drinking water showed that a free chlorine residual (0.52–1.08 mg/L) was sufficient to inactivate virus by more than three orders of magnitude within an exposure time of one minute (Rice et al. 2007).

Epidemiology

EIV is highly contagious and endemic in horse populations in most countries. All ages and breeds of equids can be infected with virus experimentally (Nyaga et al. 1980). In endemic countries, outbreaks can occur at any time of year and timing probably depends on husbandry and management factors (Radostits et al. 2007). There is little information available on prevalence and incidence in endemic populations, or on vaccine usage in populations outside regular competition.

In susceptible populations, explosive outbreaks of EI can occur with clinical disease in nearly all exposed horses (Radostits et al. 2007), as was seen when EI entered the largely naïve Australian horse population. In populations of horses of mixed ages and various serum titres to EIV, the rate of clinical disease during outbreaks may be much lower (16–28%), with epidemics in large groups of stabled horses at a racetrack lasting approximately one month (Morley et al. 2000a). The case fatality rate from EI is generally less than 1% with most deaths due to secondary bacterial infection (Radostits et al. 2007).

Transmission of EIV can occur by aerosol inhalation, direct contact and via fomites (Radostits et al. 2007). Aerosol spread of droplet-borne EIV from an infected horse is reported over at least 32 metres (Miller 1965) and infection by aerosol facilitates spread within closed groups (Radostits et al. 2007). On a few occasions, transmission over longer distances has been attributed to windborne spread due to the apparent absence of other obvious routes of transmission. Windborne spread of up to 8 km has anecdotally been reported from a group of infected horses (Huntington 1990).

Similarly, spread of EI between infected premises over 1–2 km apart, possibly consistent with windborne aerosol, was described in the 2007 Australian EI outbreak (Davis et al. 2009) but the possibility of alternative routes of transmission could not be definitely ruled out (EI Epidemiology Support Group 2009).

The importance of transmission of EIV by human contact and fomites was described in a naïve population during the 1986 South African outbreak (Guthrie et al. 1999). Indirect spread was also thought to be responsible for transmission of EI from imported horses to local horses in the 2003 outbreak in South Africa (King and Macdonald 2004; Guthrie 2006). Persistence of virus in moist, partially protected conditions (e.g. transport vehicles, tack, veterinary equipment and clothing) can result in transmission of EI in the absence of aerosol exposure.

EI has a short incubation period, ensuring rapid transmission between susceptible horses. Naïve, experimentally infected horses show clinical signs of disease as early as two days after infection, lasting up to ten days, and virus is shed from within 48 hours of infection for up to seven days (Soboll et al. 2003; Crouch et al. 2004; Heldens et al. 2004; Edlund Toulemonde et al. 2005).

Vaccination can significantly reduce both the clinical signs of EI and the extent of viral shedding in both horses and ponies. However, viral shedding can occur in vaccinated horses in the absence of clinical signs of disease (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007). In experimentally infected, vaccinated horses, the duration of viral shedding was up to four days (Soboll et al. 2003; Heldens et al. 2004; Crouch et al. 2004; Edlund Toulemonde et al. 2005).

Susceptibility to reinfection and the presence of mild clinical signs in infected vaccinated horses can make diagnosis difficult and this has been a major contributor to the spread of infection to susceptible populations (Hannant and Mumford 1996). In Hong Kong in 1992, EIV infection remained unrecognised among imported vaccinated horses that underwent a 14-day post-arrival quarantine period (Powell et al. 1995). Retrospective investigation suggested EIV escaped quarantine and infection occurred in a large proportion of the vaccinated local population; clinical signs of disease were not reported in Hong Kong until 25 to 32 days after importation (Powell et al. 1995).

No species other than equids is known to play a significant role in the epidemiology of EI. Transmission of EIV to dogs was reported in 2004 in racing greyhounds in Florida and subsequent horizontal spread of canine-adapted influenza within the dog population has been reported in the United States (Crawford et al. 2005; Beeler 2009). EI has also been reported to infect dogs in the United Kingdom (Daly et al. 2008; Newton et al. 2009) and experimentally infected horses were able to infect dogs in close contact, although no dogs in this study showed clinical signs of disease (Yamanaka et al. 2009). EI infection of dogs was reported during the 2007 EI outbreak in Australia; no serological evidence of dog-to-dog transmission was present in the infected population (EI Epidemiology Support Group 2009). There is no evidence of natural transmission of influenza virus from dogs to horses.

Sequencing of A/equine/Jilin/89 (H3N8), which emerged in northeast China in 1989 with high morbidity and mortality in horses, revealed a genome dissimilar to that of known equine viruses but similar to some of recent avian origin. This implied the

possibility of transfer of an avian virus direct to horses without reassortment (Guo et al. 1992).

Clinical signs

In horses, the incubation period is 1–4 days (Park et al. 2004) after which there is onset of pyrexia (38.5–41 °C) peaking at 48 to 96 hours after infection (Landolt et al. 2007; Radostits et al. 2007). Shortly after the onset of pyrexia, a dry and hacking cough develops and can last for 1–3 weeks. Serous nasal discharge can occur and can become mucopurulent. Most horses recover in 7–14 days, although the cough can persist for weeks (Radostits et al. 2007). Isolation and rest of affected horses assists in recovery and reduces spread of EIV among other susceptible horses.

In an EI outbreak there is a range of disease severity (Morley et al. 2000b). Clinical signs of disease in vaccinated and previously infected animals can be difficult to discern. Vaccination reduces the incidence and severity of clinical signs (Powell et al. 1995) and the duration of clinical disease (Morley et al. 1999).

Complications, usually associated with secondary bacterial infections, and more severe disease can occur in some horses (Radostits et al. 2007).

Diagnosis

Serology

Serological tests can be performed on paired sera to demonstrate a rise in antibody concentration, with the first sample being taken as early in the course of infection as possible and the second approximately two weeks later (OIE 2008a).

Haemagglutination inhibition (HI) and single radial haemolysis (SRH) testing can be used to detect neutralising antibody (Morley et al. 1995b). An increase in titre of fourfold or more between paired sera for HI and an increase in area equivalent to a twofold or more increase in antibody concentration for SRH indicate recent infection (OIE 2008a) or recent vaccination. To minimise variability, paired serum samples should be tested together at the same time (OIE 2008a).

A blocking ELISA, that uses recombinant influenza A nucleoprotein rather than whole virus as the antigen (Heine et al. 2007), was used to detect EI antibodies during the 2007 outbreak in Australia. In horses vaccinated with only the canarypox recombinant vaccine, the blocking ELISA was used alongside HI testing to differentiate infected from vaccinated animals (EI Epidemiology Support Group 2009).

Viral detection

Definitive diagnosis of EI is achieved by detecting virus or viral product from nasopharyngeal swabs. Swabs should be sufficiently long to pass through the ventral meatus into the nasopharynx and should be transferred to transport media and transported on ice (OIE 2008a). Swabs should be collected within 24 hours of the onset of pyrexia (Hannant and Mumford 1996).

EIV can be isolated in embryonated hens' eggs or cell culture (OIE 2008a). However, up to five passages may be required to isolate virus if it is present only at low levels (e.g. in vaccinated animals) (Hannant and Mumford 1996; OIE 2008a).

Quantitative polymerase chain reaction (PCR) tests have been developed that allow rapid detection and quantification of viral RNA in swab material. Viral detection by real-time reverse transcriptase (RT) PCR has been shown to be faster, less labour-intensive and 17% and 29% more sensitive than isolation of virus in eggs or cell culture, respectively (Quinlivan et al. 2005). However, isolation is necessary for more detailed phylogenetic characterisation and examination of antigenic properties of the virus (OIE 2008a).

Antigen may be detected using an antigen-capture ELISA based on a monoclonal antibody to the nucleoprotein (Cook et al. 1988; Livesay et al. 1993). This assay may be particularly useful if transport of samples to the laboratory is delayed or storage and transport occur in conditions that are less than ideal (Cook et al. 1988). Identification of virus-infected epithelial cells by immunofluorescence has also been described (Anestad and Maagaard 1990).

Quantitative RT-PCR has been shown to have a greater analytical sensitivity than the ELISA. It is able to detect EIV earlier and for longer than the ELISA or virus isolation in eggs, even if no clinical signs of EI are present (Bryant et al. 2010).

Influenza detection kits, such as Directigen™[15] and Espline®, are commercially available. Directigen™ is an enzyme immunoassay designed to detect influenza A nucleoprotein in suitable specimens from symptomatic human patients and gives a result in 15 minutes. Compared with virus culture, the Directigen™ assay exhibited a sensitivity of 83% and specificity of 78% on nasopharyngeal swabs from horses and ponies (Chambers et al. 1994). In a study of horses naturally infected with EI, 33–45% of nasal secretion samples from clinically infected horses tested positive on the Directigen™ assay (Morley et al. 1995a). Rapid antigen detection kits are not as sensitive as viral isolation or RT-PCR, particularly when only a small amount of virus is shed, and a negative result does not preclude the possibility of infection with EIV (Chambers et al. 1994; Morley et al. 1995; Quinlivan et al. 2004; Yamanaka et al. 2008). Espline® may have a higher specificity than Directigen™ (Yamanaka et al. 2008).

Vaccination

Inactivated and live whole virus EI vaccines and recombinant-vector-based EI vaccines are commercially available overseas. Immunity following natural infection with EI is longer-lasting than immunity to vaccination, as both humoral and cellular immune responses occur, and newer vaccine strategies have attempted to mimic this (Paillot et al. 2006).

Vaccine efficacy can be influenced by strain composition, antigenic content, adjuvant, timing of administration and individual response (Minke et al. 2004). Vaccine heterogenicity to the challenge strain may contribute to vaccine breakdown (Daly et al. 2003; Park et al. 2004). EIV is susceptible to antigenic drift, which occurs when mutations affect the antigenic sites of the H (and N) proteins, and the new virus may be less recognisable by pre-existing antibody (Daly et al. 2004a), resulting in reduced protection by vaccines (Daly and Mumford 2001). Antigenic drift was suggested as a

[15] Becton Dickinson DirectigenTM Flu A package insert, dated 10 October 2006. Available at http://www.bd.com/ds/productCenter/256020.asp. Accessed 13 March 2009.

major contributing factor in an EI outbreak in vaccinated horses in the United Kingdom in 1989 (Binns et al. 1993) and in Croatia in 2004 (Barbic et al. 2009).

Antigenically and genetically distinct American and European variants of H3N8 EIV are recognised and the co-circulation of the two lineages has implications for the selection of strain for vaccines (Daly et al. 1996; Yates and Mumford 2000; Daly et al. 2004b). European strains of virus have been isolated in North America and *vice versa* (Mumford 1999), demonstrating the ability of EIV to spread with horses despite vaccination requirements of importing countries. Virus strains isolated in North America and Europe during 2006 and 2007 demonstrated that Florida sublineage clade 1 viruses appear to predominate in North America and clade 2 viruses predominate in Europe (Bryant et al. 2009).

Since 2004, the OIE Expert Surveillance Panel for Equine Influenza Vaccines has recommended that vaccine manufacturers update the American lineage H3N8 component of vaccines to an A/eq/South Africa 03-like virus (other viruses such as A/eq/Wisconsin/03, A/eq/Ohio/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are also suitable) (OIE 2008b). There is no requirement for H7N7 virus in EI vaccines, and the Expert Surveillance Panel no longer recommends inclusion of a Eurasian lineage H3N8 component (OIE 2009b). Vaccines that meet the OIE recommendations are currently commercially available in some approved countries, although most vaccines also include a Eurasian lineage H3N8 virus.

Conclusion

EI is present in approved countries and there are recommendations in the Code (OIE 2009a). Australia's current quarantine measures for EI differ to those in the Code. The Expert Panel thus considered that a risk assessment was appropriate.

5.14.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

This risk assessment is based on the assumption that a response consistent with AUSVETPLAN Disease Strategy Manual for EI (Animal Health Australia 2007) would be implemented.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of EIV being present in an imported horse.

- EIV has a worldwide distribution (OIE 2008a), and all ages and breeds of equids can be infected (Nyaga et al. 1980).
- Subclinical infection with shedding can occur in vaccinated horses (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007). Vaccination is practised in many EI-endemic countries.
- In susceptible populations, explosive outbreaks of EI can occur with clinical disease in nearly all exposed horses (Radostits et al. 2007). In populations of horses of mixed ages and various serum titres to EIV, the rate of clinical disease during an outbreak is lower (Morley et al. 2000a).

Based on these considerations, the likelihood of release of EIV associated with horses from a county where the disease is present was estimated to be '*moderate*'.

Exposure assessment

Direct contact and fomite spread are the most likely exposure pathways for EIV.

The exposure group considered is equids (including feral equids).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to EIV via an imported horse.

EIV is highly contagious (Radostits et al. 2007).

Naïve, experimentally infected equids shed virus from within 48 hours of infection for a duration of up to seven days (Soboll et al. 2003; Crouch et al. 2004; Heldens et al. 2004; Edlund Toulemonde et al. 2005).

Vaccination can significantly reduce both the clinical signs of EI and the extent of viral shedding in both horses and ponies. However, viral shedding can occur in vaccinated horses in the absence of clinical signs of disease (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).

Importation of EI-infected horses has been associated with EI outbreaks in a number of previously free countries (Daly et al. 2004a).

Based on these considerations, the likelihood of susceptible animals being exposed to EIV via an infected imported horse was estimated to be '*high*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*moderate*' combined with the likelihood of exposure estimated to be '*high*', the likelihood of release and exposure for EIV was estimated to be ***'moderate'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to EIV is considered to be establishment and/or spread to populations of susceptible animals (equids) within multiple states/territories through direct contact and fomite transmission.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to EIV.

- EIV is highly contagious (Radostits et al. 2007).
- The largely immunologically naïve Australian horse population is highly susceptible to EIV.
- EIV can persist, remain infective (Bean et al. 1982) and be spread on fomites, including humans and transport vehicles (Guthrie et al. 1999).
- Spread can occur through movement of incubating or subclinically infected horses (Radostits et al. 2007). Commingling of horses is common at events and horses travel long distances, including interstate, to participate.
- In the 2007 outbreak in Australia, EI spread within two states before confirmation of diagnosis.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of EI was estimated to be '*high*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of EI for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Although EI has a high morbidity, the case fatality rate is generally less than 1%, with most deaths due to secondary bacterial infection (Radostits et al. 2007).
- Infected horses can experience a temporary loss of performance of variable duration. Complications, usually associated with secondary bacterial infections, and more severe disease can occur in some horses (Radostits et al. 2007).

Based on these considerations, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- EI is not considered to have any direct effects on the environment.

Based on this consideration, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- If EI was identified in Australia, the policy as outlined in AUSVETPLAN Disease Strategy Manual for EI (Animal Health Australia 2007) is eradication by quarantine and movement controls, decontamination, tracing and surveillance (including feral equids) and an awareness campaign. Vaccination may be used during the eradication program. Eradication was achieved in approximately four months in the 2007 EI outbreak in Australia (DAFF 2008).
- EI is scheduled as Category 4 under the Australian Emergency Animal Disease Response Agreement (EADRA) for cost-sharing arrangements (Animal Health Australia 2001). Should it be activated, EADRA states that costs of the response would be covered by government and relevant industries by contributions of 20% and 80%, respectively (Animal Health Australia 2001). However, currently the horse industry is not a signatory to this agreement.

Based on these considerations, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- As a result of the detection of EI, movement restrictions would be imposed on all equids and potentially infected fomites.
- Horse racing and other equestrian events would be prohibited for varying periods.
- Following detection of EI in one state/territory of Australia, other states/territories would close their borders to all equids until the extent of the outbreak was ascertained.
- Industries supporting equine activities such as stockfeed manufacturers, veterinarians, farriers and saddlers would also be affected.

Based on these considerations, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- The effects on international trade of a confirmed outbreak of EI in Australia would result in national disruption to exports of horses, particularly to Asia and New Zealand. Resumption of trade and associated conditions would depend on negotiations with trading partners and additional measures, such as testing for EI, may be required.
- If EI were to become established, renegotiations of trade conditions would be necessary.

Based on these considerations, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- EI is not considered to lead to any indirect effects on the environment.

Based on this consideration, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Disruption of horse events would have significant social consequences for people involved.
- Where equids were important to the local economy, the economic viability of communities within affected regions may be compromised due to the effect on associated industries.
- During the 2007 Australian EI outbreak, major disruption to the social functioning for horse owners and those employed in the horse industry was reported (Taylor et al. 2008a). Elevated levels of physiological distress, compared to general population levels, were experienced by horse owners nationally, and particularly so in the infected and buffer zones (Taylor et al. 2008b).
- Socioeconomic effects during the 2007 Australian EI outbreak were essentially due to disease control measures put in place. If a response consistent with the AUSVETPLAN Disease Strategy Manual for EI were not implemented, the socioeconomic effects would be expected to be much lower.

Based on these considerations, the effect of the establishment and/or spread of EI in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, where the effect of a disease with respect to one or more criteria is 'E', the overall effect associated with the outbreak scenario is considered to be '*moderate*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*high*') is combined with the estimate of the overall effect of establishment and/or spread ('*moderate*'), resulting in '***moderate***' likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of EI introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure ('***moderate***') is combined with the likely consequences of establishment and/or spread ('***moderate***'), resulting in a risk estimation of **MODERATE**.

Conclusion

The unrestricted risk associated with EI is determined to be **MODERATE**. The unrestricted risk estimate exceeds Australia's ALOP and, therefore, risk management is deemed necessary.

A summary of the risk assessment for EI is shown in Figure 5.3 and Table 5.3.

Figure 5.3 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for EI.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Moderate*
Likelihood of *exposure*	Likelihood of *exposure*	*High*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Moderate***
Consequences assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*High*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**MODERATE**

Table 5.3 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for EI.

References

Anestad G, Maagaard O (1990) Rapid diagnosis of equine influenza. *The Veterinary Record* 126: 550-551.

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

Animal Health Australia (2007) *Disease strategy: equine influenza (version 3.0)*. Primary Industries Ministerial Council, Canberra.

Barbic L, Madic J, Turk N, Daly J (2009) Vaccine failure caused an outbreak of equine influenza in Croatia. *Veterinary Microbiology* 133: 164-171.

Bean B, Moore BM, Sterner B, Peterson LR, Gerding DN, Balfour HH Jr (1982) Survival of influenza viruses on environmental surfaces. *The Journal of Infectious Diseases* 146: 47-51.

Beeler E (2009) Influenza in dogs and cats. Veterinary Clinics of North America: Small Animal Practice 39: 251-264.

Binns MM, Daly JM, Chirnside ED, Mumford JA, Wood JM, Richards CM, Daniels RS (1993) Genetic and antigenic analysis of an equine influenza H3 isolate from the 1989 epidemic. *Archives of Virology* 130: 33-43.

Bryant NA, Paillot R, Rash AS, Medcalf E, Montesso F, Ross J, Watson J, Jeggo M, Lewis NS, Newton JR, Elton DM (2010) Comparison of two modern vaccines and previous influenza infection against challenge with an equine influenza virus from the Australian 2007 outbreak. *Veterinary Research* 41: 1-23.

Bryant NA, Rash AS, Russell CA, Ross J, Cooke A, Bowman S, MacRae S, Lewis NS, Paillot R, Zanoni R, Meier H, Griffiths LA, Daly JM, Tiwari A, Chambers TM, Newton JR, Elton DM (2009) Antigenic and genetic variations in European and North American equine influenza virus strains (H3N8) isolated from 2006 to 2007. *Veterinary Microbiology* 138: 41-52.

Chambers TM, Holland RE, Tudor LR, Townsend HGG, Cook A, Bogdan J, Lunn DP, Hussey S, Whitaker-Dowling P, Youngner JS, Sebring RW, Penner SJ, Stiegler GL (2001) A new modified live equine influenza virus vaccine: phenotypic stability, restricted spread and efficacy against heterologous virus challenge. *Equine Veterinary Journal* 33: 630-636.

Chambers TM, Shortridge KF, Li PH, Powell DG, Watkins KL (1994) Rapid diagnosis of equine influenza by the Directigen FLU-A enzyme immunoassay. *The Veterinary Record* 135: 275-279.

Cook RF, Sinclair R, Mumford JA (1988) Detection of influenza nucleoprotein antigen in nasal secretions from horses infected with A/equine influenza (H3N8) viruses. *Journal of Virological Methods* 20: 1-12.

Crawford PC, Dubovi EJ, Castleman WL, Stephenson I, Gibbs EPJ, Chen L, Smith C, Hill RC, Ferro P, Pompey J, Bright RA, Medina M-J, Johnson CM, Olsen CW, Cox NJ, Klimov AI, Katz JM, Donis RO (2005) Transmission of equine influenza virus to dogs. *Science* 310: 482-485.

Crouch CF, Daly J, Hannant D, Wilkins J, Francis MJ (2004) Immune responses and protective efficacy in ponies immunised with an equine influenza ISCOM vaccine containing an 'American lineage' H3N8 virus. *Vaccine* 23: 418-425.

Crouch CF, Daly J, Henley W, Hannant D, Wilkins J, Francis MJ (2005) The use of a systemic prime/mucosal boost strategy with an equine influenza ISCOM vaccine to induce protective immunity in horses. *Veterinary Immunology and Immunopathology* 108: 345-355.

DAFF (2008) Recovery of EI country free status: Australian report to the World Organisation for Animal Health (OIE). Department of Agriculture, Fisheries and Forestry, Canberra.

Daly JM, Blunden AS, MacRae S, Miller J, Bowman SJ, Kolodziejek J, Nowotny N, Smith KC (2008) Transmission of equine influenza virus to English foxhounds. *Emerging Infectious Diseases* 14: 461-464.

Daly JM, Lai ACK, Binns MM, Chambers TM, Barrandeguy M, Mumford JA (1996) Antigenic and genetic evolution of equine H3N8 influenza A viruses. *Journal of General Virology* 77: 661-671.

Daly JM, Mumford JA (2001) Influenza infections. Equine Respiratory Diseases. http://www.ivis.org/special%5Fbooks/Lekeux/toc.asp (Accessed 26 February 2009).

Daly JM, Newton JR, Mumford JA (2004a) Current perspectives on control of equine influenza. *Veterinary Research* 35: 411-423.

Daly JM, Sindle T, Tearle J, Barquero N, Newton JR, Corning S (2007) Equine influenza vaccine containing older H3N8 strains offers protection against A/eq/South Africa/4/03 (H3N8) strain in a short-term vaccine efficacy study. *Equine Veterinary Journal* 39: 446-450.

Daly JM, Yates PJ, Browse G, Swann Z, Newton JR, Jessett D, Davis-Poynter N, Mumford JA (2003) Comparison of hamster and pony challenge models for evaluation of effect of antigenic drift on cross protection afforded by equine influenza vaccines. *Equine Veterinary Journal* 35: 458-462.

Daly JM, Yates PJ, Newton JR, Park A, Henley W, Wood JLN, Davis-Poynter N, Mumford JA (2004b) Evidence supporting the inclusion of strains from each of the two co-circulating lineages of H3N8 equine influenza virus in vaccines. *Vaccine* 22: 4101-4109.

Davenport FM, Hennessy AV, Minuse E (1967) Further observations on the significance of A/equine-2/63 antibodies in man. *Journal of Experimental Medicine* 126: 1049-1061.

Davis J, Garner MG, East IJ (2009) Analysis of local spread of equine influenza in the Park Ridge region of Queensland. *Transboundary and Emerging Diseases* 56: 31-38.

Edlund Toulemonde C, Daly J, Sindle T, Guigal PM, Audonnet JC, Minke JM (2005) Efficacy of a recombinant equine influenza vaccine against challenge with an American lineage H3N8 influenza virus responsible for the 2003 outbreak in the United Kingdom. *The Veterinary Record* 156: 367-371.

EI Epidemiology Support Group (2009) *Equine influenza 2007: the Australian experience*. Animal Health Australia, Canberra.

Grayson ML, Melvani S, Druve J, Barr IG, Ballard SA, Johnson PDR, Mastorakos T, Birch C (2009) Efficacy of soap and water and alcohol-based hand-rub preparations against live H1N1 influenza virus on the hands of human volunteers. *Clinical Infectious Diseases* 48: 285-291.

Guo Y, Wang M, Kawaoka Y, Gorman O, Ito T, Saito T, Webster RG (1992) Characterization of a new avian-like influenza A virus from horses in China. *Virology* 188: 245-255.

Guo Y, Wang M, Zheng GS, Li WK, Kawaoka Y, Webster RG (1995) Seroepidemiological and molecular evidence for the presence of two H3N8 equine influenza viruses in China in 1993-94. *Journal of General Virology* 76: 2009-2014.

Guthrie AJ (2006) Equine influenza in South Africa, 2003 outbreak. In *Proceedings of the 9th international congress of World Equine Veterinary Association, Marrakech, Morocco 22-26 January 2006,* (eds. Bakkoury M, Dakkak A) pp. 1-4, World Equine Veterinary Association, [France].

Guthrie AJ, Stevens KB, Bosman PP (1999) The circumstances surrounding the outbreak and spread of equine influenza in South Africa. *Revue Scientifique et Technique de l'Office International des Epizooties* 18: 179-185.

Hannant D, Mumford JA (1996) Equine influenza. In *Virus infections of equines* (ed. Studdert MJ) pp. 285-293. Elsevier Science Publishers, Amsterdam.

Heine HG, Trinidad L, Selleck P, Lowther S (2007) Rapid detection of highly pathogenic avian influenza H5N1 virus by TaqMan reverse transcriptase-polymerase chain reaction. *Avian Diseases* 51: 370-372.

Heldens JGM, Pouwels HGW, van Loon AAWM (2004) Efficacy and duration of immunity of a combined equine influenza and equine herpesvirus vaccine against challenge with an American-like equine influenza virus (A/equi-2/Kentucky/95). *The Veterinary Journal* 167: 150-157.

Hemmes JH, Winkler KC, Kool SM (1960) Virus survival as a seasonal factor in influenza and poliomyelitis. *Nature* 188: 430-431.

Huntington P (1990) *Equine influenza: the disease and its control*. No. Technical report series 184, Department of Agriculture and Rural Affairs, Victoria.

Kasel JA, Alford RH, Knight V, Waddell GH, Sigel MM (1965) Experimental infection of human volunteers with equine influenza virus. *Nature* 206: 41-43.

Kasel JA, Couch RB (1969) Experimental infection in man and horses with influenza A viruses. *Bulletin of the World Health Organization* 41: 447-452.

Kawaoka Y, Cox NJ, Haller O, Hongo S, Kaverin N, Klenk H-D, Lamb RA, McCauley J, Palese O, Rimstad E, Webster RG (2005) Orthomyxoviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 681-693. Elsevier, San Diego.

King EL, Macdonald D (2004) Report of the Board of Inquiry appointed by the Board of the National Horseracing Authority to conduct enquiry into the causes of the equine influenza which started in the western Cape in early December 2003 and spread to the eastern Cape and Gauteng. *Australian Equine Veterinarian* 23: 139-142.

Landolt GA, Townsend HGG, Lunn DP (2007) Equine influenza infection. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 124-134. Saunders Elsevier, St. Louis.

Livesay GJ, O'Neill T, Hannant D, Yadav MP, Mumford JA (1993) The outbreak of equine influenza (H3N8) in the United Kingdom in 1989: diagnostic use of an antigen capture ELISA. *The Veterinary Record* 133: 515-519.

Loosli CG, Lemon HM, Robertson OH, Appel E (1943) Experimental air-borne influenza infection: I. influence of humidity on survival of virus in air. *Proceedings of the Society for Experimental Biology and Medicine* 53: 205-206.

Lunn DP, Hussey S, Sebring R, Rushlow KE, Radecki SV, Whitaker-Dowling P, Youngner JS, Chambers TM, Holland RE, Horohov DW (2001) Safety, efficacy, and immunogenicity of a modified-live equine influenza virus vaccine in ponies after induction of exercise-induced immunosuppression. *Journal of the American Veterinary Medical Association* 218: 900-906.

Madic J, Martinovic S, Naglic T, Hajsig D, Cvetnic S (1996) Serological evidence for the presence of A/equine-1 influenza virus in unvaccinated horses in Croatia. *The Veterinary Record* 138: 68.

McQueen JL, Davenport FM, Minuse E (1966) Studies of equine influenza in Michigan, 1963. I. etiology. *American Journal of Epidemiology* 83: 271-279.

Miller WMC (1965) Equine influenza: further observations on the 'coughing' outbreak, 1965. *The Veterinary Record* 77: 455-456.

Minke JM, Audonnet J-C, Fischer L (2004) Equine viral vaccines: the past, present and future. *Veterinary Research* 35: 425-443.

Minke JM, Edlund Toulemonde C, Coupier H, Guigal P-M, Dinic S, Sindle T, Jessett D, Black L, Bublot M, Pardo MC, Audonnet J-C (2007) Efficacy of a canarypox-vectored recombinant vaccine expressing the hemagglutinin gene of equine influenza H3N8 virus in the protection of ponies from viral challenge. *American Journal of Veterinary Research* 68: 213-219.

Morley PS, Bogdan JR, Townsend HGG, Haines DM (1995a) Evaluation of Directigen Flu A assay for detection of influenza antigen in nasal secretions of horses. *Equine Veterinary Journal* 27: 131-134.

Morley PS, Hanson LK, Bogdan JR, Townsend HGG, Appleton JA, Haines DM (1995b) The relationship between single radial hemolysis, hemagglutination inhibition, and virus neutralization assays used to detect antibodies specific for equine influenza viruses. *Veterinary Microbiology* 45: 81-92.

Morley PS, Townsend HGG, Bogdan JR, Haines DM (1999) Efficacy of a commercial vaccine for preventing disease caused by influenza virus infection in horses. *Journal of the American Veterinary Medical Association* 215: 61-66.

Morley PS, Townsend HGG, Bogdan JR, Haines DM (2000a) Descriptive epidemiologic study of disease associated with influenza virus infections during three epidemics in horses. *Journal of the American Veterinary Medical Association* 216: 535-544.

Morley PS, Townsend HGG, Bogdan JR, Haines DM (2000b) Risk factors for disease associated with influenza virus infections during three epidemics in horses. *Journal of the American Veterinary Medical Association* 216: 545-550.

Mumford JA (1999) Control of influenza from an international perspective. In Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates, (eds. Wernery U, Wade JF, Mumford JA, Kaaden O.R.) pp. 11-24, R & W Publications (Newmarket) Limited, Newmarket.

Newton JR (2008) *Report to the equine influenza inquiry, Sydney, Australia*. No. AHT.0001.001, Animal Health Trust, Newmarket.

Newton JR, Blunden A, Bryant N, Bowman S, Cooke AF, Daly J, Hammond TA, Miller J, Rash A, Elton DE (2009) Cross-species transmission of H3N8 equine influenza virus into foxhounds in the United Kingdom. In *Proceedings of the 12th Symposium of the International Society for Veterinary Epidemiology and Economics, Durban, South Africa: ISVEE, 10-14 August, 2009,* p. 189, [International Society for Veterinary Epidemiology and Economics], [South Africa].

Nyaga PN, Wiggins AD, Priester WA (1980) Epidemiology of equine influenza, risk by age, breed and sex. *Comparative Immunology, Microbiology and Infectious Diseases* 3: 67-73.

OIE (2008a) Equine influenza. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.07_EQ_INF.pdf (Accessed 1 September 2009).

OIE (2008b) Expert Surveillance Panel on equine influenza vaccines: conclusions and recommendations. *OIE Bulletin* 2: 42-45.

OIE (2009a) Equine influenza. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.7.htm (Accessed 19 August 2009a).

OIE (2009b) Expert Surveillance Panel on equine influenza vaccine composition: conclusions and recommendations. *OIE Bulletin* 2: 42-43.

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009c).

Paillot R, Hannant D, Kydd JH, Daly JM (2006) Vaccination against equine influenza: quid novi? *Vaccine* 24: 4047-4061.

Park AW, Wood JLN, Daly JM, Newton JR, Glass K, Henley W, Mumford JA, Grenfell BT (2004) The effects of strain heterology on the epidemiology of equine influenza in a vaccinated population. *Proceedings of the Royal Society of London B* 271: 1547-1555.

Powell DG, Watkins KL, Li PH, Shortridge KF (1995) Outbreak of equine influenza among horses in Hong Kong during 1992. *The Veterinary Record* 136: 531-536.

Prince HN, Prince DL (2001) Principles of viral control and transmission. In *Disinfection, sterilization, and preservation* (ed. Block SS) pp. 543-571. Lippincott Williams & Wilkins, Philadelphia.

Quinlivan M, Cullinane A, Nelly M, van Maanen K, Heldens J, Arkins S (2004) Comparison of sensitivities of virus isolation, antigen detection, and nucleic acid amplification for detection of equine influenza virus. *Journal of Clinical Microbiology* 42: 759-763.

Quinlivan M, Dempsey E, Ryan F, Arkins S, Cullinane A (2005) Real-time reverse transcription PCR for detection and quantitative analysis of equine influenza virus. *Journal of Clinical Microbiology* 43: 5055-5057.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

Rice EW, Adcock NJ, Sivaganesan M, Brown JD, Stallknecht DE, Swayne DE (2007) Chlorine inactivation of highly pathogenic avian influenza virus (H5N1). *Emerging Infectious Diseases* 13: 1568-1570.

Soboll G, Horohov DW, Aldridge BM, Olsen CW, McGregor MW, Drape RJ, Macklin MD, Swain WF, Lunn DP (2003) Regional antibody and cellular immune responses to equine influenza virus infection, and particle mediated DNA vaccination. *Veterinary Immunology and Immunopathology* 94: 47-62.

Swayne DE, Halvorson DA (2003) Influenza. In *Diseases of poultry* (eds. Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE) pp. 135-160. Iowa State University Press, Ames.

Taylor M, Agho K, Griffin E (2008a) *Human impacts of equine influenza*. University of Western Sydney, Sydney.

Taylor M, Agho K, Stevens G, Raphael B (2008b) Factors influencing psychological distress during a disease epidemic: data from Australia's first outbreak of equine influenza. *BMC Public Health* 8: 1-13.

Townsend HGG, Penner SJ, Watts TC, Cook A, Bogdan J, Haines DM, Griffin S, Chambers T, Holland RE, Whitaker-Dowling P, Youngner J, Sebring R (2001) Efficacy of a cold-adapted, intranasal, equine influenza vaccine: challenge trials. *Equine Veterinary Journal* 33: 637-643.

Webster RG (1993) Are equine 1 influenza viruses still present in horses? *Equine Veterinary Journal* 25: 537-538.

Yadav MP, Uppal PK, Mumford JA (1993) Physico-chemical and biological characterization of A/Equi-2 virus isolated from 1987 equine influenza epidemic in India. *International Journal of Animal Sciences* 8: 93-98.

Yamanaka T, Nemoto M, Tsujimura K, Kondo T, Matsumura T (2009) Interspecies transmission of equine influenza virus (H3N8) to dogs by close contact with experimentally infected horses. *Veterinary Microbiology* In press: In press.

Yamanaka T, Tsujimura K, Kondo T, Matsumura T (2008) Evaluation of antigen detection kits for diagnosis of equine influenza. *The Journal of Veterinary Medical Science* 70: 189-192.

Yates P, Mumford JA (2000) Equine influenza vaccine efficacy: the significance of antigenic variation. *Veterinary Microbiology* 74: 173-177.

5.15 Equine paratyphoid

5.15.1 Technical information

Background

Salmonella enterica subspecies *enterica* serovar Abortusequi (*Salmonella* Abortusequi) is a host-adapted salmonella of horses and donkeys. It causes equine paratyphoid, which manifests mainly as abortion but also as arthritis, fistulous withers, orchitis and septicaemia (Akiba et al. 2003; Radostits et al. 2007). Although *S. typhimurium* infection has also been referred to as equine paratyphoid, for the purposes of this risk assessment, *S.* Abortusequi is considered the causative agent of equine paratyphoid.

S. Abortusequi was widely reported in the early 1900s but is now rare in developed countries (Radostits et al. 2007). Since the 1970s, outbreaks have been recorded in Albania, Austria, Croatia, India, Italy, Japan, South Africa and South America (Madic et al. 1997; Anzai 2003; Collett and Mogg 2004; Hofer et al. 2004). The disease has not been reported in Australia and is nationally notifiable (DAFF 2008).

In Japan, *S.* Abortusequi is considered endemic only in Kushiro, an area of Hokkaido historically used for breeding horses. Outbreaks elsewhere in Japan have been traced to the movement of horses from Kushiro. The disease is notifiable in Japan and a control program is in place to manage it (Akiba et al. 2003; Anzai 2003).

Equine paratyphoid is not an OIE-listed disease (OIE 2009) although it was previously listed until 1993.

Epidemiology

S. Abortusequi is spread mainly via ingestion of pastures contaminated with infective aborted materials, including amniotic fluid, foetal and placental tissue, and uterine and vaginal discharges. Fomite transmission is possible (Radostits et al. 2007). Continuous discharge in urine is reported by Fritzschke and Söntgen (cited in Hofer et al. 2004). The organism has been isolated from testicular tissue and urethral fluid in equids with orchitis (Singh et al. 1971) and it is possible that *S.* Abortusequi can be transmitted via semen (Singh et al. 1971; Anzai 2003; Hofer et al. 2004; Radostits et al. 2007). It has not been isolated from faeces (Singh et al. 1971; Collett and Mogg 2004).

S. Abortusequi is highly infectious. Abortion storms occur in naïve populations and in naïve animals introduced to endemic populations (Collett and Mogg 2004). A carrier state has been described (Sharma 1998; Iribarren and Pidre 2002; Anzai 2003) and carrier animals have been implicated as the source of infection in outbreaks in non-endemic areas (Madic et al. 1997; Akiba et al. 2003; Hofer et al. 2004). Stress, such as transport, can precipitate shedding of *S.* Abortusequi and lead to outbreaks of abortion (Tewari et al. 1989; Swerczek 1991).

Clinical signs

The incubation period for most Salmonella infections is 6–72 hours. Some mares show no clinical signs other than abortion. Infection in pregnant mares can cause

transient pyrexia followed by either abortion or neonatal septicaemia and foetal death. Foals that survive can have localised infections such as polyarthritis and omphalitis (Collett and Mogg 2004; Radostits et al. 2007). Retained placenta, metritis and copious vaginal discharge are common sequelae in mares (Radostits et al. 2007).

S. Abortusequi has been isolated from stallions with inflammatory testicular lesions and oedema of the prepuce and scrotum (Singh et al. 1971; Radostits et al. 2007), and from cases of fistulous withers, pneumonia, purulent tendovaginitis and bursitis (Collett and Mogg 2004; Radostits et al. 2007).

S. Abortusequi has also been reported to cause abortion (Hofer et al. 2004) and chronic orchitis (Singh et al. 1971) in donkeys.

Diagnosis

S. Abortusequi can be cultured from tissues using standard culture techniques and is readily isolated from aborted materials and vaginal discharges (Anzai 2003; CFSPH 2005).

Serological tests are available but antibodies cross-react with those of other enterobacteria, and serology is an unreliable indicator of infection or immune status (Collett and Mogg 2004). Interpretation of serological findings should take into account clinical history (Anzai 2003).

Pulsed-field gel electrophoresis has been used to characterise strains within the serovar to assist outbreak investigation (Akiba et al. 2003; Hofer et al. 2004).

Vaccination provides good immunity in naturally infected horses and has contributed to the virtual eradication of the disease in some countries (Radostits et al. 2007).

Conclusion

Equine paratyphoid is present in some approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.15.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of *S.* Abortusequi being present in an imported horse.

- The disease is now considered rare, but there are recent reports of cases from Austria, Brazil, Croatia, India and in limited areas of Japan.
- Outbreaks in other countries have been attributed to the introduction of carrier animals.
- The prevalence of infection in approved countries is unknown, and serological data are unreliable due to antibodies cross-reacting with other enterobacteria (Collett and Mogg 2004).

Based on these considerations, the likelihood of release of equine paratyphoid associated with horses from a country where the disease is present was estimated to be '*very low*'.

Exposure assessment

The single most likely pathway is via ingestion of pastures contaminated with infective aborted materials and uterine and vaginal discharges.

The only exposure group considered was equids (including feral equids). *S.* Abortusequi is a host-specific organism and is only known to cause disease in horses and donkeys.

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to equine paratyphoid via an imported horse.

- Shedding of *S.* Abortusequi can occur during periods of stress, such as after transport.
- There may be continuous shedding of the organism in urine and through semen of infected stallions.
- *S.* Abortusequi is highly infectious to naïve horses.
- Pasture contamination with infective aborted materials can be high.
- The organism can survive in the environment for several weeks.
- Transmission via fomites can occur.

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with *S.* Abortusequi was estimated to be '*high***'**.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*very low*' combined with the likelihood of exposure estimated to be '*high***'**, the likelihood of release and exposure for was estimated to be ***'very low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to *S.* Abortusequi is considered to be establishment and/or spread to populations of susceptible animals within a local area through direct transmission of infected vaginal or uterine discharges or aborted material from a carrier mare.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to *S.* Abortusequi.

- Carrier animals have been implicated as the source of infection in outbreaks in non-endemic areas and are likely to spread the disease prior to detection.
- *S.* Abortusequi is highly infectious and likely to cause abortion storms in naïve populations.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of equine paratyphoid was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of equine paratyphoid for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Infected mares and stallions exhibit transitory clinical signs and loss of productivity through abortions and orchitis.
- Infected foals can die or develop polyarthritis and omphalitis.
- Naïve animals are more likely to develop clinical signs of disease.

Based on these considerations, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- *S.* Abortusequi is an equid-specific pathogen and is not considered to have any direct effects on the environment.

Based on this consideration, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Salmonellosis due to *S.* Abortusequi is nationally notifiable in Australia (DAFF 2008).
- There is no AUSVETPLAN Disease Strategy Manual for equine paratyphoid and the disease is not scheduled in Australia's Emergency Animal Disease Response Agreement.
- It is likely that an outbreak of equine paratyphoid would be rapidly detected and controlled before it had spread out of the local area using biosecurity measures including movement controls, decontamination and tracing.
- Management practices, such as segregation for mares in breeding establishments, might need to be developed.

Based on these considerations, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- There may be a decrease in the value of stallions due to potential carrier status or infection in semen.
- Equine paratyphoid is a nationally notifiable disease in Australia and if it was detected in any state, movement restrictions would be imposed and other states/territories may close their borders to all susceptible animals.
- Associated businesses and industries such as stockfeed manufacturers', veterinarians and farriers could be affected.

Based on these considerations, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- If equine paratyphoid was detected in Australia, there could be some disruption to the export of horses to countries that currently require premises freedom from equine paratyphoid.

Based on this consideration, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be significant at the local

level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- *S.* Abortusequi is unlikely to have any affect on the environment.

Based on this consideration, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- An outbreak of equine paratyphoid would have a minor effect on communities relating to rural and regional economics as it is likely to be contained and managed quickly.

Based on this consideration, the effect of the establishment and/or spread of equine paratyphoid in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria 'C', the overall effect associated with the outbreak scenario is considered to be '*very low*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*very low*') which results in ***'very low***' likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of equine paratyphoid introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure ('***very low***') is combined with the likely consequences of establishment and/or spread ('***very low***') which results in a risk estimation of **NEGLIGIBLE**.

Conclusion

The overall risk associated with equine paratyphoid is determined to be **NEGLIGIBLE**. As the unrestricted risk estimate achieves Australia's ALOP, no risk management is considered necessary.

A summary of the risk assessment for equine paratyphoid is shown in Figure 5.4 and Table 5.4.

Figure 5.4 Summary of risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for equine paratyphoid.

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Very Low*
Likelihood of *exposure*	Likelihood of *exposure*	*High*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socio-economic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5.	*Very low*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Very low***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**NEGLIGIBLE**

Table 5.4 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for equine paratyphoid.

References

Akiba M, Uchida I, Nishimori K, Tanaka K, Anzai T, Kuwamoto Y, Wada R, Ohya T, Ito H (2003) Comparison of *Salmonella enterica* serovar Abortusequi isolates of equine origin by pulsed-field gel electrophoresis and fluorescent amplified-fragment length polymorphism fingerprinting. *Veterinary Microbiology* 92: 379-388.

Anzai T (2003) Equine paratyphoid and salmonellosis. *Journal of Veterinary Medicine, Japan* 56: 663-667.

CFSPH (2005) Salmonellosis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.htm (Accessed 13 January 2009).

Collett MG, Mogg TD (2004) Equine salmonellosis. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 1608-1616. Oxford University Press, Oxford.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

Hofer E, Berghold C, Sick K (2004) *Salmonella enterica* subsp. *enterica* Serovar Abortusequi: infection in horses in Austria. *Wiener Tierarztliche Monatsschrift* 91: 292-296.

Iribarren FE, Pidre GA (2002) Paratyphoid abortion in equines: bibliographic review. *Veterinaria Argentina* 19: 607-610.

Madic J, Hajsig D, Sostaric B, Curic S, Seol B, Naglic T, Cvetnic Z (1997) An outbreak of abortion in mares associated with *Salmonella abortusequi* infection. *Equine Veterinary Journal* 29: 230-233.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with bacteria III. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 847-1006. Saunders Elsevier, Edinburgh.

Sharma PC (1998) Infectious abortions in equines. *Haryana Veterinary* 117: 67-79.

Singh IP, Sharma VK, Kaura YK (1971) Some aspects of the epidemiology of *Salmonella abortus-equi* infection in equines. *British Veterinary Journal* 127: 378.

Swerczek TW (1991) Identifying the bacterial causes of abortion in mares. *Veterinary Medicine* 86: 1210-1216.

Tewari SC, Sharma PC, Prasad S, Kaura YK (1989) Equine herpesvirus 1 and neonatal foal mortality in northern India. *Revue Scientifique et Technique de l'Office International des Epizooties* 8: 103-110.

5.16 Equine piroplasmosis

5.16.1 Technical information

Background

Equine piroplasmosis — also known as equine babesiosis — is a tick-borne, protozoal infection of horses, mules, donkeys and zebras, and is characterised by acute anaemia. The causative organisms, *Babesia caballi* and *Theileria equi* (formerly *Babesia equi*) are transmitted primarily by ixodid ticks. Prevalence of equine piroplasmosis is higher in tropical and subtropical regions (Radostits et al. 2007). Zebras are an important reservoir of the disease in Africa. In Spain, dogs and foxes have tested seropositive to *T. equi* but are not thought to be significant in the epidemiology and transmission of equine piroplasmosis (Criado-Fornelio et al. 2003).

Equine piroplasmosis is widespread but Australia is free of the disease. It is endemic in Africa, Asia (except Siberia), and Central and South America (de Waal et al. 1988). Historically considered free of infection, a recent survey of 2019 horses in Japan found 5.4% of horses seropositive for *B. caballi* and 2.2% seropositive for *T. equi* (Ikadai et al. 2002). High prevalence has been reported from the Middle and Near East. In Europe, equine piroplasmosis extends from Portugal and Spain, through France and Italy to the Balkans, Hungary, Romania, and Russia. Austria, Belgium, the Czech Republic, Poland and Switzerland are marginal areas where autochthonous infections can occur (Friedhoff and Soulé 1996).

Equine piroplasmosis is not endemic in Germany, the Netherlands, Scandinavia or the United Kingdom, and although suitable tick vectors are present, autochthonous infections have not been reported (de Waal and van Heerden 2004). Most infections of horses in Germany and the United Kingdom have been traced back to imported horses from France, Italy or Spain (Friedhoff and Soulé 1996). Although equine piroplasmosis is primarily a tick-borne disease, iatrogenic transmission via contaminated hypodermic needles has resulted in the spread of *T. equi* in non-endemic areas (Callow 1984). In the United Kingdom over seven years, 92% of horses on a research farm were infected with *T. equi* before being diagnosed during routine serological testing (Callow 1984; Gerstenberg et al. 1999).

The United States was considered free from the disease since 1988 until recent outbreaks occurred in Florida (Promed Mail 2009d), Missouri (Promed Mail 2009b) and Texas (Promed Mail 2009c). Infection is thought to have been introduced by imported horses, with subsequent iatrogenic transmission (DOACS 2009). A recent outbreak has also been reported in Ireland, however there is no information available on the origin of the disease or the extent of any spread (Promed Mail 2009a). In Australia, four introductions of *T. equi* between 1950 and 1976, were diagnosed retrospectively and traced to imported horses from Texas and Spain (Callow et al. 1979; Callow 1984). The parasite failed to establish despite the presence of ticks (Callow 1984). In 2000, a retired thoroughbred from Hong Kong tested positive to *T. equi* while in Australian post-arrival quarantine and was subsequently euthanased (Promed Mail 2000).

Equine piroplasmosis is an OIE-listed disease (OIE 2009c).

Epidemiology

Prevalence of *B. caballi* and *T. equi* in tropical and subtropical regions is affected by host and vector densities, and management procedures. Serological studies in endemic areas have shown prevalences of 30–98% for *B. caballi* and more than 90% for *T. equi* (Donnelly et al. 1980; Tenter and Friedhoff 1986; Tenter et al. 1988; Avarzed et al. 1997). Both parasites are transmitted to vertebrate hosts by tick bites, and to ticks by ingestion of infected equine erythrocytes.

Differences in the replication cycles of *B. caballi* and *T. equi* affect their modes of transmission. Both ticks and horses are the reservoir of infection for *B. caballi* because in ticks, *B. caballi* is passed transovarially and trans-stadially (Mehlhorn and Schein 1998). Infected horses are the reservoir of infection for *T. equi*, not vector ticks, because in ticks there is no transovarial, only trans-stadial transmission (de Waal 1990; de Waal and Potgeiter 1987).

Both *B. caballi* and *T. equi* are stimulated to complete their maturation in tick salivary glands after the tick attaches to the vertebrate host to feed. The tick must remain attached to the host for 5–10 days before the parasite becomes infective. Over 15 species of the tick genera *Dermacentor, Hyalomma* and *Rhipicephalus* transmit equine piroplasmosis (Friedhoff et al. 1990; Battsetseg et al. 2002). Two tick species found in Australia are capable of naturally transmitting both protozoa — *Rhipicephalus* (*Boophilus) microplus*, the cattle tick and *R. sanguineus*, the brown dog tick (Battsetseg et al. 2002; Rothschild and Knowles 2007). *Haemophysalis longicornis*, the bush tick, has been used as a vector for *T. equi* in experimental situations (Ikadai et al. 2007). It is not known whether *Otobius megnini* — a tick introduced into Western Australia — is able to transmit either *B. caballi* or *T. equi.*

B. caballi and *T. equi* can be transmitted iatrogenically by needles, surgical instruments, administration of contaminated blood transfusions or failure to properly sterilise equipment that contacts equine blood, including stomach tubes and dental instruments. Following diagnosis of an infected pony in Australia at Moss Vale in New South Wales in 1976 (Churchill and Best 1976), 100 carriers in four separate locations and three states were identified. Transmission was via contaminated needles and surgical instruments (Callow 1984). The 2008 outbreak in Florida in the United States affected 20 horses in seven locations and, as in the 2009 outbreak (Promed Mail 2009e), spread was iatrogenic via shared needles (Promed Mail 2008).

Transplacental transmission of *T. equi* can occur in horses (de Waal and van Heerden 2004; Ikadai et al. 2007). Maternal antibodies persist for 1–4 months for *B. caballi* and 1–5 months for *T. equi* (Heuchert et al. 1999). *B. caballi* has been reported as a cause of clinical disease in a three month old foal (Butler et al. 2005).

Transmission of parasites in semen has not been documented; however, this may be possible if blood contamination occurs (Brüning 1996).

Clinical signs

The incubation period for *B. caballi* infections is 10–30 days and for *T. equi* infections 12–19 days. The majority of seropositive horses in endemic areas are inapparent carriers with low levels of parasitaemia and no clinical signs of infection (Rothschild and Knowles 2007). Signs of disease progress from depression, thirst, inappetence and congestion of mucous membranes, to an acute and/or chronic stage.

Acute *T. equi* infection is characterised by severe pyrexia, elevated pulse and respiratory rates, anaemia, haemolysis, icterus, haemoglobinuria and bilirubinuria. Cardiac arrhythmias have been reported in rare cases (Diana et al. 2007). Pregnant mares can abort, neonates may show peracute signs, and surviving foals can become latent carriers (Allsopp et al. 2007).

Acute disease is generally not seen in endemic areas except in horses stressed by exercise, pregnancy, environmental conditions or concurrent disease. *B. caballi* infection is rarely associated with disease. When it is, the signs are similar to, but less severe than, those seen in *T. equi* infections. Outbreaks of equine piroplasmosis can occur when large numbers of susceptible animals are exposed, but clinical signs may not be seen in affected horses (Radostits et al. 2007).

Some chronically infected horses show non-specific signs of ill thrift, poor exercise tolerance and splenomegaly (de Waal and van Heerden 2004). The disease is fatal in up to 50% of previously unexposed animals (Rothschild and Knowles 2007). Once infected with *T. equi* horses remain carriers for life (Zweygarth et al. 1997). While infection with *B. caballi* lasts at least four years, relapses can occur (Thompson 1969; de Waal and van Heerden 2004).

Diagnosis

Equine piroplasmosis may be difficult to diagnose due to variable and nonspecific clinical signs. Clinical presentation, blood smears, serology, xenodiagnosis and sub-inoculation of blood into a susceptible animal assist with diagnosis. Parasitaemia is low in infections of *B. caballi* and thick blood smears may be required to identify organisms (Rothschild and Knowles 2007).

High antibody titres correlate with infection and are first detected 7–11 days after experimental infection, peaking at 45 days (Tenter and Friedhoff 1986). Passively transferred antibodies may persist for 4–5 months. Foals born infected, but without signs of clinical disease, may remain seropositive for life (Donnelly et al. 1980).

The primary serological tests used to detect antibody are the indirect fluorescent antibody test (IFAT) and the competitive enzyme-linked immunosorbent assay (C-ELISA) (OIE 2008). These have replaced the complement fixation test as they are more sensitive and effective at detecting chronically infected animals, latent carriers and those treated with anti-parasitic drugs (Donnelly et al. 1980; Böse et al. 1995; Ogunremi et al. 2008; OIE 2008). However, the IFAT requires large amounts of antigen and is difficult to standardise due to subjectivity in interpreting fluorescence (Brüning 1996), and the C-ELISA requires further validation (OIE 2008). The C-ELISA and IFAT can give conflicting results on the same sample (OIE 2009b).

DNA probes have been used experimentally to detect low grade parasitaemia, but there is poor correlation between probe results and serological results (Potgeiter et al. 1992). A quantitative real time polymerase chain reaction assay (real time-PCR) has been developed and appears sensitive and specific for laboratory diagnosis of equine piroplasmosis (Kim et al. 2008).

Treatment

De Waal and van Heerden (2004) consider that anti-babesial agents do not sterilise *T. equi* infections. High doses of imidocarb do not reliably eliminate either *B. caballi*

(Butler et al. 2008) or *T. equi* (Kuttler et al. 1988; Friedhoff et al. 1990) infection in carriers. Treatment with imidocarb can temporarily clear *B. caballi* (Butler et al. 2008) and *T. equi* (Friedhoff et al. 1990) from the blood and result in transiently negative CFT and real time-PCR results. However, *B. caballi* DNA was again detected eight weeks after treatment started (Butler et al. 2008).

There is no cross immunity between *B. caballi* and *T. equi* infections (Ali et al., 1996). Infection with *B. caballi* elicits both cell mediated and humoral immunity. As with other protozoa, the diversity of antigens produced, along with intraerythrocytic stages, makes activity of specific acquired immune mechanisms unpredictable. For this reason vaccines are difficult to develop and there are no vaccines produced against *B. caballi* or *T. equi* (CFSPH 2008). Constant exposure along with an extended carrier state induces some immunity to reinfection in endemic areas.

Conclusion

Equine piroplasmosis is present in approved countries and there are recommendations in the Code (OIE 2009a). Australia's current quarantine measures for equine piroplasmosis differ to those in the Code. The Expert Panel thus considered that a risk assessment was appropriate.

5.16.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of equine piroplasmosis being present in an imported horse.

- Equine piroplasmosis is widespread worldwide and has a reported prevalence of 30–98% in endemic areas.
- The incubation period can be up to 30 days.
- Subclinical infections are common, especially in endemic areas.
- A latent carrier state exists.
- Diagnosis is difficult (Rothschild and Knowles 2007).

Based on these considerations, the likelihood of release of equine piroplasmosis associated with horses from a country where the disease is present was estimated to be '*moderate*'.

Exposure assessment

The most likely exposure pathway is via transmission of infected blood either by competent tick vector or iatrogenically.

The exposure group considered is equids (including feral equids).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to equine piroplasmosis via an imported horse.

- Infected blood can be transmitted iatrogenically via contaminated needles, syringes and surgical instruments, and historically this has been the most

commonly reported method of transmission in outbreaks in non-endemic countries.

- Infected blood can be transmitted by a tick vector. Tick species, shown experimentally to be competent vectors, are present in Australia.

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with equine piroplasmosis was estimated to be '*moderate*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*moderate*' combined with the likelihood of exposure estimated to be '*moderate*', the likelihood of release and exposure for equine piroplasmosis was estimated to be '***low***'.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to widespread establishment of disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to equine piroplasmosis is considered to be establishment and/or spread to limited populations of susceptible animals in several states, through movement of infected horses followed by iatrogenic spread.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to equine piroplasmosis.

- The practice of sharing needles and instruments for blood collection and other invasive procedures is common in certain segments of the industry, but less common than it was 30 years ago when equine piroplasmosis was identified in a small population of horses in Australia (Churchill and Best 1976).

- The disease would establish and spread via iatrogenic transmission to most horses (or other equids) kept on the same property as an infected imported horse, and would be unlikely to be diagnosed for some time (Gerstenberg et al. 1999).
- Movement of incubating, subclinically infected or carrier animals, or of contaminated needles or instruments would spread the disease to other properties prior to detection (Callow 1984; Promed Mail 2008).
- Once established in an area, spread may also occur via ticks as is seen in countries where equine piroplasmosis is endemic. However, spread via ticks did not occur in the equine piroplasmosis cases diagnosed in Australia 1976 (Callow 1984).

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of equine piroplasmosis was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of equine piroplasmosis for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Losses due to mortality range from 5% to 10% in endemic areas, and up to 50% when previously unexposed horses are infected (Rothschild and Knowles 2007).
- Equine piroplasmosis is responsible for 11% of reproductive failures in South Africa and surviving foals can become asymptomatic carriers (de Waal et al. 1999). Due to the persistence of *T. equi* infection, a carrier mare can produce more than one infected foetus (de Waal and van Heerden 2004).
- Horses become carriers for life after infection with *T. equi*, and for at least four years after infection with *B. caballi*. Chronic infection can cause significant disease and relapses can occur (Thompson 1969; de Waal and van Heerden 2004).
- No treatment is fully effective and there are significant side-effects associated with the treatment drugs.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- Equids are the only species in which clinical disease has been reported.
- Equine piroplasmosis is not considered to have any direct effects on the environment.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Equine piroplasmosis is nationally notifiable in Australia (DAFF 2008), however there is no AUSVETPLAN Disease Strategy Manual for the disease.
- Equine piroplasmosis is scheduled as Category 4 under Australia's Emergency Animal Disease Response Agreement (EADRA) for cost sharing arrangements. Should it be activated, EADRA states that the costs of the response would be covered by government and relevant industries by contributions of 20% and 80%, respectively (Animal Health Australia 2001). However, currently the horse industry is not a signatory to this agreement.
- To eradicate equine piroplasmosis, a combination of strategies would be employed — including quarantine and movement control of equids, tracing and surveillance, tick control, decontamination, epidemiological investigations and a public awareness campaign to prevent iatrogenic transmission.
- Eradication or control of tick vectors would require treating animals with chemical acaricides and sanitation procedures to remove ticks.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- Equine piroplasmosis is a nationally notifiable disease in Australia and if it was detected in any state, movement restrictions would be imposed and other states/territories may close their borders to all susceptible animals.
- Infected premises would face increased costs in caring for sick horses and in assisting the veterinarian taking samples, mailing test samples and laboratory testing. In addition, the premises would undergo intensive tick surveillance for domestic ticks testing positive for equine piroplasmosis.
- Associated businesses and industries such as farriers, stockfeed manufacturers' and veterinarians could also be affected.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- The effect on international trade of an outbreak of equine piroplasmosis in Australia would result in disruption to horse exports and may include additional testing of horses prior to export.
- Piroplasmosis-positive horses could be denied export opportunities.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Increased use of acaricides to control ticks could have an effect on a range of arthropod species and disrupt the food source of wildlife, lead to environmental contamination (including water sources) and resistance to acaricides.
- Increased use of anti-babesial agents such as imidocarb could lead to resistance.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Disruption of horse events would have social consequences for people directly involved. Horse racing also contributes significantly to government revenue.
- The financial cost of owning horses would increase due to additional diagnostic tests, tick surveillance and loss of income from piroplasmosis-positive horses.

Based on these considerations, the effect of the establishment and/or spread of equine piroplasmosis in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria 'E', the overall effect associated with the outbreak scenario was considered to be '*moderate*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread (‘*moderate*’) is combined with the estimate of the overall effect of establishment and/or spread (‘*moderate’*) which results in ***‘moderate***’ likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of equine piroplasmosis introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure (‘***low***’) is combined with the likely consequences of establishment and/or spread (‘***moderate***’) which results in a risk estimation of **LOW**.

Conclusion

The overall risk associated with equine piroplasmosis is determined to be **LOW**. The unrestricted risk estimate exceeds Australia’s ALOP and, therefore, risk management is deemed necessary.

A summary of the risk assessment for equine piroplasmosis is shown in Figure 5.5 and Table 5.5.

Figure 5.5 Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for equine piroplasmosis.

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Moderate*
Likelihood of *exposure*	Likelihood of *exposure*	*Moderate*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socio-economic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5.	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**LOW**

Table 5.5 Summary of the release, exposure and consequence assessments, resulting in an unrestricted risk estimate for equine piroplasmosis.

References

Allsopp MTEP, Lewis BD, Penzhorn BL (2007) Molecular evidence for transplacental transmission of *Theileria equi* from carrier mares to their apparently healthy foals. *Veterinary Parasitology* 148: 130-136.

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

Avarzed A, de Waal DT, Igarashi I, Saito A, Oyamada T, Toyoda Y, Suzuki N (1997) Prevalence of equine piroplasmosis in central Mongolia. *Onderstepoort Journal of Veterinary Research* 64: 141-145.

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

Böse R, Jorgensen WK, Dalgliesh RJ, Friedhoff KT, de Vos AJ (1995) Current state and future trends in the diagnosis of babesiosis. *Veterinary Parasitology* 57: 61-74.

Brüning A (1996) Equine piroplasmosis an update on diagnosis, treatment and prevention. *British Veterinary Journal* 152: 139-151.

Bryant NA, Paillot R, Rash AS, Medcalf E, Montesso F, Ross J, Watson J, Jeggo M, Lewis NS, Newton JR, Elton DM (2010) Comparison of two modern vaccines and previous influenza infection against challenge with an equine influenza virus from the Australian 2007 outbreak. *Veterinary Research* 41: 1-23.

Butler CM, Nijhof AM, van der Kolk JH, de Haseth OB, Taoufik A, Jongejan F, Houwers DJ (2008) Repeated high dose imidocarb dipropionate treatment did not eliminate *Babesia caballi* from naturally infected horses as determined by PCR-reverse line blot hybridization. *Veterinary Parasitology* 151: 320-322.

Butler CM, van Gils JA, van der Kolk JH (2005) Een literatuuroverzicht van equine piroplasmose naar aanleiding van acute babesiose bij een Nederlands draverveulen na verblijf in Normandië (A literature review of equine piroplasmosis after an episode of acute babesiosis in a Dutch Standardbred foal after a stay in Normandy). *Tijdschrift voor Diergeneeskunde* 130: 726-731.

Callow LL (1984) Equine babesiosis in Australia. In *Protozoal and rickettsial diseases* pp. 165-167. Australian Government Publishing Service, Canberra.

Callow LL, McGregor W, Rodwell BJ, Rogers RJ, Fraser GC, Mahoney DF, Robertson GM (1979) Evaluation of an indirect fluorescent antibody test to diagnose *Babesia equi* infection in horses. *Australian Veterinary Journal* 55: 555-559.

CFSPH (2008) Equine piroplasmosis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 28 January 2009).

Churchill RC, Best DR (1976) Babesiosis of a horse in Australia. *Australian Veterinary Journal* 52: 487.

Criado-Fornelio A, Martinez-Marcos A, Buling-Saraña A, Barba-Carretero JC (2003) Molecular studies on *Babesia*, *Theileria* and *Hepatozoon* in southern Europe: part I. epizootiological aspects. *Veterinary Parasitology* 113: 189-201.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

de Waal DT (1990) The transovarial transmission of *Babesia caballi* by *Hyalomma truncatum*. *Onderstepoort Journal of Veterinary Research* 57: 99-100.

de Waal DT, Horn E, Josemans AI (1999) Epidemiology of equine piroplasmosis in South Africa: detection and duration of colostral antibodies and the incidence of new infection in foals. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 592-593, R & W Publications (Newmarket) Limited, Newmarket.

de Waal DT, Potgeiter FT (1987) The transstadial transmission of *Babesia caballi* by *Rhipicephalus evertsi evertsi*. *Onderstepoort Journal of Veterinary Research* 54: 655-656.

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

de Waal DT, van Heerden J, Van Den Berg SS, Stegmann GF, Potgieter FT (1988) Isolation of pure *Babesia equi* and *Babesia caballi* organisms in splenectomized horses from endemic areas in South Africa. *The Onderstepoort Journal of Veterinary Research* 55: 33-35.

Diana A, Guglielmini C, Candini D, Pietra M, Cipone M (2007) Cardiac arrhythmias associated with piroplasmosis in the horse: a case report. *The Veterinary Journal* 174: 193-195.

DOACS (2009) Equine piroplasmosis (EP) update: February 20, 2009. Florida Department of Agriculture and Consumer Services: Division of Animal Industry. http://www.doacs.state.fl.us/ai/pdf/Equine_Piroplasmosis_Situation_Web_Update.pdf (Accessed 11 November 2008).

Donnelly J, Joyner LP, Graham-Jones O, Ellis CP (1980) A comparison of the complement fixation and immunofluorescent antibody tests in a survey of the prevalence of *Babesia equi* and *Babesia caballi* in horses in the Sultanate of Oman. *Tropical Animal Health and Production* 12: 50-60.

Friedhoff KT, Soulé C (1996) An account on equine babesiosis. *Revue Scientifique et Technique de l'Office International des Epizooties* 15: 1191-1201.

Friedhoff KT, Tenter AM, Muller I (1990) Haemoparasites of equines: impact on international trade of horses. *Revue Scientifique et Technique de l'Office International des Epizooties* 9: 1187-1194.

Gerstenberg C, Allen WR, Phipps LP (1999) Mechanical transmission of *Babesia equi* infection in a British herd of horses. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 217-222, R & W Publications (Newmarket) Limited, Newmarket.

Heuchert CMS, de Giulli VJr, de Athaide DF, Böse R, Friedhoff KT (1999) Seroepidemiologic studies on *Babesia equi* and *Babesia caballi* infections in Brazil. *Veterinary Parasitology* 85: 1-11.

Ikadai H, Nagai A, Xuan X, Igarashi I, Kamio T, Tsuji N, Oyamada T, Suzuki N, Fujisaki K (2002) Seroepidemiologic studies on *Babesia caballi* and *Babesia equi* infections in Japan. *The Journal of Veterinary Medical Science* 64: 325-328.

Ikadai H, Sasaki M, Ishida H, Matsuu A, Igarashi I, Fujisaki K, Oyamada T (2007) Molecular evidence of *Babesia equi* transmission in *Haemaphysalis longicornis*. *The American Journal of Tropical Medicine and Hygiene* 76: 694-697.

Kim C, Blanco LBC, Alhassan A, Iseki H, Yokoyama N, Xuan X, Igarashi I (2008) Diagnostic real-time PCR assay for the quantitative detection of *Theileria equi* from equine blood samples. *Veterinary Parasitology* 151: 158-163.

Kuttler KL, Goff WL, Gipson CA, Blackburn BO (1988) Serologic response of *Babesia equi*-infected horses as measured by complement-fixation and indirect fluorescent antibody tests. *Veterinary Parasitology* 26: 199-205.

Mehlhorn H, Schein E (1998) Redescription of *Babesia equi* Laveran 1901, as *Theileria equi* Mehlhorn, Schein 1998. *Parasitology Research* 84: 467-475.

Ogunremi O, Halbert G, Mainar-Jaime R, Benjamin J, Pfister K, Lopez-Rebollar L, Georgiadis MP (2008) Accuracy of an indirect fluorescent-antibody test and of a complement-fixation test for the diagnosis of *Babesia caballi* in field samples from horses. *Preventive Veterinary Medicine* 83: 41-51.

OIE (2008) Equine piroplasmosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.08_EQUINE_PIROPLASMOSIS.pdf (Accessed 1 September 2009).

OIE (2009a) Equine piroplasmosis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.8.htm (Accessed 1 September 2009).

OIE (2009b) Equine piroplasmosis, United States of America. World Animal Health Information System (WAHIS). http://www.oie.int/wahis/public.php?page=single_report&pop=1&reportid=8173 (Accessed 1 July 2009).

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Potgeiter FT, de Waal DT, Posnett ES (1992) Transmission and diagnosis of equine babesiosis in South Africa. *Memoirs of Instituto Oswaldo Cruz (Rio de Janeiro)* 87: 139-142.

Promed Mail (2000) Piroplasmosis, equine - Australia ex Hong Kong (03). ProMED Mail. http://www.promedmail.org (Accessed 28 January 2009).

Promed Mail (2008) Piroplasmosis, equine - USA (04): (Florida). ProMED Mail. http://www.promedmail.org (Accessed 28 January 2009).

Promed Mail (2009a) Equine piroplasmosis - Ireland. ProMED Mail. http://www.promedmail.org (Accessed 29 October 2009).

Promed Mail (2009b) Equine piroplasmosis - USA (02): (Missouri). ProMED Mail. http://www.promedmail.org (Accessed 29 October 2009).

Promed Mail (2009c) Equine piroplasmosis - USA (06): (Texas) OIE. ProMED Mail. http://www.promedmail.org (Accessed 29 October 2009).

Promed Mail (2009d) Equine piroplasmosis - USA: (Florida). ProMED Mail. http://www.promedmail.org (Accessed 29 October 2009).

Promed Mail (2009e) Piroplasmosis, equine - USA (03): (Kansas, Missouri). ProMED Mail. http://www.promedmail.org (Accessed 1 September 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1483-1540. Saunders Elsevier, Edinburgh.

Rothschild CM, Knowles DP (2007) Equine piroplasmosis. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 465-473. Saunders Elsevier, St. Louis.

Tenter AM, Friedhoff KT (1986) Serodiagnosis of experimental and natural *Babesia equi* and *B. caballi* infections. *Veterinary Parasitology* 20: 49-61.

Tenter AM, Otte MJ, Gonzalez CA, Abuabara Y (1988) Prevalence of piroplasmosis in equines in the Colombian province of Cordoba. *Tropical Animal Health and Production* 20: 93-98.

Thompson PH (1969) Ticks as vectors of equine piroplasmosis. *Journal of the American Veterinary Medical Association* 155: 454-457.

Zweygarth E, Just MC, de Waal DT (1997) *In vitro* cultivation of *Babesia equi*: detection of carrier animals and isolation of parasites. *Onderstepoort Journal of Veterinary Research* 64: 51-56.

5.17 Equine protozoal myeloencephalitis

5.17.1 Technical information

Background

Equine protozoal myeloencephalitits (EPM) is a parasitic disease caused primarily by the apicomplexan parasite *Sarcocystis neurona.* Definitive hosts of *S. neurona* are American opossums (*Didelphis virginiana* and *D. albiventris*). Another protozoan, *Neospora hughesi*, has also been associated with EPM in the United States. However, the hosts of *N. hughesi* remain unknown and therefore this chapter will only consider *S. neurona*. Any conclusions relating to *S. neurona* would apply equally to *N. hughesi,* as their geographic range and infection in horses are similar. EPM is the most common infectious neurological disease of horses in the United States (Sellon and Dubey 2007).

Neurological disease associated with *S. neurona* has been reported in cats, horses, mink, racoons, skunks, Pacific harbour seals and Southern sea otters, leading to the premise that some of these species could be intermediate hosts (Stanek et al. 2003; Sellon and Dubey 2007). Clinical disease in horses has been reported from Brazil, Canada, Panama and the United States (Dubey et al. 2001).

Cases of the disease have been associated with horses exported from the United States to Hong Kong (Lam et al. 1999), France (Pitel et al. 2003), Japan (Katayama et al. 2003) and India (Brown et al. 2006) — countries where the definitive host is not known to be present.

A single Indian-born horse reported as positive to *S. neurona* antibodies may have been due to misclassification or the presence of cross-reacting antibodies (Brown et al. 2006). EPM-like cases associated with *S. neurona* antibodies have also been reported in horses that had never left France. However, this was most likely attributed to contamination of food imported from the United States or exposure to other *Sarcocystis* species that cross-reacted with *S. neurona* (Pitel et al. 2003).

EPM is not an OIE-listed disease (OIE 2009).

Epidemiology

Definitive hosts are infected by ingesting sarcocyst-containing tissue of intermediate hosts (MacKay et al. 2000). Ingested sarcocysts undergo sexual reproduction in the intestine of the definitive host and sporocysts are excreted in their faeces. Intermediate hosts ingest infective sporocysts and after a series of asexual reproductive cycles, sarcocysts locate in skeletal muscle (MacKay et al. 2000). Horses are infected through ingestion of feed or water contaminated with sporocyst-containing opossum faeces (Sellon and Dubey 2007). Despite extensive research, sarcocysts had not been demonstrated in horses (Dubey et al. 2001). Horses are considered aberrant intermediate hosts as the infective stage migrates to the central nervous system (CNS) and does not encyst in muscle. Therefore, horses cannot be involved in completion of the life cycle, even if ingested, as no cysts are present in muscle tissue. Although finding mature, intact *S. neurona* schizonts and sarcocysts in muscle tissues of a single horse has been reported (Mullaney et al. 2005), Koch's postulates were not

fulfilled in the case study. There is no evidence of transplacental transmission in horses (Duarte et al. 2004).

Occurrence of disease is sporadic in endemic areas although outbreaks confined to individual farms have been recorded. Seroprevalence of *S. neurona* infection in horses is 30–50% in the United States and Argentina (Dubey et al. 2001), and 36–69.6% in Brazil (Dubey et al. 1999; Hoane et al. 2006).

Clinical signs

In experimental infection, the incubation period is 28–42 days (Sellon and Dubey 2007). However, it is likely many horses do not show clinical signs of disease for months to years after infection. Clinical signs of disease can progress rapidly or remain stable for a prolonged period (Sellon and Dubey 2007). Neurological signs vary depending on the area of CNS involvement. Most horses are bright and alert but focal muscle asymmetry can occur. Gait abnormalities are the usual presenting signs. Common clinical signs of brain or brain-stem involvement are depression, facial paralysis, head tilt and dysphagia. Some neurological lesions can present as behavioural or training problems. Most horses with EPM suffer from a gradual progression of the disease — both in severity and range of clinical signs of infection (Sellon and Dubey 2007). The annual incidence of EPM in the United States is approximately 14 cases per 10 000 horses. The reported case fatality rate is about 7% (NAHMS 2001).

Treatment with antiprotozoal drugs is prolonged and relapses can occur when treatment ends (Radostits et al. 2007).

Diagnosis

Definitive diagnosis can be difficult due to the high prevalence of antibody to *S. neurona* in North American horses (Sellon and Dubey 2007). Ante mortem diagnostic tests based on detection of antibody to *S. neurona* in cerebrospinal fluid or serum are not definitive. The gold standard diagnostic test is post mortem identification of the characteristic lesions and parasites within the CNS (Sellon and Dubey 2007).

Conclusion

EPM is present in some approved countries. The definitive host of *S. neurona* is not present in Australia and horses are considered aberrant dead-end hosts.

EPM was not considered further in the IRA.

References

Brown CM, Morrow JK, Carleton CL, Ramanathan B, Reddy R, Vaidya V, Karthikeyan SM, Zulfikar AA, Kannadkar VS (2006) Persistence of serum antibodies to *Sarcocystis neurona* in horses moved from North America to India. *Journal of Veterinary Internal Medicine* 20: 994-997.

Duarte PC, Conrad PA, Barr BC, Wilson WD, Ferraro GL, Packham AE, Carpenter TE, Gardner IA (2004) Risk of transplacental transmission of S*arcocystis neurona* and *Neospora hughesi* in California horses. *The Journal of Parasitology* 90: 1345-1351.

Dubey JP, Kerber CE, Granstrom DE (1999) Serologic prevalence of *Sarcocystis neurona*, *Toxoplasma gondii*, and *Neospora caninum* in horses in Brazil. *Journal of the American Veterinary Medical Association* 215: 970-972.

Dubey JP, Lindsay DS, Saville WJA, Reed SM, Granstrom DE, Speer CA (2001) A review of *Sarcocystis neurona* and equine protozoal myeloencephalitis (EPM). *Veterinary Parasitology* 95: 89-131.

Hoane JS, Gennari SM, Dubey JP, Ribeiro MG, Borges AS, Yai LEO, Aguiar DM, Cavalcante GT, Bonesi GL, Howe DK (2006) Prevalence of *Sarcocystis neurona* and *Neospora* spp. infection in horses from Brazil based on presence of serum antibodies to parasite surface antigen. *Veterinary Parasitology* 136: 155-159.

Katayama Y, Wada R, Kanemaru T, Sasagawa T, Uchiyama T, Matsumura T, Anzai T (2003) First case report of *Sarcocystis neurona*-induced equine protozoal myeloencephalitis in Japan. *The Journal of Veterinary Medical Science* 65: 757-759.

Lam KKH, Watkins KL, Chan CW (1999) First report of equine protozoal myeloencephalitis in Hong Kong. *Equine Veterinary Education* 11: 54-56.

MacKay RJ, Granstrom DE, Saville WJ, Reed SM (2000) Equine protozoal myeloencephalitis. *Veterinary Clinics of North America: Equine Practice* 16: 405-425.

Mullaney T, Murphy AJ, Kiupel M, Bell JA, Rossano MG, Mansfield LS (2005) Evidence to support horses as natural intermediate hosts for *Sarcocystis neurona*. *Veterinary Parasitology* 133: 27-36.

NAHMS (2001) *Equine protozoal myeloencephalitis (EPM) in the U.S.* No. #N312.0501, National Animal Health Monitoring System, United States Department of Agriculture, Fort Collins.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pitel PH, Linsay DS, Caure S, Romand S, Pronost S, Gargala G, Michell SM, Hary C, Thulliez P, Fortier G, Ballet JJ (2003) Reactivity against *Sarcocystis neurona* and *Neospora* by serum antibodies in healthy French horses from two farms with previous equine protozoal myeloencephalitis-like cases. *Veterinary Parasitology* 111: 1-7.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1483-1540. Saunders Elsevier, Edinburgh.

Sellon DC, Dubey JP (2007) Equine protozoal myeloencephalitis. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 453-464. Elsevier, St. Louis.

Stanek JF, Stich RW, Dubey JP, Reed SM, Njoku CJ, Lindsay DS, Schmall LM, Johnson GK, LaFave BM, Saville WJA (2003) Epidemiology of *Sarcocystis neurona* infections in domestic cats (*Felis domesticus*) and its association with equine protozoal myeloencephalitis (EPM) case farms and feral cats from a mobile spay and neuter clinic. *Veterinary Parasitology* 117: 239-249.

5.18 Equine viral arteritis

5.18.1 Technical information

Background

Equine viral arteritis (EVA) is caused by equine arteritis virus (EAV), the prototype of the genus Arterivirus, family Arteriviridae, order Nidovirales (Snijder et al. 2005). EVA is characterised by panvasculitis, respiratory disease and abortion (Del Piero 2000). The only countries in which EAV has not been reported are Iceland, Japan and Singapore (Timoney and McCollum 1993a). EVA is a disease of horses. Infection of donkeys and mules has been reported (McCollum et al. 1995; Paweska et al. 1996; Turnbull et al. 2002), and of an alpaca that aborted in Germany (Weber et al. 2006). However, there is no evidence that donkeys and other equids play a significant role in the epidemiology of EVA (Paweska et al. 1997).

EVA is an OIE-listed disease (OIE 2009b).

Epidemiology

There is only one known serotype of EAV, but several strains differ in abortigenic potential, virulence in the respiratory and reproductive tracts, and in the severity of the clinical disease they cause (Murphy et al. 1992; Timoney and McCollum 1993a; Balasuriya et al. 1999a; Balasuriya et al. 1999b; Belák et al. 1999). Acutely infected horses shed EAV in body secretions and excretions for up to 21 days after infection (McCollum et al. 1971; Neu et al. 1988).

The most common routes of transmission are via semen and the respiratory tract (Timoney and McCollum 1993a) and both can occur in an outbreak. The carrier stallion does not shed virus in respiratory secretions or urine but constantly sheds EAV in semen after recovery from infection (Timoney et al. 1987). Venereal transmission during natural service or artificial insemination is the primary method of infection on breeding farms and transmission rates can be as high as 85–100% (Glaser et al. 1997).

In the 2006 United States outbreak, the principal mode of transmission on the index farm was respiratory via an infected pregnant mare who aborted. The mare also infected a stallion and subsequent venereal transmission led to widespread dissemination of the virus via cool-shipped semen and mare and foal transport, to 18 states, affecting over 200 horses in boarding stables, private farms, a breeding facility, training stables and a veterinary clinic (Timoney et al. 2006).

In 2007 an infected Percheron stallion in France spread EAV to other stallions. Before the disease was recognised and control measures implemented, cool-shipped semen from the infected stallions spread EVA to 26 farms causing neonatal deaths, abortions, the development of persistently infected stallions and clinical cases of disease (Holyoak et al. 2008).

The carrier state occurs in 30–70% of stallions exposed to EAV and constitutes the natural reservoir of the virus (Timoney et al. 1986; Timoney et al. 1987; Balasuriya et al. 1998). Duration of the carrier state varies from months to life in mature stallions — with no adverse effects on stallion health or fertility (Holyoak et al. 1993).

Testosterone plays an essential role in the establishment and maintenance of the carrier state in stallions (Timoney et al. 1986). Suppression of testosterone production by immunisation against gonadotrophin releasing hormone (GnRH) is being investigated as a means of eliminating the carrier state in stallions (Timoney 2002; Burger et al. 2005). While temporary down-regulation of circulating testosterone levels using a GnRH antagonist or by immunisation with GnRH would appear to have expedited clearance of the carrier state in some stallions, the efficacy of either treatment strategy has yet to be fully established. Concern has been expressed that such a therapeutic approach could be used to deliberately mask existence of the carrier state (OIE 2008b).

There is no evidence of a carrier state in mares, foetuses, foals under six months of age, or geldings (Timoney and McCollum 1988). Congenital infections can occur and the placenta, placental fluids and foetus are sources of virus (Vaala et al. 1992).

EAV can also be transmitted by fomites and personnel (Timoney and McCollum 1993a). Horizontal transmission can occur between stallions via contamination of bedding or fomites with EAV-infected semen (Guthrie et al. 2003; Metz et al. 2008).

Typically in outbreaks of EVA, virus is transmitted venereally — via natural service or artificial insemination — from a carrier stallion to a seronegative mare. The mare develops respiratory illness and transmits the virus horizontally via aerosol, fomites and personnel. Aerosol transmission occurs via respiratory secretions, urine and other body secretions of acutely infected horses, aborted foetuses and their membranes, and the masturbates of acutely and chronically infected stallions (Balasuriya and MacLachlan 2007).

Outbreaks are controlled by quarantine and surveillance. The amount of time required for, and the inherent difficulties in, diagnosing EVA (Holyoak et al. 2008) can allow the virus to be widely disseminated before control measures are implemented. This occurred in the United States where 22 days elapsed between the first abortion and the diagnosis of EVA — allowing the virus to spread to 18 states and affect 200 horses in boarding stables, private farms, a breeding facility, training stables and a veterinary clinic, before the spread was controlled by quarantine and close surveillance. Although both the primary and the index case were quarter horses, the virus spread to warmbloods, paint horses, arabians and thoroughbreds. Approximately 70% of direct exposures resulted from the use of fresh-cooled semen from one particular quarter horse stallion, and 30% resulted from movement of mares from the index farm to other premises (Timoney et al. 2006).

The level of EAV infection within breeds and populations is determined by the number of carrier stallions, and there is considerable variation in seroprevalence between countries and breeds (Holyoak et al. 2008). Studies in 1996 reported that in Austria, 27% of horses were seropositive and 3.8% of abortions were attributed to EAV; in France, 1–3% of horses were seropositive; in Germany, seroprevalence was 1.8% in 1988 and 24.8% in 1994; in Ireland, 0.3% — and clinical disease had never been reported; in the Netherlands, up to 45% of horses older than four years of age were seropositive; in Sweden, 35% of standardbreds and 16% of warmbloods were seropositive; in the United Kingdom, seroprevalence increased to 2–3%, from 0.5% prior to 1993 when the first outbreak of disease was recorded there, and in the United States, 15–30% of horses were seropositive to EAV (Timoney and Edwards 1996). Reports of outbreaks in North America and Europe have increased over the last

decade with the most recent outbreaks of clinical disease in the United States (Timoney et al. 2006) and France (Animal Health Trust 2007). This may reflect the increase in international trade in horses and semen (Metcalf et al. 1996; Timoney 2000).

Although no clinical cases of EVA have been diagnosed (OIE 2008a), there is serological evidence that EAV has been circulating in the Australian horse population since before 1975 (Huntington et al. 1990; Animal Health Australia 2008). In addition, there is historical evidence that clinical cases and deaths due to EVA may have occurred intermittently in Australia between 1842 and 1912 (Ellis 2000). There have been no reports of clinical disease since 1912 and the strain of virus in Australia today is thought to be avirulent (Zheng and Sabine 1989; Ellis 1999).

Standardbreds and warmbloods exhibit the highest prevalence of EAV worldwide (Huntington et al. 1990; Burki et al. 1992; Timoney and McCollum 1993b). In Australia, serological and virological investigations found 8% of thoroughbred and 72.5% of standardbred stallions, and 0.8% of thoroughbred and 71.2% of standardbred mares, were seropositive (Huntington et al. 1990).

There is strong circumstantial evidence that genetic and phenotypic divergence can occur during persistent EAV infection of carrier stallions (Hedges et al. 1999). Major outbreaks are thought to be the result of periodic emergence of novel genetic and phenotypic variants of the virus (Balasuriya et al. 1999a). Emergence of strains of the virus with the ability to cause clinical illness, including abortion, remains an unpredictable event, the basis of which has not been elucidated (Timoney and McCollum 1993a). Further studies are planned to investigate whether viral carriage in the stallion is a source of variants with enhanced pathogenicity (P. Timoney, University of Kentucky, pers. comm. February 2009). Results from ongoing serological testing indicate that Australian horses have antibodies to EAV, but there is no information on the breed prevalence, the strain of virus or the vaccination history (Animal Health Australia 2008). The strain of EAV endemic to Australia appears to be low virulence and eradication has not been attempted. EVA is a nationally notifiable disease in Australia (DAFF 2008) and quarantine restrictions are maintained to prevent the introduction of pathogenic strains (Ellis 2000).

Clinical signs

The incubation period is 2–14 days; however, the majority of cases are subclinical, especially in mares bred to carrier stallions (Cole et al. 1986; Timoney and McCollum 1993a; Glaser et al. 1997). In the 2006 outbreak in the United States, the disease was only detected when a 50% pregnancy loss found during routine 60 day pregnancy examinations, prompted the stallion owner to seek veterinary advice. Diagnosis took a further 19 days (Timoney et al. 2006).

Clinical outbreaks are characterised by any of the following: abortion at 3–10 months gestation, severe interstitial pneumonia or enteritis in neonates, systemic illness in adult horses and persistent infection in stallions. Abortion occurs 3–8 weeks after infection (Cole et al. 1986), may not be preceded by clinical signs of EVA in the mare, and rates vary from 10% to 60%. There are no resultant adverse effects on mare fertility (Timoney and McCollum 1993a).

The most consistent clinical features of EVA are pyrexia of 2–9 days duration, and leukopaenia (Timoney and McCollum 1993a). Other signs vary in range and severity,

and between outbreaks — depending on the strain and dose of virus, age and physical condition of the animal, mode of infection and environmental conditions (Timoney and McCollum 1993a). They occur in 10% of clinical cases and include rhinitis, conjunctivitis, anorexia, depression, urticaria and oedema of the limbs, peri-orbital area, mammary glands, scrotum and prepuce. Mortality in natural cases of EVA is rare, but may occur in congenitally infected foals (Vaala et al. 1992; McCollum et al. 1999). Mortalities in newborn foals following severe pneumonia, oedema and pyrexia were reported on studs where the disease was introduced via infected semen (Fortier et al. 2008). During acute infections stallions may undergo a period of reduced fertility for up to 16 weeks (Timoney and McCollum 1993a).

Diagnosis

EVA can be confused clinically with several other diseases. Diagnosis should be confirmed by any combination of virus isolation, viral nucleic acid or antigen detection, and serology (Timoney and McCollum 1996; Timoney 2000). EAV can be isolated from body fluids (nasopharyngeal washings, blood, semen and foetal fluids) and tissues as early as two days and up to 60 days post-infection (de Vries et al. 1996; Glaser et al. 1997). Paired sera can be tested for either seroconversion or a fourfold or greater increase in serum antibody titre.

The virus neutralisation (VN) assay is used for detection of serum antibodies to EAV (Holyoak et al. 2008), however there are significant difficulties in test interpretation. These difficulties arise from the use of related, tissue-culture cell lines for growing vaccines and as indicator cells in diagnostic tests — the EAV VN assay recommended by the OIE (OIE 2008b) uses rat kidney (RK-13) indicator cells. These cells are also used for the production of a widely used equid herpesvirus vaccine (HBLB 2007). Horses that have been vaccinated with the herpesvirus vaccine can develop an antibody response against RK-13 cells, which causes cytopathic effects (CPE) in the RK-13 cells used in the EAV VN assay. These CPE can affect up to 73% of tests, making them uninterpretable (Newton et al. 2004). Modifications to the VN assay based on RK-13 cells and alternative VN methods have been developed which reduce the level of cytotoxicity (P. Timoney, University of Kentucky, pers. comm. February 2009). Polymerase chain reaction assays require further standardisation and validation before they can be adopted as a reliable screening assay (Lu et al. 2008).

The carrier state can be confirmed either by isolation of EAV or detection of viral nucleic acid in a sample of semen containing the sperm-rich fraction of the ejaculate (Timoney et al. 1987). Alternatively, suspect stallions can be test mated to two mares that are then monitored clinically and serologically for up to 28 days.

Immunology

Both natural and experimental infection — with either virulent or avirulent strains of EAV — results in long-lasting immunity against reinfection with all strains (Doll et al. 1968; McCollum 1986).

A modified-live virus attenuated vaccine (ARVAC, Fort Dodge Animal Health, Iowa) is licensed for use in the United States and Canada for prevention of EVA infection in horses. A killed virus vaccine (Artervac, Fort Dodge Animal Health, Iowa) is licensed for use in Denmark, France, Germany, Hungary, Ireland, Sweden and the United Kingdom. Vaccinated horses are serologically indistinguishable from naturally exposed horses and therefore evidence of serological status prior to vaccination is

important for breeding and export purposes (Timoney 2002). Vaccination protects against clinical EVA but does not consistently prevent infection of vaccinated horses or subsequent limited replication of field strains of the virus. Horses can shed infectious virus for up to 28 days after they have been vaccinated. To prevent infection of seronegative horses, vaccinated horses should be isolated for 21 days immediately after they have been vaccinated (OIE 2009a).

The carrier state has never been confirmed in a seronegative stallion and there is no evidence that a vaccinated stallion will develop the carrier state with vaccine virus (Timoney et al. 1987). Stallions with a titre of 1:4 or greater without a recommended vaccination history should be considered carriers until shown otherwise (Timoney et al. 1987). Testosterone plays an essential role in the establishment and maintenance of the carrier state in stallions (Timoney et al. 1986). Suppression of testosterone production by immunisation against GnRH is being investigated as a means of eliminating the carrier state in stallions (Timoney 2002; Burger et al. 2005).

The protective immunisation of prepubertal colts is central to effective control of the spread of EVA infection (Balasuriya and MacLachlan 2007). Maternal antibodies disappear between two and six months of age and it is recommended that foals be vaccinated at 180-270 days of age, before the onset of puberty (Hullinger et al 1998).

Conclusion

Strains of EAV are present in approved countries and in Australia. The strains present in Australia are low virulence and quarantine restrictions are maintained to prevent the introduction of pathogenic strains. EVA is a nationally notifiable disease and there is ongoing surveillance. The Code recommendations (OIE 2009a) include isolation, diagnostic testing and vaccination. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Animal Health Australia (2008) Equine viral arteritis: serological testing for equine viral arteritis from routine submission to state laboratories (summary). National Animal Health Information System (NAHIS). http://www.animalhealthaustralia.com.au/NAHIS/public.php?page=out_showtable&outputid=33&sm=7&sy=2007&em=1&ey=2009&date_submit=OK (Accessed 9 January 2009).

Animal Health Trust (2007) Interim report - June 2007: France: equine viral arteritis (EVA). International Breeders' Meeting: International Collating Centre. http://www.aht.org.uk/icc/Interim_Report4_june2007.html (Accessed 2008).

Balasuriya BDR, Everman JF, Hedges JF, McKiernan AJ, Mitten JQ, Beyer JC, McCollum WH, Timoney PJ, MacLachlan NJ (1998) Serologic and molecular characterisation of an abortigenic strain of equine arteritis virus isolated from infective frozen semen and an aborted equine fetus. *Journal of the American Veterinary Medical Association* 213: 1586-1589.

Balasuriya UBR, Hedges JF, Nadler SA, McCollum WH, Timoney PJ, MacLachlan NJ (1999a) Genetic stability of equine arteritis virus during horizontal and vertical transmission in an outbreak of equine viral arteritis. *Journal of General Virology* 80: 1949-1958.

Balasuriya UBR, Hedges JF, Patton JF, Timoney PJ, McCollum WH, Hullinger PJ, MacLachlan NJ (1999b) Genetic variation of equine arteritis virus: role of the carrier stallion in emergence of novel genotypes. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) p. 520, R & W Publications (Newmarket) Limited, Newmarket.

Balasuriya UBR, MacLachlan NJ (2007) Equine viral arteritis. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 153-164. Saunders Elsevier, St. Louis.

Belák S, Stadejek T, Bjorklund H, Ros Bascunana C, Ciabatti IM, Scicluna MT, Amaddeo D, McCollum WH, Paton DJ, Autorino GL, Timoney PJ, Klingeborn B (1999) Genetic diversity among field isolates of equine arteritis virus. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 177-183, R & W Publications (Newmarket) Limited, Newmarket.

Burger D, Vidament M, Janett F, Fortier G, Zientara S, Timoney PJ, Thun R (2005) Treatment of equine arteritis virus-shedding stallions by immunization against GnRH. In *9th Congress on Equine Medicine and Surgery in Geneva, December 13-15, 2005,* p. 163, International Veterinary Information Service, Ithaca.

Burki F, Hofer A, Nowotny N (1992) Objective data plead to suspend import-bans for seroreactors against equine arteritis virus except for breeder stallions. *Journal of Applied Animal Research* 1: 31-42.

Cole JR, Hall RF, Gosser HS, Hendricks JB, Pursell AR, Senne DA, Pearson JE, Gipson CA (1986) Transmissibility and abortogenic effect of equine viral arteritis in mares. *Journal of the American Veterinary Medical Association* 189: 769-717.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

de Vries AAF, Rottier PJM, Glaser AL, Horzinek MC (1996) Equine viral arteritis. In *Virus infections of equines* (ed. Studdert MJ) pp. 171-200. Elsevier Science Publishers, Amsterdam.

Del Piero F (2000) Equine viral arteritis. *Veterinary Pathology* 37: 287-296.

Doll ER, Bryans JT, Wilson JC, McCollum WH (1968) Immunization against equine viral arteritis using modified live virus propagated in cell cultures of rabbit kidney. *The Cornell Veterinarian* 48: 497-524.

Ellis P (1999) Viral diseases of horses in Australia. In *1999 world trotting conference and driving championship, Australia, 20th - 27th November 1999,* p. 1, International Trotting Association, Canada.

Ellis PM (2000) The beauty of isolation. pp. 1-5. Department of Natural Resources and Environment, Attwood.

Fortier G, Miszczak F, Pitel P-H, Fortier C, Legrand L, Tapprest J, Hamon M, Marcillaud-Pitel C, Pronost S (2008) Equine viral arteritis: detection and characterization of high virulence strains in France (Normandy) during 2007 summer. In *Proceedings of the 10th international congress of World Equine Veterinary Association, Jan 28-Feb 1, 2008, Moscow, Russia,* pp. 573-574, International Veterinary Information Service, [Moscow].

Glaser AL, Chirnside ED, Horzinek MC, de Vries AAF (1997) Equine arteritis virus. *Theriogenology* 47: 1275-1295.

Guthrie AJ, Howell PG, Hedges JF, Bosman AM, Balasuriya UBR, McCollum WH, Timoney PJ, MacLachlan NJ (2003) Lateral transmission of equine arteritis virus amongst Lipizzaner stallions in South Africa. *Equine Veterinary Journal* 35: 596-600.

HBLB (2007) Codes of Practice on equine diseases. Horserace Betting Levy Board. http://www.hblb.org.uk/ (Accessed 3 August 2007).

Hedges JF, Balasuriya UBR, Timoney PJ, McCollum WH, MacLachlan NJ (1999) Genetic divergence with emergence of novel phenotypic variants of equine arteritis virus during persistent infection of stallions. *Journal of Virology* 73: 3672-3681.

Holyoak GR, Balasuriya UBR, Broaddus CC, Timoney PJ (2008) Equine viral arteritis: current status and prevention. *Theriogenology* 70: 403-414.

Holyoak GR, Little TV, McCollum WH, Timoney PJ (1993) Relationship between onset of puberty and establishment of persistent infection with equine arteritis virus in the experimentally infected colt. *Journal of Comparative Pathology* 109: 29-46.

Huntington PJ, Forman AJ, Ellis PM (1990) The occurrence of equine viral arteritis virus in Australia. *Australian Veterinary Journal* 67: 432-435.

Lu Z, Branscum AJ, Shuck KM, Zhang J, Dubovi EJ, Timoney PJ, Balasuriya UBR (2008) Comparison of two real-time reverse transcription polymerase chain reaction assays for the detection of Equine arteritis virus nucleic acid in equine semen and tissue culture fluid. *Journal of Veterinary Investigations* 20: 147-155.

McCollum WH (1986) Responses of horses vaccinated with avirulent modified-live equine arteritis virus propagated in the E. Derm (NBL-6) cell line to nasal inoculation with virulent virus. *American Journal of Veterinary Research* 47: 1931-1934.

McCollum WH, Prickett ME, Bryans JT (1971) Temporal distribution of equine arteritis virus in respiratory mucosa, tissues and body fluids of horses infected by inhalation. *Research in Veterinary Science* 12: 459-464.

McCollum WH, Timoney PJ, Lee JW, Jr., Habacker PL, Balasuriya UBR, MacLachlan NJ (1999) Features of an outbreak of equine viral arteritis on a breeding farm associated with abortion and fatal interstitial pneumonia in neonatal foals. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF,

Mumford JA, Kaaden O.R.) pp. 559-560, R & W Publications (Newmarket) Limited, Newmarket.

McCollum WH, Timoney PJ, Tengelsen LA (1995) Clinical, virological and serological responses of donkeys to intranasal inoculation with the KY-84 strain of equine arteritis virus. *Journal of Comparative Pathology* 112: 207-211.

Metcalf HE, Blackwell JH, Acree JA (1996) Application of risk assessment to international trade in animals and animal products. *Annals of the New York Academy of Sciences* 791: 280-295.

Metz GE, Serena MS, Ocampos GM, Panei CJ, Fernandez VL, Echeverría MG (2008) Equine arteritis virus: a new isolate from the presumable first carrier stallion in Argentina and its genetic relationships among the four reported unique Argentinean strains. *Archives of Virology* 153: 2111-2115.

Murphy TW, McCollum WH, Timoney PJ, Klingeborn BW, Hyllseth B, Golnik W, Erasmus B (1992) Genomic variability among globally distributed isolates of equine arteritis virus. *Veterinary Microbiology* 32: 101-115.

Neu SN, Timoney PJ, McCollum WH (1988) Persistent infection of the reproductive tract in stallions experimentally infected with equine arteritis virus. In *Equine infectious diseases V: proceedings of the fifth international conference, October 7-10, 1987, Lexington, Kentucky,* (ed. Powell DG) pp. 149-154, The University Press of Kentucky, Lexington.

Newton JR, Geraghty RJ, Castillo-Olivares J, Cardwell JM, Mumford JA (2004) Evidence that use of an inactivated equine herpesvirus vaccine induces serum cytotoxicity affecting the equine arteritis virus neutralisation test. *Vaccine* 22: 4117-4123.

OIE (2008a) Disease timelines: equine viral arteritis. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_timelines&disease_type=Terrestrial&disease_id=65&empty=999999 (Accessed 2 January 2009).

OIE (2008b) Equine viral arteritis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.10_EVA.pdf (Accessed 1 September 2009).

OIE (2009a) Equine viral arteritis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/en_chapitre_1.12.10.pdf (Accessed 19 August 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Paweska JT, Aitcheson H, Chirnside ED, Barnard BJH (1996) Transmission of the South African asinine strain of equine arteritis virus (EAV) among horses and between donkeys and horses. *Onderstepoort Journal of Veterinary Research* 63: 189-196.

Paweska JT, Binns MM, Woods PS, Chirnside ED (1997) A survey for antibodies to equine arteritis virus in donkeys, mules and zebra using virus neutralisation (VN) and enzyme linked immunosorbent assay (ELISA). *Equine Veterinary Journal* 29: 40-43.

Snijder EJ, Brinton MA, Faaberg KS, Godeny EK, Gorbalenya AE, MacLachlan NJ, Mengeling WL, Flanagan PGW (2005) Arteriviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 965-974. Elsevier, San Diego.

Timoney PJ (2000) The increasing significance of international trade in equids and its influence on the spread of infectious diseases. *Annals of the New York Academy of Sciences* 916: 55-60.

Timoney PJ (2002) Equine viral arteritis. Equine Respiratory Diseases. http://www.ivis.org (Accessed 2002).

Timoney PJ, Creekmore L, Meade B, Fly D, Rogers E, King B (2006) 2006 multi-state occurrence of EVA. *The American Veterinary Medical Association* 213: 1586-1589.

Timoney PJ, Edwards S (1996) *Report of the first European symposium on equine viral arteritis*. Office International des Epizooties, Paris.

Timoney PJ, McCollum WH (1988) Equine viral arteritis: epidemiology and control. *Journal of Equine Veterinary Science* 8: 54-59.

Timoney PJ, McCollum WH (1993a) Equine viral arteritis. *Veterinary Clinics of North America: Equine Practice* 9: 295-309.

Timoney PJ, McCollum WH (1993b) Equine viral arteritis in perspective in relation to international trade. *Journal of Equine Veterinary Science* 13: 50.

Timoney PJ, McCollum WH (1996) Equine viral arteritis. *Equine Veterinary Education* 8: 97-100.

Timoney PJ, McCollum WH, Murphy TW, Roberts AW, Willard JG, Carswell GG (1987) The carrier state in equine arteritis virus infection in the stallion with specific emphasis on the venereal mode of virus transmission. *Journal of Reproduction and Fertility* 35: 95-102.

Timoney PJ, McCollum WH, Roberts AW (1986) Detection of the carrier state in stallions persistently infected with equine arteritis virus. In *Proceedings of the 32nd annual convention of the American Association of Equine Practitioners,* pp. 57-65, American Association of Equine Practitioners, Lexington.

Turnbull A, Wernery U, Wernery R, Anandh JP, Kinne J (2002) Survey of six infectious diseases of feral donkeys in the United Arab Emirates. *Equine Veterinary Education* 14: 33-38.

Vaala WE, Hamir AN, Dubovi EJ, Timoney PJ, Ruiz B (1992) Fatal, congenitally acquired infection with equine arteritis virus in a neonatal thoroughbred. *Equine Veterinary Journal* 24: 155-158.

Weber H, Beckmann K, Haas L (2006) Case report: equine arteritis virus (EAV) as the cause of abortion in alpacas? *Deutsche Tierärztliche Wochenschrift* 113: 162-163. (Abstract only)

Zheng M, Sabine M (1989) Equine viral arteritis. In Virology for Australian veterinarians: papers prepared for a course arranged by the Veterinary Science Post-Graduate Committee at the University of Queensland, 10-13 July 1989 (eds. Spradbrow P, Sabine M) pp. 227-229. University of Queensland, Veterinary Science Post-Graduate Committee, Brisbane.

5.19 Equine viral encephalitides

5.19.1 Technical information

Background

Equine viral encephalitides, also known as equine encephalomyelitides, caused by Eastern, Western and Venezuelan equine encephalitis (EEE, WEE and VEE, respectively) viruses, are arthropod-borne infections of horses and humans. EEE and WEE viruses occasionally also cause disease in birds and other mammals (Geering et al. 1995). They are Alphaviruses belonging to the family Togaviridae (Weaver et al. 2005).

WEE occurs in Canada, Central and South America and the United States (Zacks and Paessler 2009). There are two variants of EEE virus — one found in Canada and the United States, and the other in Central and South America. The North American variant is more pathogenic. VEE viruses are divided into epidemic and endemic groups. Epidemic VEE virus is found in Central and South America. The United States is free of the epidemic variant of VEE virus — the last reported epidemics of VEE were during 1969–1972, when outbreaks of VEE spread from Central and South America and extended into adjacent countries, including the United States (Weaver et al. 2004). Endemic VEE virus is found Central and South America and in parts of the United States.

EEE, WEE and VEE are OIE-listed diseases (OIE 2009b).

Epidemiology

EEE and WEE viruses are maintained in nature by alternating cycles of infection in birds and mosquitoes. EEE virus is transmitted by mosquitoes belonging to *Culiseta* spp. and WEE virus by *Culex* spp.

Mosquitoes become infected by feeding on a viraemic host. Although the infected mosquito is only able to transmit the virus for up to ten days, infection persists in the mosquito (Griffin 2007). EEE and WEE viruses cannot survive outside the host. Disease occurs when mosquito numbers increase in summer in temperate climates, and in the wet season in tropical and subtropical climates. Birds are the major amplifying hosts, with horses considered to be dead-end hosts for both viruses (Radostits et al. 2007).

VEE viruses are divided into endemic and epidemic groups. Subtypes ID, IE and IF are the endemic viruses and IAB, AC and IE are the epidemic viral subtypes (Griffin 2007). Sequencing of the endemic subtype ID and the epidemic IC subtype has revealed similarities between the two, suggesting that the epidemic subtype has mutated from an endemic ID virus (Griffin 2007). The endemic viruses are found in natural cycles between marsupials and sylvatic rodents, and mosquitoes, mostly *Culex* spp. The endemic (sylvatic) subtypes are generally found in limited geographic areas. They are normally not pathogenic for horses, and are usually not amplified in horses — however there is one report of an outbreak of VEE in horses in Mexico in 1993 caused by an endemic subtype (CFSPH 2008). Horses are dead-end hosts for the

endemic viruses (CFSPH 2008). In contrast, the epidemic viruses are amplified in equids and are responsible for most epidemics.

Vectors commonly isolated during epidemics are *Ochlerotatos sollicitans*, *Oc. taeniorhyncus, Psorophara columbiae* and *P. confinnis* (Griffin 2007). Epidemics could be related to mutations resulting in increased infectivity for the mosquito vector *Oc. taeniorhyncus* or the level of viraemia in horses (Griffin 2007). The epidemic viruses are amplified in horses during outbreaks producing sufficiently high viraemia for infection of mosquitoes and transmission (Griffin 2007). Transplacental transmission has been reported to occur experimentally (Justines et al. 1980). The natural host between epidemics has not been identified.

Clinical Signs

The incubation period for EEE and WEE is 1–14 days, and for VEE is 1–5 days. Infection with EEE and WEE viruses can be clinical or subclinical (CFSPH 2008). Clinical infections are characterised by a sudden onset of pyrexia, anorexia and depression. In severe cases, this is followed by encephalitis. Some horses exhibit periods of excitement or severe pruritus. Death can occur within a few days, mainly with EEE virus infections. A large number of horses that survive have residual neurological deficits. Morbidity varies according to the season and number of insect vectors present, but prevalence of infection is higher than clinical disease (Radostits et al. 2007). The case fatality rate in equids ranges from 20% to 30% in infection with WEE virus and from 40% to 80% in EEE virus infections (Radostits et al. 2007).

Infection with endemic forms of VEE virus causes subclinical disease in horses. Infection with epidemic forms of the virus causes clinical signs similar to EEE and WEE viruses. VEE virus can also cause severe respiratory disease in horses. Deaths can occur soon after the onset of neurological signs or after prolonged illness. Recovered animals can exhibit permanent neurological deficits (CFSPH 2008). Equine mortality rates range from 19% to 83% (Powers et al. 1997).

Diagnosis

A presumptive diagnosis of the equine viral encephalitides can be based on clinical signs. All three diseases can be confirmed by isolation of virus or serology. Viruses can be identified and classified antigenically by complement fixation, haemagglutination inhibition, plaque-reduction neutralisation and immunofluorescence tests (OIE 2008a; OIE 2008b).

Vaccines for EEE and WEE are formalin-inactivated and are safe and effective (OIE 2008a). However, VEE vaccines must be attenuated, since formalin-inactivated virulent VEE vaccines can cause severe illness in horses and result in epidemics of VEE (OIE 2008b).

Conclusion

EEE and WEE are present in some approved countries. The Code recommendations (OIE 2009a) include premises freedom or vaccination. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

VEE (epidemic form) is not present any approved country. While this remains the case, certification of county freedom, in accordance with the Code recommendations (OIE 2009c), will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

CFSPH (2008) Eastern equine encephalomyelitis, western equine encephalomyelitis and Venezuelan equine encephalomyelitis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/Factsheets/pdfs/easter_wester_venezuelan_equine_encephalomyelitis.pdf (Accessed 8 December 2008).

Geering WA, Forman AJ, Nunn MJ (1995) Equine viral encephalomyelitis. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 106-111. Australian Government Publishing Service, Canberra.

Griffin DE (2007) Alphaviruses. In *Fields virology* (eds. Knipe DM, Howley PM) pp. 1023-1057. Lippincott Williams & Wilkins, Philadelphia.

Justines G, Sucre H, Alvarez O (1980) Transplacental transmission of Venezuelan equine encephalitis virus in horses. *The American Journal of Tropical Medicine and Hygiene* 29: 653-656.

OIE (2008a) Equine encephalomyelitis (Eastern and Western). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.05_EQUINE_ENCEPH.pdf (Accessed 1 September 2009).

OIE (2008b) Venezuelan equine encephalomyelitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.14_VEE.pdf (Accessed 3 September 2009).

OIE (2009a) Equine encephalomyelitis (Eastern and Western). Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.5.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2009c) Venezuelan equine encephalomyelitis. Terrestrial Animal Health Code 2008. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.14.htm (Accessed 2 September 2009).

Powers AM, Oberste MS, Brault AC, Rico-Hesse R, Schmura SM, Smith JF, Kang W, Sweeney WP, Weaver SC (1997) Repeated emergence of epidemic/epizootic Venezuelan equine encephalitis from a single genotype of enzootic subtype ID virus. *The Journal of Virology* 71: 6697-6705.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

Weaver SC, Ferro C, Barrera R, Boshell J, Navarro JC (2004) Venezuelan equine encephalitis. *Annual Review of Entomology* 49: 141-174.

Weaver SC, Frey TK, Huang HV, Kinney RM, Rice CM, Roehrig JT, Shope RE, Strauss EG (2005) Togaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 999-1008. Elsevier, San Diego.

Zacks MA, Paessler S (2009) Encephalitic alphaviruses. *Veterinary Microbiology* doi:10.1016/j.vetmic.2009.08.023: In press.

5.20 Fascioliasis

5.20.1 Technical information

Background

Fascioliasis (liver fluke disease) is caused by trematode parasites of the genus *Fasciola* in the family Fasciolidae. The disease occurs in all domestic animals including horses, and many species of wildlife. It has also been reported in humans (Endo and Morishima 2004). Its economic importance is restricted to cattle and sheep. Other species can act as reservoirs of infection. Species of *Fasciola* affecting horses include *F. hepatica* and *F. gigantica*.

F. hepatica occurs in cooler climates and has a worldwide distribution (Radostits et al. 2007). It is endemic in all Australian States apart from Western Australia, where movement controls are in place for the movement of both imported and domestic horses.

F. gigantica is restricted to warmer regions in Africa, China, southern Europe, India, Japan, the Middle East, some Pacific Islands, Pakistan, South East Asia and the United States (Urquhart et al. 1996; Kassai 1999). It has not been reported in Australia.

Fascioliasis is not an OIE-listed disease (OIE 2009).

Epidemiology

Snails of the genus *Lymnaea* are the intermediate hosts. These snails are mainly aquatic and are found in streams, irrigation channels and marshy swamps. They release infective cercaria onto surrounding vegetation — consequently, fascioliasis is associated with animals grazing in flooded areas or around permanent water channels or dams (Urquhart et al. 1996).

Fascioliasis occurs seasonally during warm, wet months, with numbers of infected snails determining the risk of disease. Numbers of snails as well as the rate of development of fluke eggs and larvae increase when temperature and rainfall are high (Radostits et al. 2007).

Adult liver flukes are located in bile ducts where they produce eggs, which are excreted in faeces. The eggs hatch in moist conditions and the miracidia invade the tissues of suitable species of snails. After asexual multiplication in snails, cercariae leave the snails to attach to herbage, from where they are ingested by grazing livestock. In the intestine, immature flukes emerge and migrate through the peritoneal cavity to the liver (Radostits et al. 2007).

Although the epidemiology, clinical signs, clinical pathology, diagnosis and control of the two *Fasciola* species are reported to be similar in ruminants, information on the epidemiology of *F. gigantica* in horses is scarce.

Clinical signs

The prepatent period is 6–12 weeks for *F. hepatica* and 12 weeks or greater for *F. gigantica* (Kassai 1999).

Adverse effects on the host arise from mechanical injury due to immature flukes migrating through the liver parenchyma, irritation of the bile duct epithelial lining by mature flukes, toxic effects from the excretory and secretory products of flukes and haemorrhages in the liver due to haematophagous feeding habits of flukes (Kassai 1999).

In a study in South Africa, ten horses were experimentally infected with *F. hepatica* and *F. gigantica* — neither fluke eggs nor clinical signs of disease were observed in the horses (Alves et al. 1988). Macroscopic lesions similar to those caused by *F. hepatica* were observed in two horses infected with this species. No lesions due to *F. gigantica* were observed in the liver or other organs.

Diagnosis

Diagnosis is based on a combination of clinical signs, seasonal occurrence, weather patterns, history of flukes in endemic areas, examination of faeces for fluke eggs and post-mortem findings.

Treatment

Triclabendazole at 12 mg/kg has been reported to treat flukes in equids (Holtmann et al. 1990; Trawford and Tremlett 1996).

Conclusion

F. hepatica is present in approved countries and in Australia. *F. gigantica* is present in some approved countries and there are no recommendations in the Code. A comprehensive literature review on *F. gigantica* infections in horses resulted in limited information, indicating that horses are rarely infected.

Fascioliasis was not considered further in the IRA.

References

Alves RMR, van Rensburg LJ, van Wyk JA (1988) *Fasciola* in horses in the Republic of South Africa: a single natural case of *Fasciola hepatica* and the failure to infest ten horses either with *F. hepatica* or *Fasciola gigantica*. *Onderstepoort Journal of Veterinary Research* 55: 157-163.

Endo T, Morishima Y (2004) Major helminth zoonoses in water. In *Waterborne zoonoses: identification, causes and control* (eds. Cotruvo JA, Dufour A, Rees G, Bartram J, Carr R, Cliver DO, Craun GF, Fayer R, Gannon VPJ) pp. 291-304. IWA Publishing on behalf of WHO, London.

Holtmann W, Ising S, Stoye M (1990) Effect of triclabendazole on *Fasciola hepatica* in experimentally infected horses. *Tieraerztliche Umschau* 45: 434-438.

Kassai T (1999) Phylum: Platyhelminthes flatworms. In *Veterinary helminthology* pp. 3-50. Butterworth-Heinemann, Oxford.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with helminth parasites. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1541-1583. Saunders Elsevier, Edinburgh.

Trawford AF, Tremlett JG (1996) Efficacy of triclabendazole against *Fasciola hepatica* in the donkey (*Equus asinus*). *The Veterinary Record* 139: 142-143.

Urquhart GM, Armour J, Duncan JL, Dunn AM, Jennings FW (1996) Veterinary helminthology. In *Veterinary parasitology* pp. 3-138. Blackwell Science, Oxford.

5.21 Glanders

5.21.1 Technical information

Background

Glanders is a highly contagious bacterial disease characterised by nodular lesions on the respiratory tract, skin and lymph nodes (Dvorak and Spickler 2008). It mainly affects equids and is also zoonotic. Glanders is also considered by the United States as a potential bioterrorist threat and is classified as a Category B bioterrorism agent by the Centers for Disease Control and Prevention (CDC 2008).

The causative agent of glanders is *Burkholderia mallei* (formerly known as *Pseudomonas mallei*), which is closely related to, and may have evolved from, *B. pseudomallei*, the causative agent of melioidosis (CFSPH 2007).

Horses, mules and donkeys are the major hosts of glanders and can transmit the disease to other animals and humans. Although rare, natural infections have occurred in cats, dogs, camels, goats and sheep. Guinea pigs and hamsters are also highly susceptible. Most mammals can be experimentally infected, although cattle, pigs, rats and birds are considered to be relatively resistant (Dvorak and Spickler 2008).

The only known occurrence of glanders in Australia was in 1891 and confined to imported circus horses held in quarantine in Sydney (Geering et al. 1995). Once prevalent virtually worldwide, glanders has been eradicated from many countries, including Canada, western Europe and the United States (Dvorak and Spickler 2008). However, the disease persists in some Asian, African and South American countries (OIE 2008a). Between 1996 and 2007, the OIE received reports of cases of glanders in Belarus, Bolivia, Brazil, Eritrea, Ethiopia, India, Iran, Iraq, Mongolia, Philippines, Russia and Turkey (OIE 2008b; OIE 2008c).

An incident of glanders in the United Arab Emirates in a post-import isolation premises was detected in April 2004 (OIE 2004). The initial cases were seen in three of ten horses imported by road from another Middle-Eastern country. The disease was also detected in June 2004 in local horses sharing the same post-import isolation premises. No horses left the premises following entry of the imported horses. Glanders was contained within the post-import isolation premises and did not enter the general equine population.

There was a case of glanders in Germany in 2006 in a horse imported from Brazil. Despite having certification of testing negative for glanders on compliment fixation test (CFT) before export, two weeks after arrival the horse developed respiratory illness that failed to improve with treatment. Glanders was subsequently diagnosed (by CFT and mallein skin test) and the horse was euthanased. The horse had been kept isolated from other horses (Elschner et al. 2009).

There have been no other recent reports of glanders in approved countries.

Glanders is an OIE-listed disease (OIE 2009b).

Epidemiology

Among equids, glanders is highly contagious, particularly in crowded, stressful or unclean conditions (Dvorak and Spickler 2008). Acute disease occurs mainly in donkeys and mules; disease generally follows a more chronic course in horses (OIE 2008a).

Transmission occurs mainly through contact with skin exudates and respiratory secretions, which may contain large numbers of organisms (CFSPH 2007). These can be spread easily on fomites (such as food, water and equipment) and in the environment. Transmission in equids most commonly occurs through ingestion of the organism, respiratory exposure or by entry through skin abrasions or mucous membranes (Dvorak and Spickler 2008).

Large cats and other carnivores have developed *B. mallei* infection from eating infected horse meat (Dvorak and Spickler 2008). Small ruminants may be infected if in close contact with infected horses (OIE 2008a). Humans can contract disease through direct contact with diseased animals, or infected or contaminated material (OIE 2008a).

Diagnosis may be missed in some chronically or latently infected animals with minimal signs of disease, and these animals may serve as a pathogen reservoir and are often responsible for the maintenance and spread of disease (Dvorak and Spickler 2008). Chronic and subclinically infected cases can shed bacteria permanently or intermittently (OIE 2008a).

Although the organism is inactivated by heat and sunlight, survival may be prolonged in wet or humid conditions. Survival may vary from a few months to more than a year in favourable environments. In unfavourable conditions, the organism is likely to be inactivated within two weeks (CFSPH 2007).

Clinical signs

In animals, the incubation period is typically 2–6 weeks but can vary from a few days to many months in natural infection (CFSPH 2007). Horses, mules and donkeys can develop acute, chronic or latent forms of glanders.

In the acute form, clinical signs include marked pyrexia, coughing, inspiratory dyspnoea, thick nasal discharge and deep, rapidly spreading ulcers on nasal mucosa. Submaxillary lymph nodes may be swollen and painful and facial lymphatic vessels thickened. Secondary skin infections may occur. Death usually occurs within 1–2 weeks of the onset of clinical signs (Geering et al. 1995).

Chronic disease is insidious in onset and signs may include coughing, malaise, unthriftiness, intermittent pyrexia and a chronic purulent nasal discharge (usually unilateral). Other signs may include ulcers (healed ulcers form star-shaped white scars) and nodules on the nasal mucosa; cutaneous lesions (called farcy; most numerous on the legs) that rupture and ulcerate; chronic enlargement and induration of the lymphatics and lymph nodes; painful oedema of the legs and swollen joints (Geering et al. 1995). Chronic infection can develop over weeks to months and may be subclinical initially (Dvorak and Spickler 2008). It is slowly progressive and often fatal, although animals may survive for years (CFSPH 2007).

Few signs may be seen in the latent form of glanders and lesions occur only in the lungs. Animals may show nasal discharge and occasional laboured breathing (Geering et al. 1995). Animals with latent infection may be sick for a number of months then appear to recover, while infection actually persists for years (Dvorak and Spickler 2008).

Diagnosis

Isolation and identification of *B. mallei* in cultures of samples from lesions or exudates is considered the gold standard for diagnosis of glanders (Dvorak and Spickler 2008). The mallein test is sensitive and specific for hypersensitivity against *B. mallei* but may give inconclusive results. Various serological tests are available, but the CFT and enzyme-linked immunosorbent assay are the more accurate and reliable for diagnostic use (OIE 2008a).

There are no vaccines available.

Conclusion

Glanders is not present in any approved country. While this remains the case certification of country freedom, in accordance with the Code recommendations (OIE 2009a), will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

CDC (2008) Emergency preparedness and response: bioterrorism agents/diseases. Centers for Disease Control and Prevention. http://emergency.cdc.gov/agent/agentlist-category.asp (Accessed 1 December 2008).

CFSPH (2007) Glanders. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/Factsheets/pdfs/Glanders.pdf (Accessed 23 October 2008).

Dvorak GD, Spickler AR (2008) Glanders. *Journal of the American Veterinary Medical Association* 233: 570-577.

Elschner MC, Klaus CU, Liebler-Tenorio E, Schmoock G, Wohlsein P, Tinschmann O, Lange E, Kaden V, Klopfleisch R, Melzer F, Rassback A, Newbauer H (2009) *Burkholderia mallei* infection in a horse imported from Brazil. *Equine Veterinary Education* 21: 147-150.

Geering WA, Forman AJ, Nunn MJ (1995) Glanders. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 324-329. Australian Government Publishing Service, Canberra.

OIE (2004) Glanders in the United Arab Emirates in post-import isolation premises. Disease Information. ftp://ftp.oie.int/infos_san_archives/eng/2004/en_041015v17n42.pdf (Accessed 1 September 2009).

OIE (2008a) Glanders. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.11_GLANDERS.pdf (Accessed 1 September 2009).

OIE (2008b) Handistatus II: prototype. World Organisation for Animal Health (OIE). http://www.oie.int/hs2/report.asp (Accessed 17 November 2008).

OIE (2008c) WAHID interface. World Animal Health Information Database: version 1.2. http://www.oie.int/wahid-prod/public.php?page=home (Accessed 14 November 2008).

OIE (2009a) Glanders. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.11.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

5.22 Horse pox

5.22.1 Technical information

Background

Historically, horse pox was recorded throughout Europe. However, no cases have been reported since the early 20th century (Fenner 1996). There are no reports of the disease occurring elsewhere.

The causative agent of horse pox is the same as that of vaccinia in cattle. Experimentally, vaccinia virus and orf virus can also produce lesions in horses (Bruner and Gillespie 1966). Uasin Gishu disease, a poxviral infection of wildlife in Africa, has occasionally been transferred to horses (Fenner 1996).

Horse pox is not an OIE-listed disease (OIE 2009b); however, the Code includes recommendations for the importation of horses with respect to horse pox (OIE 2009a).

Epidemiology

Horse pox is highly contagious and transmission is thought to be by direct contact and fomites. There is no vaccine and only topical, astringent treatment is available.

Clinical signs

Horse pox lesions typically develop, with or without mild pyrexia, in either a leg or a buccal form, also known as contagious pustular stomatitis. The leg form commonly affects the pastern and fetlock, causing pain and lameness. Lesions begin as nodules resembling papillomas and progress to vesicles, pustules and scabs. In the buccal form, similar lesions first appear inside the lips and spread over the entire mucosa, causing stomatitis, salivation and anorexia. Lesions may extend to the pharynx, larynx, nostrils, conjunctiva, vulva and, in severe cases, can become generalised over the whole body. Both leg and buccal forms usually resolve within 2–4 weeks and produce good immunity (Radostits et al. 2007).

Diagnosis

Diagnosis can be confirmed by electron microscopic examination of scabs or lesion scrapings. The virus can also be grown in cell culture and identified as poxvirus by its cross-reactivity with vaccinia virus (Fenner 1996).

Conclusion

Horse pox has not been reported in any approved country since the early 1900s. While this remains the case, certification of premises freedom, in accordance with the Code recommendations (OIE 2009a) will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Bruner DW, Gillespie JH (1966) Virus diseases characterized by lesions of the skin. In *Hagan's infectious diseases of domestic animals with special reference to etiology, diagnosis, and biologic therapy* (eds. Bruner DW, Gillespie JH) pp. 791-841. Bailliere, Tindall & Cassell, London.

Fenner F (1996) Poxvirus infections. In *Virus infections of equines* (ed. Studdert MJ) pp. 5-9. Elsevier Science Publishers, Amsterdam.

OIE (2009a) Horse pox. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.13.htm (Accessed 1 September 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

5.23 Japanese encephalitis

5.23.1 Technical information

Background

Japanese encephalitis (JE) is a mosquito-borne viral disease that causes encephalitis in horses and humans, and abortion and stillbirths in sows. Pigs and ardeids (bitterns, egrets and herons) are the main amplifier hosts. Other mammalian species can become infected but are not considered significant in the epidemiology of the disease (Brown 2008).

JE virus is the prototype of the JE serogroup of flaviviruses, which includes Murray Valley encephalitis virus, St Louis encephalitis virus, and West Nile virus (Thiel et al. 2005). There are four to five genotypes of JE virus, all belonging to a single serotype and having similar virulence (Gubler et al. 2007).

JE virus is widely distributed in Asia (including Japan) and Papua New Guinea (van den Hurk et al. 2002). It was last reported in 2000 in Hong Kong and in 1998 in Singapore (OIE 2009a; OIE 2009c). It has not been reported in the Americas or Europe (CDC 2008). Testing conducted in 1995 detected serologically positive dogs, horses and pigs on several islands in the Torres Strait (Geering et al. 1995). JE virus was first detected on mainland Australia in 2005, with subsequent isolation of the virus from *Culex* mosquitoes (van den Hurk et al. 2006). There have been no reports of clinical cases of JE in animals in Australia and JE has not become established on mainland Australia.

JE is an OIE-listed disease (OIE 2009d).

Epidemiology

Mosquitoes belonging to the genera *Culex* and *Aedes* transmit the virus. Other genera have also been shown to harbour the virus but their role in transmission remains unconfirmed (CFSPH 2007). Vertical transmission occurs in mosquitoes (Brown 2008).

Disease occurs in late summer and early autumn in temperate and subtropical areas when infection builds up in birds, pigs, and then mosquitoes which feed on these amplifier hosts. In the tropics, disease circulates constantly but case numbers can spike during the wet season when mosquito numbers increase (Brown 2008).

Horses and humans are susceptible to infection and can become clinically ill after being bitten by an infected mosquito. Infection can cause severe illness, and have adverse implications for the national and international movement of horses (Lam et al. 2005). However, infection in horses leads to only a low grade viraemia that is insufficient to infect mosquitoes (Gould et al. 1964).

Rates of natural infection in unvaccinated horses in Japan, where JE is endemic, range from 15 to 67% (Konishi et al. 2004). Increased vaccination of both humans and horses, in combination with improved mosquito control, has resulted in a decrease in prevalence of JE cases in Japan (Brown 2008). However, cases of JE are increasing in other parts of the world, such as India and Pakistan (Mackenzie et al. 2002).

Clinical Signs

The incubation period in horses is generally 8–10 days (CFSPH 2007). Most infected horses do not show clinical signs of the disease. In those that do, three forms have been described: transient, lethargic and hyperexcitable forms. The transient form is characterised by pyrexia lasting up to three days, accompanied by anorexia, stupor, impaired locomotion, and congested or icteric mucous membranes, followed by recovery (Geering et al. 1995). In the lethargic form, neurological signs accompany fluctuating pyrexia and recovery occurs within a week. The hyperexcitable form is the most severe and is characterised by marked pyrexia, aimless wandering, violent and demented behaviour, blindness, profuse sweating, muscle tremors, collapse and death (Brown 2008). A case of the hyperexcitable form in Hong Kong in a gelding that had been vaccinated against JE has also been described (Lam et al. 2005).

Diagnosis

Diagnosis can be based on clinical signs and confirmed by laboratory testing. The definitive diagnosis of JE in horses depends on isolation of virus from sick or dead horses. This can be difficult due to viral instability (Lian et al. 2002). Similarly, presence of antibodies to other flaviriruses causes serological cross reactivity and false positive results (OIE 2008).

Conclusion

JE is present in some approved countries. The Code recommendations (OIE 2009b) include vaccination. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Brown C (2008) Japanese encephalitis. In *Foreign animal diseases* (ed. Committee on Foreign and Emerging Diseases of the United States Animal Health Association) pp. 311-315. United States Animal Health Association, St. Joseph.

CDC (2008) Prevention of specific infectious diseases: Japanese encephalitis. CDC Health Information for International Travel 2008. http://wwwn.cdc.gov/travel/yellowBookCh4-JapaneseEncephalitis.aspx (Accessed 6 November 2008).

CFSPH (2007) Japanese encephalitis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 2 December 2008).

Geering WA, Forman AJ, Nunn MJ (1995) Japanese encephalitis. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 140-144. Australian Government Publishing Service, Canberra.

Gould DJ, Byrne RJ, Hayes DE (1964) Experimental infection of horses with Japanese encephalitis virus by mosquito bite. *The American Journal of Tropical Medicine and Hygiene* 13: 724-746.

Gubler DJ, Kuno G, Markoff L (2007) Flaviviruses. In *Fields virology* (eds. Knipe DM, Howley PM) pp. 1153-1252. Lippincott Williams & Wilkins, Philadelphia.

Konishi E, Shoda M, Kondo T (2004) Prevalence of antibody to Japanese encephalitis virus nonstructural 1 protein among racehorses in Japan: indication of natural infection and need for continuous vaccination. *Vaccine* 22: 1097-1103.

Lam KHK, Ellis TM, Williams DT, Lunt RA, Daniels PW, Watkins KL, Riggs CM (2005) Japanese encephalitis in a racing thoroughbred gelding in Hong Kong. *The Veterinary Record* 157: 168-173.

Lian WC, Liau MY, Mao CL (2002) Diagnosis and genetic analysis of Japanese encephalitis virus infected in horses. *Journal of Veterinary Medicine, Series B* 49: 361-365.

Mackenzie JS, Johansen CA, Ritchie SA, van den Hurk AF, Hall RA (2002) Japanese encephalitis as an emerging virus: the emergence and spread of Japanese encephalitis virus in Australasia. In *Japanese encephalitis and West Nile viruses* (eds. Mackenzie JS, Barrett ADT, Deubel V) pp. 49-73. Springer-Verlag, Wien, Berlin.

OIE (2008) Japanese encephalitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.07_JEV.pdf (Accessed 1 September 2009).

OIE (2009a) Detailed country (ies) disease incidence: Asia, Japanese encephalitis, 2005-2008. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php (Accessed 7 September 2009).

OIE (2009b) Japanese encephalitis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.7.htm (Accessed 1 September 2009).

OIE (2009c) Japanese encephalitis: multiannual animal disease status (disease occurrence). Handistatus II: prototype. http://www.oie.int/hs2/sit_mald_freq_pl.asp?c_cont=3&c_mald=70 (Accessed 8 September 2009).

OIE (2009d) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Thiel HJ, Collett MS, Gould EA, Heinz FX, Houghton M, Meyer G, Purcell RH, Rice CM (2005) Flaviviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 981-998. Elsevier, San Diego.

van den Hurk AF, Montgomery BL, Northill JA, Smith IL, Zborowski P, Ritchie SA, Mackenzie JS, Smith GA (2006) Short report: the first isolation of Japanese encephalitis virus from mosquitos collected from mainland Australia. *The American Journal of Tropical Medicine and Hygiene* 75: 21-25.

van den Hurk AF, Nisbet DJ, Foley PN, Ritchie SA, Mackenzie JS, Beebe NW (2002) Isolation of arboviruses from mosquitoes (Diptera: Culicidae) collected from the Gulf Plains region of northwest Queensland, Australia. *Journal of Medical Entomology* 39: 786-792.

5.24 Leptospirosis

5.24.1 Technical information

Background

Leptospirosis affects mammals worldwide and has been reported in birds, reptiles, amphibians and arthropods. Horses appear to be incidental hosts of seven leptospiral serovars, and suspected maintenance hosts of serovar Bratislava (Divers et al. 1992; Ellis 1999).

The genus *Leptospira* can be classified into serovars or species, with the former method most commonly used. The serovar is the basic systematic unit distinguishing leptospires on antigenic similarities and differences using the cross agglutination absorption test. There are more than 200 pathogenic serovars divided into 25 serogroups (WHO 2003). In Australia, serovars isolated from horses include Pomona and Tarassovi (Biosecurity Australia 2000). In addition, serological surveys suggest that horses have been exposed to serovars Pomona, Icterohaemorrhagicae, Tarassovi, Hardjo, Canicola, Grippotyphosa, Bratislava and Australis (Slatter and Hawkins 1982; Hogg 1983; Biosecurity Australia 2000). However, horses develop high titres of anti-leptospiral antibodies and this can cause considerable cross-reaction with other serovars and confound interpretation of serosurveys (Biosecurity Australia 2000). Leptospires can also be classified into more than 16 species through DNA analysis; eight of these being pathogenic (le Febvre 2004).

Leptospirosis is a multiple species OIE-listed disease (OIE 2009). There is a chapter in the OIE Manual relating to the disease (OIE 2008) but there are no recommendations in the Code for the importation of animals with respect to leptospirosis.

Epidemiology

Horses can be infected by direct or indirect transmission. Direct transmission occurs when blood or body fluids (urine, semen, milk) pass from an infected or carrier animal to a susceptible animal. Indirect transmission results from environmental exposure to leptospires from an infected or carrier animal.

Leptospires are thought to enter the body via breaches in the integument, through some mucous membranes (conjunctiva, genital tract), and possibly the nose and lungs following inhalation. Leptospires damage the walls of small blood vessels, leading to vessel leakage and haemorrhage. They attach to cells in renal tubules providing a mechanism for the carrier state and long term spread of infection (Faine et al. 1999).

Humans are most likely to be infected by urine from carrier animals or contaminated surface waters, mud and soil (Faine 1998). In 2007, 74 human cases of leptospirosis were reported in Australia, with most attributed to environmental contamination from rat or marsupial urine (Burns 2007).

Sporadic cases of leptospirosis occur in horses in Australia. The disease is not nationally notifiable. The prevalence of leptospirosis is difficult to determine due to lack of reporting and difficulties culturing the organism. Seroprevalence studies have

shown 30% of eastern Australian horses have evidence of exposure (Slatter and Hawkins 1982; Dickeson and Love 1993).

Investigations in the United States implicated leptospirosis in 3% of abortions in mares (Szeredi and Haake 2006).

Vaccination is not practical in horses as they are susceptible to multiple serovars and cross-immunity between serovars does not occur (Faine et al. 1999).

Clinical signs

The incubation period in animals is 2–20 days. Most leptospiral infections in horses are subclinical. Clinical signs of disease usually last 5–18 days, and include mild pyrexia with anorexia — and in more severe forms — haemoglobinuria, icterus, mucosal petechiae and conjunctival oedema (Faine et al. 1999). Pregnant mares may abort sporadically, usually in the last trimester, with leptospires evident in the mare's urine and in foetal and placental tissue. Pomona is the most common serovar associated with abortion, although Australis, Grippotyphosa, Icterohaemorrhagiae and Sejroe have been isolated (Donahue and Williams 2000). Foals born live after *in utero* infection can die.

In Australia, a group of pregnant mares gave birth to premature and weak foals displaying a range of clinical signs including hyphaema. Foals apparently healthy at birth became ill and over half subsequently died. Pomona was isolated and leptospires found in renal lesions (Hogg 1983). Recurrent iridocyclitis or uveitis can occur some months after infection (Faine 1998; Hartskeerl et al. 2004).

Diagnosis

Clinical signs of leptospirosis are non-specific, and diagnosis can be confirmed by demonstration of antibody, demonstration of *Leptospira* nucleic acid or culture of leptospires from blood, urine, cerebrospinal fluid or body tissues.

Biosecurity Australia conducted a scientific review of leptospirosis in all import-eligible species in 2000 (Biosecurity Australia 2000). This review recommended that Australia did not require quarantine measures for leptospirosis in horses. The requirement for contagious equine metritis, that imported pregnant mares must be kept under quarantine surveillance until the delivery of a healthy foal, is likely to identify any abortions due to leptospirosis.

Conclusion

Leptospirosis is present in approved countries and in Australia. The disease in horses is not subject to official control in Australia.

Leptospirosis was not considered further in the IRA.

References

Biosecurity Australia (2000) *A scientific review of leptospirosis and implications for quarantine policy*. Agriculture Fisheries and Forestry - Australia, Canberra.

Burns M-A (2007) *National leptospirosis surveillance report number 16: January-December 2007*. No. 16, Queensland Health Forensic and Scientific Services, Queensland.

Dickeson D, Love DN (1993) A serological survey of dogs, cats and horses in south-eastern Australia for leptospiral antibodies. *Australian Veterinary Journal* 70: 389-390.

Divers TJ, Byars TD, Shin SJ (1992) Renal dysfunction associated with infection of *Leptospira interrogans* in a horse. *Journal of the American Veterinary Medical Association* 201: 1391-1392.

Donahue JM, Williams NM (2000) Emergent causes of placentitis and abortion. *Veterinary Clinics of North America: Equine Practice* 16: 443-456. (Abstract only)

Ellis WA (1999) Equine leptospirosis. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 155-158, R & W Publications (Newmarket) Limited, Newmarket.

Faine S (1998) *Leptospira*. In *Topley & Wilson's microbiology and microbial infections* (eds. Collier L, Balows A, Sussman M) pp. 1287-1303. Oxford University Press, New York.

Faine S, Adler B, Bolin C, Perolat P (1999) *Leptospira and Leptospirosis*. MediSci Writing, Melbourne.

Hartskeerl RA, Goris MGA, Brem S, Meyer P, Kopp H, Gerhards H, Wollanke B (2004) Classification of *Leptospira* from the eyes of horses suffering from recurrent uveitis. *Journal of Veterinary Medicine, Series B* 51: 110-115.

Hogg GG (1983) Equine leptospirosis. In *Refresher course for veterinarians proceedings No.65, 8-12 August 1983, The University of Sydney: equine practice - diagnosis & therapy,* pp. 221-240, The University of Sydney, The Post-Graduate Foundation in Veterinary Science, Sydney.

le Febvre RB (2004) Spiral-curved organisms V: *Leptospira*. In *Veterinary microbiology* (eds. Hirsh DC, MacLachlan NJ, Walker R) pp. 148-152. Blackwell Publishing, Oxford.

OIE (2008) Leptospirosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.09_LEPTO.pdf (Accessed 1 September 2009).

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Slatter DH, Hawkins CD (1982) Prevalence of leptospiral titres in normal horses. *Australian Veterinary Journal* 59: 84-86.

Szeredi L, Haake DA (2006) Immunohistochemical identification and pathologic findings in natural cases of equine abortion caused by leptospiral infection. *Veterinary Pathology* 43: 755-761.

WHO (2003) *Human leptospirosis: guidance for diagnosis, surveillance and control*. World Health Organization, Switzerland.

5.25 Louping ill

5.25.1 Technical information

Background

Louping ill is a tick-borne viral encephalitis caused by a virus belonging to the genus Flavivirus in the family Flaviviridae (Thiel et al. 2005). It is primarily a disease of sheep, although other species such as grouse, dogs, horses, cattle, goats, pigs, deer and llamas can be affected (Brown 2008). Humans can also be affected although this is rare (Radostits et al. 2007).

The disease has been reported in Europe in areas where the tick vector *Ixodes ricinus* is distributed (Radostits et al. 2007).

Louping ill is not an OIE-listed disease (OIE 2009).

Epidemiology

Ixodes ricinus ticks are the main reservoir and natural vectors of louping ill. The tick requires at least three years to complete its life cycle, feeding for three weeks once a year at each developmental stage. Adult female ticks need larger vertebrate hosts to engorge and mate on; larvae and nymphs are able to feed on any vertebrate hosts (Radostits et al. 2007). Other ixodid tick species capable of transmitting the virus include *I. persulcatus*, *Haemaphysalis anatolicum,* and *Rhipicephalus appendiculatus* (CFSPH 2005). There are no known tick vector species in Australia. In the absence of a competent vector, it is unlikely that the louping ill virus could become established (Geering et al. 1995).

Incidence of disease follows seasonal tick activity and peaks during spring. Transmission within tick vectors can occur trans-stadially but not transovarially. Infected ticks can transmit the virus to a large number of hosts, but only sheep, grouse and possibly horses attain a viraemia sufficient to infect other ticks and act as maintenance hosts for the virus (Radostits et al. 2007). There are no reports of transmission, other than by tick vectors, in horses.

Clinical signs

Clinical signs of disease in horses are similar to those seen in sheep but most infections in horses are subclinical (Radostits et al. 2007). An incubation period of 2–4 days is followed by a sudden onset of pyrexia. Horses then either recover, suffer from progressive neurological disease or show a transient disorder in locomotion followed by recovery in 10–12 days (Radostits et al. 2007).

Diagnosis

Diagnosis is by isolation of virus, detection of viral antigens and by serology (haemagglutination-inhibition, serum neutralisation, and enzyme-linked immunosorbent assay) (CFSPH 2005).

Conclusion

Louping ill is present in some approved countries. The tick vector is not present in Australia.

Louping ill was not considered further in the IRA.

References

Brown C (2008) Louping-ill. In *Foreign animal diseases* (ed. Committee on Foreign and Emerging Diseases of the United States Animal Health Association) pp. 321-324. United States Animal Health Association, St. Joseph.

CFSPH (2005) Louping ill. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 17 February 2009).

Geering WA, Forman AJ, Nunn MJ (1995) Louping ill. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 151-154. Australian Government Publishing Service, Canberra.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

Thiel HJ, Collett MS, Gould EA, Heinz FX, Houghton M, Meyer G, Purcell RH, Rice CM (2005) Flaviviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 981-998. Elsevier, San Diego.

5.26 Lyme disease

5.26.1 Technical information

Background

Lyme disease is caused by the tick-borne spirochaete *Borrelia burgdorferi* senso lato (s.l.) (Burgdorfer et al. 1982). Within the species *B. burgdorferi* s.l., there are several genospecies, including *B. burgdorferi* senso stricto, *B. afzelli, B. garinii, B. lusitaniae, B. valaisiana* and *B. japonica* (Burgdorfer 1991; Kawabata et al. 1993; Ryffel et al. 1999). Pathogenicity, geographical distribution, and host and vector preferences vary between genospecies (Wang et al. 1999). Infection, with or without clinical signs of disease, has been reported in cats (Magnarelli et al. 1990), dogs (Madigan and Teitler 1988), cattle (Burgess et al. 1993), sheep (Fridriksdottir et al. 1992) horses (Divers 2007) and humans (Parker and White 1992). Lyme disease is the most commonly reported tick-borne disease in humans in Asia, Europe and the United States (Steere et al. 2004).

Serosurveillance in humans and animals, and attempts at isolation from possible tick vectors, have failed to reveal conclusive evidence of Lyme disease in Australia (Russell 1995; Doggett et al. 1997). *B. garinii* was isolated from a human patient in New South Wales in 1996, although it is possible the infection was acquired overseas (Hudson et al. 1998). A tentative diagnosis in a cow was reported in New South Wales but the causal organism was not isolated (Rothwell et al. 1989), and serological surveys of dogs in south-eastern Queensland did not find evidence of *B. burgdorferi* s.l. (Baldock et al. 1993).

Lyme disease is not an OIE-listed disease (OIE 2009).

Epidemiology

B. burgdorferi s.l. is maintained in a two year cycle between ixodid ticks and small mammals (Burgdorfer et al. 1982; Anda et al. 1996). *B. burgdorferi* s.l. has been found in other blood-sucking arthropods including chigger mites, body lice, horse flies and mosquitoes, but effective transmission from these vectors has not been proven (Magnarelli et al. 1986; Roux and Raoult 1999). Ticks become infected when taking a blood meal from an infected host and, on subsequent feedings, transmit infection through saliva to a new host. Transmission of *B. burgdorferi* s.l. is unlikely to occur until the tick has been attached for at least 24 hours (Berger et al. 1995).

Horses serve as hosts for adult and nymphal stages of *Ixodes* spp. ticks (Bushmich 1994); however, there is evidence that *Ixodes holocyclus* nymphs infected with *B. burgdorferi* s.l. do not retain infection after moulting (Piesman and Stone 1991). There is no information on whether larval *I. holocyclus* can be infected and whether they retain infection after moulting.

In endemic areas, prevalence of *B. burgdorferi* s.l. infection in vector-competent ticks varies geographically and is a good predictor of Lyme disease incidence. Prevalence in adult ticks averages 50%, in part because an adult has two chances of acquiring an infectious blood meal, having fed as both a larva and a nymph (Barbour and Fish 1993). Vector ticks harbour and transmit *B .burgdorferi* s.l. to humans and animals.

Other ticks, which do not bite humans, may play a key role in maintaining enzootic life cycles of *B. burgdorferi* s.l. in nature as vectors to reservoir hosts (Xu et al. 2003). Rodents and white-tailed deer are reservoir hosts in endemic countries (Anderson et al. 1985). Birds, rabbits and sheep may be involved in maintaining infectious cycles and introducing the disease to a wider geographical area (Lane and Regnery 1989; Telford, III and Spielman 1989; Olsen et al. 1993; Olsen et al. 1995; Ogden et al. 1997).

The life cycle of the spirochaete depends on horizontal transmission from infected tick nymphs to small mammals in early summer, and from infected small mammals to tick larvae in late summer (Parker and White 1992). Trans-stadial transmission occurs within ticks and, in some tick species, transovarial transmission also occurs (Lane and Burgdorfer 1987; Magnarelli et al. 1987). Multiple infections of vector ticks with other *Borrelia* spp. and *Anaplasma phagocytophilum* have been reported (Guttman et al. 1996; Leutenegger et al. 1999).

There are 25 species of *Ixodes* tick in Australia, none of which has been examined for competence in transmitting *B. burgdorferi* s.l. However, dissection and polymerase chain reaction testing of over 11 000 ixodid ticks from coastal New South Wales failed to demonstrate evidence of *B. burgdorferi* s.l. (Russell et al. 1994).

B. burgdorferi s.l. can be transmitted without an arthropod vector. Nonvector transmission has been demonstrated experimentally in dogs and mice (Burgess et al. 1986) via oral, intramuscular and subcutaneous routes. In horses, the organism has been isolated from blood, urine and synovial fluid (Burgess 1988; Madigan 1993; Manion et al. 1998), and transplacental transmission has been reported (Burgess et al. 1988; Burgess 1988). Iatrogenic transmission is possible (Parker and White 1992), and there is potential for zoonotic spread (Marcelis et al. 1987; Manion et al. 1998).

Seroprevalence of *B. burgdorferi* s.l. in horses in Austria is 33% (Kitrz and Leidinger 2008), in areas of the United Kingdom up to 35% (Carter et al. 1994), and in endemic areas of the United States ranges from 20% to 75% (Marcus et al. 1985; Lindenmayer et al. 1989; Bernard et al. 1990). The geographic distribution of *B. burgdorferi* s.l. infection in horses corresponds closely to the distribution of Lyme disease in humans (Burgess 1988). *B. burgdorferi* s.l. infection persists for months (Chang et al. 2000) and some authors believe for life (Chang et al. 2005).

Clinical signs

The incubation period for Lyme disease in horses is not known, but it is 2–5 months in dogs (CFSPH 2005) and can be months to years in humans (Steere et al. 2004). A wide variety of clinical signs have been attributed to *Borrelia* infection in horses including anterior uveitis, behavioural changes, chronic weight loss, hyperaesthesia, low-grade pyrexia, muscle tenderness, stiffness and lameness in multiple limbs and, rarely, encephalitis, joint swelling, neonatal mortality and skin lesions (Burgess et al. 1988; Magnarelli et al. 1988; Parker and White 1992; Hahn et al. 1996). Approximately 10% of seropositive horses develop clinical signs of disease (Magnarelli et al. 1988) and the incidence is higher in foals and yearlings (Cohen et al. 1988). Infections can be persistent and refractory to treatment. In addition, because *B. burgdorferi* s.l. has the ability to convert and reconvert to cystic forms both *in vivo* and *in vitro*, infections can reactivate (Brorson and Brorson 2004). Experimental

infection of ponies produced lesions in the skin, muscle, fascia, nerves and peri-synovial tissues, but no clinical signs of disease (Chang et al. 2000).

Koch's postulates have not been fulfilled for *B. burgdorferi* s.l. in horses and some authors believe the association of *B. burgdorferi* s.l. infection with clinical disease in horses remains speculative (Maloney and Lindenmayer 1992; Browning et al. 1993; Divers 2007).

Diagnosis

Diagnosis of Lyme disease is difficult (Bushmich 1994; Ogden et al. 1997; Divers 2007; Stricker and Johnson 2008) and is usually based on a history of possible exposure, clinical signs of disease, elimination of other diseases, epidemiology, response to antibiotics and serology (Parker and White 1992). *Borrelia* may be identified using dark field or transmission light microscopy of infected tissues or fluids, or by culture of infected material (Chang et al. 1999); however, culture of the organism is slow and susceptible to overgrowth with contaminants (Barbour 1988). Diagnosis is further complicated by the range of clinical signs of disease and possible co-infection with other pathogens, such as *A. phagocytophilum* (Foley et al. 2004).

Serological diagnosis is complicated by the long incubation period, presence of latent infections, cross-reactions with other spirochaetes and persistence of antibody titres for months or years (CFSPH 2005).

Antibodies take 4–6 weeks to develop in horses. Enzyme-linked immunosorbent assay (ELISA) and the indirect fluorescent antibody test (IFAT) are used to diagnose exposure (Bosler et al. 1988; Magnarelli and Anderson 1989). An ELISA value less than 110 units indicates either non-exposure, previous exposure but no current infection, or infection within the previous two months (Chang et al. 2000). The Western blot assay can differentiate antibody responses to the pathogen from vaccine responses. After treatment, titres do not decrease in the IFAT, whole cell ELISA or Western blot tests (CFSPH 2005).

Seronegative Lyme disease has been reported in humans following early antibiotic treatment, which is thought to halt the rise in antibodies (Dattwyler 1988), and false negatives can occur with the ELISA during the first weeks after infection, prior to seroconversion (Cohen et al. 1992).

Treatment

Intravenous tetracycline for four weeks was effective but oral doxycycline was ineffective in eradicating *Borrelia* spp. in experimental infections (Chang et al. 2005). No treatment has been found to be effective in naturally acquired infections. To be considered effective, antibody titres should decline before treatment is discontinued and continue to decline to less than 110 ELISA units (Divers 2007). Antibiotic treatment can accelerate cyst formation of *B. burgdorferi* s.l. (Brorson and Brorson 2004; Murgia and Cinco 2004). It is unknown whether the cystic form of *B. burgdorferi* s.l. is generated after antibiotic treatment in equine Lyme disease (Chang et al. 2005).

Vaccination

Recombinant outer surface lipoprotein A vaccines were trialled in horses and found to be effective in blocking transmission of most infections from the vector tick (Chang et

al. 1999). However there is no commercial vaccine available and the long term viability of vaccination programs in horses has not been evaluated.

Conclusion

Lyme disease is present in approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.26.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of *B. burgdorferi* s.l. being present in an imported horse.

- Seroprevalence in horses in Austria is 33% (Kitrz and Leidinger 2008), up to 35% in endemic areas of the United Kingdom (Carter et al. 1994), and 20–75% in the United States (Lindenmayer et al. 1989).
- The incubation period in horses is not known, but can be years in other species.
- Infections can be subclinical.
- Diagnosis of *B. burgdorferi* s.l. infection is difficult.

Based on these considerations, the likelihood of release of Lyme disease associated with horses from a country where the disease is present was estimated to be '*moderate*'.

Exposure assessment

Transmission is primarily via infected *Ixodes* spp. ticks transferring the organism during feeding on a susceptible host.

Exposure groups are equids, other domestic species (cats, dogs, cattle, sheep) and wildlife.

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to *B. burgdorferi* s.l. via an infected imported horse.

- Transmission to susceptible animals occurs via the bite of *Ixodes* spp. ticks and Australia may have potential vectors.
- Iatrogenic and urine/mucosal transmission of *B. burgdorferi* s.l. in horses could spread the disease to susceptible animals.

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with *B. burgdorferi* s.l. was estimated to be '*low*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*moderate'* combined with the likelihood of exposure estimated to be '*low*', the likelihood of release and exposure for Lyme disease was estimated to be ***'low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to Lyme disease is considered to be establishment and/or spread to populations of susceptible animals nationally through tick vectors.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to Lyme disease.

- Local ticks feeding on infected animals may become infected and act as vectors.
- If potential tick vectors in Australia became infected, they could spread the infection to other animals which may then act as reservoir hosts.
- Birds and small mammals may disseminate the organism across a wide area.
- Iatrogenic transmission could lead to local spread.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of Lyme disease was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of Lyme disease for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Lyme disease is an important zoonotic disease and a range of species are susceptible, including cats, dogs, cattle, sheep, horses and rabbits. Affected animals can be clinically affected and can also act as a source of infection for ticks.
- Approximately 10% of seropositive horses develop clinical signs of disease, which include chronic weight loss, lameness in multiple limbs, joint swelling, encephalitis and neonatal mortality (Burgess et al. 1988; Magnarelli et al. 1988).
- Infections can be persistent, refractory to treatment and reactivate (Brorson and Brorson 2004).
- Clinical signs of disease are rare in reservoir hosts, but there is no information on whether small Australian native mammals would be latently infected.
- Up to 70% of untreated, infected humans suffer effects of invasion of the brain, eyes, heart, joints and nerves which, although rarely fatal, are disabling (Barbour and Fish 1993).

Based on these considerations, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- Clinical signs of disease are rare in reservoir hosts. It is not known whether Australian wildlife would be susceptible to Lyme disease.
- Lyme disease is not considered to have any direct effects on the environment

Based on these considerations, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be *unlikely to be discernible at the local level* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Lyme disease is not a notifiable disease in Australia, there is no AUSVETPLAN Disease Strategy Manual for Lyme disease and the disease is not scheduled in Australia's Emergency Animal Disease Response Agreement.
- A national tick survey and data collection on the geographic distribution and abundance of vectors would be required to assess the extent of *B. burgdorferi* s.l. spread.
- Reduction of tick exposure is limited to insecticide use. Because of the tick's life cycle of two or more years and the redistribution of ticks between each host-feeding event, several applications of an insecticide over a large area are necessary to suppress tick populations.

- Because of the difficulty in controlling ticks, attempts to eradicate Lyme disease via tick control would be unlikely.

Based on these considerations, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- There may be costs associated with tick surveillance and treatment of animals moving between some states.
- Lyme disease is a zoonosis and may have a detrimental effect on tourism in affected rural and regional communities.
- Increased vigilance by owners to treat animals for ticks would increase the cost of owning pets.

Based on these considerations the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- There would be minimal effects as Lyme disease occurs in America, Asia and Europe. It does not occur in New Zealand but New Zealand does not currently have quarantine requirements for Lyme disease.

Based on this consideration, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- In areas where Lyme disease is endemic, reservoirs include birds, dogs, ruminants and wild rodents, in which subclinical infection is common.
- Lyme disease has a wide mammalian reservoir host range however it is not known if Australian wildlife would be susceptible to disease.
- Increased use of acaricides to control ticks could have an effect on a range of arthropod species and disrupt the food source of wildlife, lead to environmental contamination (including water sources) and resistance to acaricides.

Based on these considerations, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Public concern over zoonotic disease may have a detrimental effect on tourism in affected rural and regional communities.
- Resources would be required to manage public health issues.

Based on these considerations, the effect of the establishment and/or spread of Lyme disease in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is 'E', the overall effect associated with the outbreak scenario was considered to be '*moderate*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*moderate*') which results in ***'moderate***' likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of Lyme disease introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure ('***low***') is combined with the likely consequences of establishment and/or spread ('***moderate***') which results in a risk estimation of **LOW**.

Conclusion

The unrestricted risk associated with Lyme disease is determined to be **LOW**. The unrestricted risk estimate exceeds Australia's ALOP and, therefore, risk management is deemed necessary.

A summary of the risk assessment for equine Lyme disease is shown in Figure 5.6 and Table 5.6.

Figure 5.6 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for Lyme disease.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Moderate*
Likelihood of *exposure*	Likelihood of *exposure*	*Low*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socio-economic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5.	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread* associated with each exposure group	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**LOW**

Table 5.6 **Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for Lyme disease.**

References

Anda P, Sanchez-Yebra W, Del Mar Vitutia M, Pastrana EP, Rodriguez I, Miller NS, Backenson PB, Benach JL (1996) A new *Borrelia* species isolated from patients with relapsing fever in Spain. *The Lancet* 348: 162-165.

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

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

Barbour AG (1988) Laboratory aspects of Lyme borreliosis. *Clinical Microbiology Reviews* 1: 399-414.

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

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

Bernard WV, Cohen D, Bosler E, Zamos D (1990) Serologic survey for *Borrelia burgdorferi* antibody in horses referred to a mid-Atlantic veterinary teaching hospital. *Journal of the American Veterinary Medical Association* 196: 1255-1258.

Bosler EM, Cohen DP, Schulze TL, Olsen C, Bernard W, Lissman B (1988) Host responses to *Borrelia burgdorferi* in dogs and horses. *Annals of the New York Academy of Sciences* 539: 221-234.

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

Browning A, Carter SD, Barnes A, May C, Bennett D (1993) Lameness associated with *Borrelia burgdorferi* infection in the horse. *The Veterinary Record* 132: 610-611.

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

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

Burgess EC (1988) *Borrelia burdgorferi* infection in Wisconsin horses and cows. *Annals of the New York Academy of Sciences* 539: 235-243.

Burgess EC, Amundson TE, Davis JP, Kaslow RA, Edelman R (1986) Experimental inoculation of *Peromyscus* spp. with *Borrelia burgdorferi*: evidence of contact transmission. *American Journal of Tropical Medicine and Hygiene* 35: 355-359.

Burgess EC, Gendron-Fitzpatrick A, Mattison M (1988) Foal mortality associated with natural infection of pregnant mares with *Borrelia burgdorferi*. In *Equine infectious diseases V: proceedings of the fifth international conference, October 7-10, 1987, Lexington, Kentucky,* (ed. Powell DG) pp. 217-220, The University Press of Kentucky, Lexington.

Burgess EC, Wachal MD, Cleven TD (1993) *Borrelia burgdorferi* infection in dairy cows, rodents, and birds from four Wisconsin dairy farms. *Veterinary Microbiology* 35: 61-77.

Bushmich SL (1994) Lyme borreliosis in domestic animals. *Journal of Spirochetal and Tick-borne Diseases* 1: 1-6.

Carter SD, May C, Barnes A, Bennett D (1994) *Borrelia burgdorferi* infection in UK horses. *Equine Veterinary Journal* 26: 187-190.

CFSPH (2005) Lyme disease. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 25 February 2009).

Chang Y-F, Ku Y-W, Chang C-F, Chang C-D, McDonough SP, Divers T, Pough M, Torres A (2005) Antibiotic treatment of experimentally *Borrelia burgdorferi*-infected ponies. *Veterinary Microbiology* 107: 285-294.

Chang Y-F, Novosol V, McDonough SP, Chang C-F, Jacobson RH, Divers T, Quimby FW, Shin S, Lein DH (1999) Vaccination against Lyme disease with recombinant *Borrelia burgdorferi* outer-surface protein A (rOspA) in horses. *Vaccine* 18: 540-548.

Chang YF, Novosol V, McDonough SP, Chang CF, Jacobson RH, Divers T, Quimby FW, Shin S, Lein DH (2000) Experimental infection of ponies with *Borrelia burgdorferi* by exposure to Ixodid ticks. *Veterinary Pathology* 37: 68-76.

Cohen D, Bosler EM, Bernard W, Meirs D, Eisner R, Schulze TL (1988) Epidemiologic studies of Lyme disease in horses and their public health significance. *Annals of the New York Academy of Sciences* 539: 244-257.

Cohen ND, Heck FC, Heim B, Flad DM, Bosler EM, Cohen D (1992) Seroprevalence of antibodies to *Borrelia burgdorferi* in a population of horses in central Texas. *Journal of the American Veterinary Medical Association* 201: 1030-1034.

Divers TJ (2007) Lyme disease. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 310-312. Saunders Elsevier, St. Louis.

Doggett SL, Russell RC, Lawrence R, Dickeson D (1997) Lyme disease. Department of Medical Entomology. http://medent.usyd.edu.au/fact/lyme%20disease.htm#intro (Accessed 24 February 2009).

Foley JE, Foley P, Brown RN, Lane RS, Dumler JS, Madigan JE (2004) Ecology of *Anaplasma phagocytophilum* and *Borrelia burgdorferi* in the western United States. *Journal of Vector Ecology* 29: 41-50.

Fridriksdottir V, Nesse LL, Gudding R (1992) Seroepidemiological studies of *Borrelia burgdorferi* infection in sheep in Norway. *Journal of Clinical Microbiology* 30: 1271-1277.

Guttman DS, Wang PW, Wang IN, Bosler EM, Luft BJ, Dykhuizen DE (1996) Multiple infections of *Ixodes scapularis* ticks by *Borrelia burgdorferi* as revealed by single-strand conformation polymorphism analysis. *Journal of Clinical Microbiology* 34: 652-656.

Hahn CN, Mayhew IG, Whitwell KE, Smith KC, Carey D, Carter SD, Read RA (1996) A possible case of Lyme borreliosis in a horse in the UK. *Equine Veterinary Journal* 28: 84-88.

Hudson BJ, Stewart M, Lennox VA, Fukunaga M, Yabuki M, Macorison H, Kitchener-Smith J (1998) Culture-positive Lyme borreliosis. *Medical Journal of Australia* 168: 500-502.

Kawabata H, Masuzawa T, Yanagihara Y (1993) Genomic analysis of *Borrelia japonica* sp. nov. isolated from *Ixodes ovatus* in Japan. *Microbiology and Immunology* 37: 843-848.

Kitrz G, Leidinger E (2008) Lyme borreliosis and anaplasmosis in horses in Austria: a seroprevalence study. In *Proceedings of the 10th international congress of World Equine Veterinary Association, Jan 28-Feb 1, 2008, Moscow, Russia,* p. 647, International Veterinary Information Service, [Moscow].

Lane RS, Burgdorfer W (1987) Transovarial and transstadial passage of *Borrelia burgdorferi* in the western black-begged tick, *Ixodes pacificus* (Acari: Ixodidae). *The American Journal of Tropical Medicine and Hygiene* 37: 188-192.

Lane RS, Regnery DC (1989) Lagomorphs as sentinels for surveillance of borreliosis in the far western United States. *Journal of Wildlife Diseases* 25: 189-193.

Leutenegger CM, Pusterla N, Mislin CN, Weber R, Lutz H (1999) Molecular evidence of coinfection of ticks with *Borrelia burgdorferi* sensu lato and the human granulocytic ehrlichiosis agent in Switzerland. *Journal of Clinical Microbiology* 37: 3390-3391.

Lindenmayer J, Weber M, Onderdonk A, Plain J (1989) *Borrelia burgdorferi* infection in horses. *Journal of the American Veterinary Medical Association* 194: 1384.

Madigan JE (1993) Lyme disease (Lyme borreliosis) in horses. *Veterinary Clinics of North America: Equine Practice* 9: 429-434.

Madigan JE, Teitler J (1988) *Borrelia burgdorferi* borreliosis. *Journal of the American Veterinary Medical Association* 192: 892-904.

Magnarelli LA, Anderson JF (1989) Class-specific and polyvalent enzyme-linked immunosorbent assays for detection of antibodies to *Borrelia burgdorferi* in equids. *Journal of the American Veterinary Medical Association* 195: 1365-1368.

Magnarelli LA, Anderson JF, Barbour AG (1986) The etiologic agent of Lyme disease in deer flies, horse flies, and mosquitoes. *The Journal of Infectious Diseases* 154: 355-358.

Magnarelli LA, Anderson JF, Fish D (1987) Transovarial transmission of *Borrelia burgdorferi* in *Ixodes dammini* (Acari: Ixodidae). *The Journal of Infectious Diseases* 156: 234-236.

Magnarelli LA, Anderson JF, Levine HR, Levy SA (1990) Tick parasitism and antibodies to *Borrelia burgdorferi* in cats. *Journal of the American Veterinary Medical Association* 197: 63-66.

Magnarelli LA, Anderson JF, Shaw E, Post JE, Palka FC (1988) Borreliosis in equids in northeastern United States. *American Journal of Veterinary Research* 49: 359-362.

Maloney EM, Lindenmayer JL (1992) Seroprevalence and clinical signs of Lyme disease in Cape Cod horses. *Equine Practice* 14: 15-19.

Manion TB, Khan MI, Dinger J, Bushmich SL (1998) Viable *Borrelia burgdorferi* in the urine of two clinically normal horses. *Journal of Veterinary Diagnostic Investigation* 10: 196-199.

Marcelis L, de Marneffe P, Chaidron E, Bigaignon G, Kageruka P, Goubau P (1987) Horse reservoir for *Borrelia burgdorferi*? *The Lancet* 1: 977.

Marcus LC, Patterson MM, Gilfillan RE, Urband PH (1985) Antibodies to *Borrelia burgdorfer*i in New England horses: serologic survey. *American Journal of Veterinary Research* 46: 2570-2571.

Murgia R, Cinco M (2004) Induction of cystic forms by different stress conditions in *Borrelia burgdorferi*. *Acta Pathologica, Microbiologica et Immunologica Scandinavica* 112: 57-62.

Ogden NH, Nuttall PA, Randolph SE (1997) Natural Lyme disease cycles maintained via sheep by co-feeding ticks. *Parasitology* 115: 591-599.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Olsen B, Duffy DC, Jaenson TG, Gylfe A, Bonnedahl J, Bergstrom S (1995) Transhemispheric exchange of Lyme disease spirochetes by seabirds. *Journal of Clinical Microbiology* 33: 3270-3274.

Olsen B, Jaenson TG, Noppa L, Bunikis J, Bergstrom S (1993) A Lyme borreliosis cycle in seabirds and *Ixodes uriae* ticks. *Nature* 362: 340-342.

Parker JL, White KK (1992) Lyme borreliosis in cattle and horses: a review of the literature. *The Cornell Veterinarian* 82: 253-274.

Piesman J, Stone BF (1991) Vector competence of the Australian paralysis tick, *Ixodes holocyclus*, for the Lyme disease spirochete *Borrelia burgdorferi*. *International Journal for Parasitology* 21: 109-111.

Rothwell JT, Christie BM, Williams C, Walker K (1989) Suspected Lyme disease in a cow. *Australian Veterinary Journal* 66: 296-298.

Roux V, Raoult D (1999) Body lice as tools for diagnosis and surveillance of reemerging diseases. *Journal of Clinical Microbiology* 37: 596-599.

Russell RC (1995) ?Lyme disease in Australia: still to be proven! *Emerging Infectious Diseases* 1: 29-31.

Russell RC, Doggett SL, Munro R, Ellis J, Avery D, Hunt C, Dickeson D (1994) Lyme disease: a search for a causative agent in ticks in south-eastern Australia. *Epidemiology and Infection* 112: 375-384.

Ryffel K, Peter O, Rutti B, Suard A, Dayer E (1999) Scored antibody reactivity determined by immunoblotting shows an association between clinical manifestations and presence of *Borrelia burgdorferi* sensu stricto, *B. garinii*, *B. afzelii*, and *B. valaisiana* in humans. *Journal of Clinical Microbiology* 37: 4086-4092.

Steere AC, Coburn J, Glickstein L (2004) The emergence of Lyme disease. *Journal of Clinical Microbiology* 113: 1093-1101.

Stricker RB, Johnson L (2008) Serologic tests for Lyme disease: more smoke and mirrors. *Clinical Infectious Diseases* 47: 1111-1112.

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

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

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

5.27 Nipah virus encephalitis

5.27.1 Technical information

Background

Nipah virus is closely related to Hendra virus, which is present in Australia. They are both members of the henipavirus genus in the family Paramyxoviridae (Lamb et al. 2005). Both are highly pathogenic in humans. Nipah and Hendra viruses vary in the range of species they infect and the manner and ease with which they are transmitted. Nipah virus appears to spread easily between pigs and from pigs to humans, whereas Hendra virus is not readily contagious between animals (Hooper and Williamson 2000). Nipah virus has naturally infected pigs, cats, dogs, horses, goats and humans (CFSPH 2007). Experimental infection has been demonstrated in guinea pigs and hamsters (Eaton et al. 2005).

In late September 1998 in Peninsular Malaysia, an outbreak in humans of severe febrile encephalitis associated with deaths was reported. This was initially thought to be Japanese encephalitis and was later attributed to the novel Nipah virus. Similar disease in pigs and humans was seen in other regions in Malaysia by February 1999 (Chua et al. 2000). Another outbreak of encephalitis and pneumonia occurred in abattoir workers in Singapore in March 1999 and was confirmed as Nipah virus infection (Paton et al. 1999). The abattoir had imported pigs from a farm in Malaysia affected by Nipah virus. The outbreak was contained when the importation of pigs was suspended and abattoir closed (Paton et al. 1999).

Nipah virus has subsequently been identified in Bangladesh and India. Unlike the Malaysian and Singaporean outbreaks, human infections in Bangladesh did not seem to be linked to exposure to bat reservoir hosts (OIE 2008). Human-to-human transmission was suspected in outbreaks of Nipah virus in Bangladesh (Hsu et al. 2004). Drinking fresh date palm sap was suggested as transmitting Nipah virus in an outbreak in Bangladesh in 2004 (Luby et al. 2006). Nipah virus has never been reported in Australia.

Nipah virus encephalitis is an OIE-listed disease of pigs (OIE 2009). There is a chapter in the OIE Manual relating to the disease (OIE 2008) but there are no recommendations in the Code for the importation of animals with respect to Nipah virus encephalitis.

Epidemiology

Fruit bats in the genus *Pteropus* are the reservoir hosts of both Nipah and Hendra viruses (OIE 2008). Pteropid bats are widely distributed from Madagascar, through the Indian subcontinent to south-east Asia and Australia, through the Pacific to the Cook Islands, and the southernmost islands of Japan (Eaton et al. 2005). Antibodies to Nipah virus, or assumed closely related viruses, have been reported in pteropid bats in Bangladesh, Cambodia, China, Indonesia, Madagascar and Thailand (Li et al. 2008; OIE 2008). Malaysian pteropid bats have a seroprevalence for Nipah virus antibodies of up to 20% (OIE 2008).

Nipah virus can be transmitted directly or indirectly from bats to pigs and the disease is highly contagious between pigs. Pigs act as amplifying hosts and transmit virus by

direct contact to other animals and humans. Direct or indirect bat-to-human transmission appears to have been responsible for some outbreaks and human-to-human spread has been reported (CFSPH 2007).

Serological surveys in Malaysia have demonstrated a seroprevalance of 15–55% in dogs, 4–6% in cats and 1.5% in goats (CFSPH 2007).

Screening of polo, equestrian event and racing horses in Malaysia was conducted in March and April 1999 (Mahendran et al. 1999). Of more than 3200 serum samples tested by neutralisation assay and enzyme-linked immunosorbent assay (ELISA), only two horses were positive by neutralisation assay. These were from 47 horses tested in a polo club (OIE 1999; Mahendran et al. 1999). The two apparently healthy animals were euthanased and no gross lesions were found on post-mortem examination. All tissues examined were negative on polymerase chain reaction (PCR) for Nipah virus and no virus was isolated, although one of the horses had mild encephalitis. Retrospective studies on serum samples collected in November 1998 from the same polo club showed three horses positive to ELISA and neutralisation assay. Subsequent sampling and testing of these horses were negative (Mahendran et al. 1999).

The affected horses in Malaysia were at one time stationed at a pig farm and the polo club was located within the Nipah virus outbreak area (Mahendran et al. 1999). The national horse population of Malaysia was found to be free from Nipah virus in subsequent serological surveillance undertaken in 1999 and 2000 (OIE 2001).

More than 500 horses in Singapore were tested and found to be seronegative for Nipah virus (Chan et al. 2002).

A serological survey to screen humans potentially exposed to Nipah virus in Singapore found 22 of 1469 people tested had antibodies suggesting Nipah virus infection, of which 12 were subclinical (Chan et al. 2002). All 22 seropositive individuals had direct association with pigs. None of the people in the study who worked with horses in Singapore had evidence of infection. This further suggests that horses in Singapore were not involved in the outbreak (Chan et al. 2002).

Infection of horses with Nipah virus is similar to infection in other species — a generalised vasculitis with possibility of localisation in the lung or brain — and the few cases of infection described in horses may have originated from pigs (Hooper and Williamson 2000).

Diagnosis

Identification methods following virus isolation include immunostaining, neutralisation and molecular characterisation (OIE 2008). Real-time PCR is useful for detection of viral genome in specimens and is particularly sensitive (OIE 2008). Immunohistochemistry can be performed on formalin-fixed tissues (OIE 2008).

Serological tests available are virus neutralisation (currently accepted as the reference procedure) and ELISA (OIE 2008).

Conclusion

Nipah virus has not been reported in horses in any approved country. Accordingly, no further analysis was necessary.

However, Nipah virus is an emerging disease present in countries neighbouring approved countries and there is an unknown potential for spread. In the event that Nipah virus is detected in domestic animals in an approved country, Australia may implement provisional quarantine measures pending further investigation and assessment.

References

CFSPH (2007) Nipah virus infection. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/Factsheets/pdfs/nipah.pdf (Accessed 3 December 2008).

Chan KP, Rollin PE, Ksiazek TG, Leo YS, Goh KT, Paton NI, Sng EH, Ling AE (2002) A survey of Nipah virus infection among various risk groups in Singapore. *Epidemiology and Infection* 128: 93-98.

Chua KB, Bellini WJ, Rota PA, Harcourt BH, Tamin A, Lam SK, Ksiazek TG, Rollin PE, Zaki SR, Shieh W-J, Goldsmith CS, Gubler DJ, Roehrig JT, Eaton B, Gould AR, Olson J, Field H, Daniels P, Ling AE, Peters CJ, Anderson LJ, Mahy BWJ (2000) Nipah virus: a recently emergent deadly paramyxovirus. *Science* 288: 1432-1435.

Eaton BT, Broder CC, Wang L-F (2005) Hendra and Nipah viruses: pathogenesis and therapeutics. *Current Molecular Medicine* 5: 805-816.

Hooper PT, Williamson MM (2000) Hendra and Nipah virus infections. *Veterinary Clinics of North America: Equine Practice* 16: 597-603.

Hsu VP, Hossain MJ, Parashar UD, Ali MM, Ksiazek TG, Kuzmin I, Niezgoda M, Rupprecht C, Bresee J, Breiman RF (2004) Nipah virus encephalitis reemergence, Bangladesh. *Emerging Infectious Diseases* 10: 2082-2087.

Lamb RA, Collins PL, Kolakofsky D, Melero JA, Nagai Y, Oldstone MBA, Pringle CR, Rima BK (2005) Paramyxoviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 655-668. Elsevier, San Diego.

Li Y, Wang J, Hickey AC, Zhang Y, Li Y, Wu Y, Zhang H, Yuan J, Han Z, McEachern J, Broder CC, Wang L-F, Shi Z (2008) Antibodies to Nipah or Nipah-like viruses in bats, China. *Emerging Infectious Diseases* 14: 1974-1976.

Luby SP, Rahman M, Hossain MJ, Blum LS, Husain MM, Gurley E, Khan R, Ahmed B-N, Rahman S, Nahar N, Kenah E, Comer JA, Ksiazek TG (2006) Foodborne transmission of Nipah virus, Bangladesh. *Emerging Infectious Diseases* 12: 1888-1894.

Mahendran R, Naseem M, Shahirudin S, Aziz AJ (1999) A preliminary study on the sero-prevalence of Nipah virus infection in horses in Malaysia. In *Proceedings of the National Congress of Animal Health and Production, Alor Gajah, Malaysia, September 3-4, 1999,* pp. 423-424, Veterinary Association of Malaysia, Malaysia.

OIE (1999) Nipah disease in Malaysia. Disease Information. ftp://ftp.oie.int/infos_san_archives/eng/1999/en_990528v12n20.pdf (Accessed 2008).

OIE (2001) 6. Malaysia: declaration of freedom from Nipah virus in domestic animals from 1 June 2001. *Bulletin de l'Office International des Epizooties* 113: 514-515.

OIE (2008) Hendra and Nipah virus disease. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/Eng/Normes/Mmanual/2008/pdf/2.09.06_HENDRA_&_NIPAH_FINAL.pdf (Accessed 1 September 2009).

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

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

5.28 Potomac horse fever

5.28.1 Technical information

Background

Potomac horse fever (PHF) is also known as equine monocytic ehrlichiosis, equine neorickettsiosis and equine ehrlichial colitis. PHF is an acute, pyrexic enterocolitis of horses, caused by *Neorickettsia risticii* (formerly *Ehrlichia risticii*) (Palmer 2004). First recognised in the United States in 1979 along the Potomac River in Maryland, PHF has subsequently been reported in 42 other states, Brazil, Canadian provinces of Nova Scotia, Ontario and Alberta, and Uruguay (Dutra et al. 2001). Serological evidence of PHF has been reported in France (Vidor, 1988), however there are no reports of clinical or epidemiological evidence of infection. In the United States, seroprevalence in the mid-west and east coast regions averages 25%. PHF has not been reported in Australia.

Serological studies in endemic regions have found antibody titres specific for *N. ristcii* in cats, coyotes, goats and pigs (Pusterla et al. 2000a). Other studies have shown that cattle and dogs are susceptible to infection (Perry et al. 1989; Pusterla et al. 2001). Eleven strains of *N. risticii* have been identified (Pusterla and Madigan 2007).

PHF is not an OIE-listed disease (OIE 2009).

Epidemiology

N. risticii is maintained in nature in a complex aquatic ecosystem. The complete life cycle is not clear, but it is known that *N. risticii* infects *Acanthatrium* spp. and *Lecithodendrium* spp. trematodes. Insectivorous bats, birds and amphibians in North America are the definitive hosts of these trematodes. There is strong evidence that *Acanthatrium* spp and *Lecithodendrium* spp. are the vectors of *N. risticii* (Barlough et al. 1998). The trematode cycles through aquatic intermediate hosts — freshwater snails; *Juga* spp. and *Elimia* spp. (Barlough et al. 1998; Pusterla et al. 2000b) — and aquatic insects: including caddis flies, mayflies, damselflies, stoneflies and dragonflies (Chae et al. 2000). Additional trematodes may act as vectors of *N. risticii* in other endemic regions (Radostits et al. 2007). It is not known if competent trematode vectors or snail and arthropod intermediate hosts are present in Australia (T. Cribb, University of Queensland, pers. comm. March 2009; I. Beveridge, University of Melbourne, pers. comm. March 2009).

N. risticii has been experimentally transmitted to horses by subcutaneous inoculation with *N. risticii*-infected virgulate cercariae from *Juga yrekaensis* snails (Pusterla et al. 2000b); by parental administration of *N. risticii* or blood from infected horses (Pusterla and Madigan 2007), and by the oral route using faeces from infected horses (Palmer and Benson 1994). However, there is no evidence of transmission of *N. ristscii* or seroconversion by direct contact (Palmer and Benson 1994). Experimental oral transmission using infected aquatic insects produces a similar clinical disease to that seen in naturally infected horses (Mott et al. 2002). The likely route of infection for horses is the ingestion, in water or pasture, of aquatic insect

larvae and nymphs that are infected with trematodes containing *N. risticii* (Pusterla and Madigan 2007).

It appears that horses are accidentally infected with *N. risticii*, are unlikely to be a source of infection (Radostits et al. 2007) and are, therefore, a dead-end host.

Clinical signs

PHF is not contagious and typically occurs from mid to late summer near freshwater rivers and on irrigated pastures (Radostits et al. 2007). The incubation period is 1–3 weeks. Clinical signs of disease range from inapparent infection, through transient pyrexia and depression to severe anorexia, diarrhoea, colic and laminitis (Radostits et al. 2007). During on-farm epidemics, morbidity is 20–50% and mortality in untreated horses varies from 5% to 30%, depending on the strain of organism involved (Pusterla and Madigan 2007). Approximately 60% of horses develop diarrhoea, the severity of which varies — but it is usually profuse and projectile. In 30% of cases, laminitis occurs within three days of initial signs of disease. Transplacental transmission has been reported in both natural and experimental infection and is associated with delayed abortion (Long et al. 1995; Coffman et al. 2008).

Affected horses usually respond well to treatment with oxytetracycline and develop a sterile immunity that persists for at least 20 months (Palmer et al. 1990).

Diagnosis

The diagnosis of PHF is usually based on clinical signs of disease, and seasonal and geographical occurrence. It is confirmed by isolation of *N. risticii* from blood or faeces. Serological testing is of limited use because antibodies to *N. risticii* may not be detectable for some time after infection. The immunofluorescent antibody test has a high rate of false-positive reactions, thought to be secondary to routine vaccination of horses with non-*N. risticii* vaccines (Madigan et al. 1995). The real-time polymerase chain reaction test on blood and faeces, based on automatic nucleic acid extraction, allows detection of *N. risticii* DNA within 24 hours (Pusterla et al. 2000b).

Conclusion

PHF is present in some approved countries. Horses are considered dead-end hosts.

PHF was not considered further in the IRA.

References

Barlough JE, Reubel GH, Madigan JE, Vrdoljak A, Vredevoe LK, Rikihisa Y (1998) Detection of *Ehrlichia risticii*, the agent of Potomac horse fever, in freshwater stream snails (Pleuroceridae: *Juga* spp.) from northern California. *Applied and Environmental Microbiology* 64: 2888-2893.

Chae J-S, Pusterla N, Johnson E, DeRock E, Lawler SP, Madigan JE (2000) Infection of aquatic insects with trematode metacercariae carrying *Ehrlichia risticii*, the cause of Potomac horse fever. *Journal of Medical Entomology* 37: 619-625.

Coffman EA, Abd-Eldaim M, Craig LE (2008) Abortion in a horse following *Neorickettsia risticii* infection. *Journal of Veterinary Diagnostic Investigation* 20: 827-830.

Dutra F, Schuch LF, Delucchi E, Curcio BR, Coimbra H, Raffi MB, Dellagostin O, Riet-Correa F (2001) Equine monocytic Ehrlichiosis (Potomac horse fever) in horses in Uruguay and southern Brazil. *Journal of Veterinary Diagnostic Investigation* 13: 433-437.

Long MT, Goetz TE, Kakoma I, Whiteley HE, Lock TE, Holland CJ, Foreman JH, Baker GJ (1995) Evaluation of fetal infection and abortion in pregnant ponies experimentally infected with *Ehrlichia risticii* . *American Journal of Veterinary Research* 56: 1307-1316.

Madigan JE, Rikihisa Y, Palmer JE, DeRock E, Mott J (1995) Evidence for a high rate of false-positive results with the indirect fluorescent antibody test for *Ehrlichia risticii* antibody in horses. *Journal of the American Veterinary Medical Association* 207: 1448-1453.

Mott J, Muramatsu Y, Seaton E, Martin C, Reed S, Rikihisa Y (2002) Molecular analysis of *Neorickettsia risticii* in adult aquatic insects in Pennsylvania, in horses infected by ingestion of insects, and isolated in cell culture. *Journal of Clinical Microbiology* 40: 690-693.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Palmer JE (2004) Potomac horse fever. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 583-591. Oxford University Press, Oxford.

Palmer JE, Benson CE (1994) Studies on oral transmission of Potomac horse fever. *Journal of Veterinary Internal Medicine* 8: 87-92.

Palmer JE, Benson CE, Whitlock RH (1990) Resistance to development of equine ehrlichial colitis in experimentally inoculated horses and ponies. *American Journal of Veterinary Research* 51: 763-765.

Perry BD, Schmidtmann ET, Rice RM, Hansen JW, Fletcher M, Turner EC, Robl MG, Hahn NE (1989) Epidemiology of Potomac horse fever: an investigation into the possible role of non-equine mammals. *The Veterinary Record* 125: 83-86.

Pusterla N, Berger Pusterla J, DeRock E, Madigan JE (2001) Susceptibility of cattle to *Ehrlichia risticii*, the causative agent of Potomac horse fever. *The Veterinary Record* 148: 86-87.

Pusterla N, Chang CC, Chomel BB, Chae JS, Foley JE, DeRock E, Kramer VL, Lutz H, Madigan JE (2000a) Serologic and molecular evidence of *Ehrlichia* spp. in coyotes in California. *Journal of Wildlife Diseases* 36: 494-499.

Pusterla N, Leutenegger CM, Sigrist B, Chae J-S, Lutz H, Madigan JE (2000b) Detection and quantitation of *Ehrlichia risticii* genomic DNA in infected horses and snails by real-time PCR. *Veterinary Parasitology* 90: 129-135.

Pusterla N, Madigan JE (2007) *Neorickettsia risticii*. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 357-362. Saunders Elsevier, St. Louis.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with Rickettsiales. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1455-1470. Saunders Elsevier, Edinburgh.

5.29 Rabies

5.29.1 Technical information

Background

Rabies is a virus in the genus Lyssavirus of the family Rhabdoviridae (Tordo et al. 2005) which causes a progressively fatal encephalitis in mammals. Lyssaviruses are classified phylogenetically into seven genotypes. Genotype 1 is 'classical rabies' and there are six other genotypes of rabies-related viruses. All genotypes can cause disease in mammals but only genotype 1 is of consequence to the IRA because the other genotypes have not been reported in horses. In the United States in 2007, there were 42 cases of rabies in horses (Blanton et al. 2008).

Rabies is a multiple species OIE-listed disease (OIE 2009a).

Epidemiology

Rabies is usually transmitted through the bite of a rabid animal, particularly of dogs and other carnivores.

Horses are mostly infected by the virus as a 'spill over' from endemic cycles. Horses are considered dead-end hosts because they usually succumb to disease and die without further transmission. Although horses are very unlikely to maintain a cycle of rabies they can be involved in secondary transmission to humans; however, this is rare.

Clinical signs

The incubation period in horses is variable. It is dependent on the distance of the bite site from the central nervous system, the dose of virus injected, the level of sensory innervation at the bite site and the rabies virus variant. One study in the United States found that the average period was 12.3 days (Hudson et al. 1996). For the purposes of importation, the Code recommends that the incubation period for rabies is six months (OIE 2009b).

Once clinical signs of rabies develop the horse will die within days. In equids, periods of marked excitement and aggressiveness alternate with periods of relative calm. In periods of excitement, animals become restless, stare, paw at the ground and can salivate excessively. Other clinical signs in equids include teeth grinding, signs similar to acute colic and biting or rubbing at the site of exposure, causing self-mutilation. As paralysis develops, the animals fall repeatedly, become comatose and die.

There is no effective treatment for infected animals.

Diagnosis

Rabies virus is usually diffusely distributed throughout the brain of infected animals. The brain stem is the most suitable site for detection of virus.

There are a number of tests available to confirm the diagnosis of rabies but these require post mortem, from samples of brain tissue. These include antigen detection assays such as fluorescent antibody tests, nucleic acid detection assays such as

polymerase chain reaction tests and viral cultures such as the rabies tissue culture infection test (OIE 2008).

No reliable diagnostic tests for rabies are available for use on a live horse.

Conclusion

Rabies is present in some approved countries. The Code recommendations (OIE 2009b) include premises freedom. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Blanton JD, Palmer D, Christian KA, Rupprecht CE (2008) Rabies surveillance in the United States during 2007. *Journal of the American Veterinary Medical Association* 233: 884-897.

Hudson LC, Weinstock D, Jordan T, Bold-Fletcher NO (1996) Clinical presentation of experimentally induced rabies in horses. *Journal of Veterinary Medicine, Series B* 43: 277-285.

OIE (2008) Rabies. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.13_RABIES.pdf (Accessed 1 January 2009).

OIE (2009a) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2009b) Rabies. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.10.htm (Accessed 19 August 2009).

Tordo N, Benmansour A, Calisher C, Dietzgen RG, Fang RX, Jackson AO, Kurath G, Nadin-Davis S, Tesh RB, Walker PJ (2005) Rhabdoviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 623-643. Elsevier, San Diego.

5.30 Rift Valley fever

5.30.1 Technical information

Background

Rift Valley fever (RVF) is an arthropod-borne viral zoonotic disease. The causal virus, Rift Valley fever virus (RVFV), belongs to the family Bunyaviridae, genus Phlebovirus (Nichol et al. 2005). RVF is endemic in Madagascar, Saudi Arabia and Yemen in the Arabian Peninsula and sub-Saharan Africa (OIE 2008a). RVFV affects a large number of species, including camels, monkeys, rodents and ruminants. Horses exhibit viraemia without signs of disease (Yedloutschnig et al. 1981; Wood et al. 1990). The disease is of most importance in ruminants (Radostits et al. 2007).

RVF is a multiple species OIE-listed disease (OIE 2009a). There are no recommendations in the Code for the purpose of international trade for live animals other than ruminants (OIE 2009b).

Epidemiology

RVF occurs as cyclical epidemics, with quiescent periods in between. Vectors for transmission of RVFV are haematophagous insects, primarily mosquitoes. Various species of *Aedes* mosquitoes have been identified as, or suspected to be, vectors of RVFV. Sandflies and culicoides midges are also vectors, but do not play a role in maintenance of the virus (Radostits et al. 2007). Several mosquito species present in Australia are considered to be competent vectors for RVFV (Turell and Kay 1998).

Mosquitoes transmit RVFV both vertically (transovarially) to the next generation of mosquitoes and horizontally to other susceptible vertebrates (Wilson 1994). Ruminants are highly susceptible to RVF and are the major amplifying hosts. Epidemics occur during periods of high rainfall, when vector numbers increase. Virus can be found in aborted foetuses, faeces and milk (Radostits et al. 2007).

Horses are not important in the maintenance of disease cycles. The level of viraemia reported in horses is less than that required to successfully infect vectors (Yedloutschnig et al. 1981; Ramachandran and Manohar 1996).

Clinical signs

Horses infected with RVFV do not show clinical signs of disease. A low grade viraemia occurs with subsequent seroconversion (Yedloutschnig et al. 1981; Wood et al. 1990).

In endemic regions, abortion storms and neonatal mortalities in cattle are the initial manifestation of an epidemic (Radostits et al. 2007).

Diagnosis

Diagnosis of RVF can be based on isolation of virus, demonstration of viral antigens and by serological tests (virus neutralisation, enzyme-linked immunosorbent assay and haemagglutination inhibition tests) (OIE 2008b).

Conclusion

RVF is not present in any approved country and is not transmitted by horses. There are no recommendations in the Code for horses.

Rift Valley fever was not considered further in the IRA.

References

Nichol ST, Beaty BJ, Elliott RM, Goldbach R, Plyusnin A, Schmaljohn CS, Tesh RB (2005) Bunyaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 695-716. Elsevier, San Diego.

OIE (2008a) List of countries by disease situation: Rift Valley fever. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_status_lists&disease_type=Terrestrial&disease_id=8&empty=999999 (Accessed 24 February 2009).

OIE (2008b) Rift Valley fever. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2008. http://www.oie.int/eng/Normes/mmanual/2008/pdf/2.01.14_RVF.pdf (Accessed 22 October 2008).

OIE (2009a) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2009b) Rift Valley fever. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/en_chapitre_1.8.11.pdf (Accessed 18 August 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia I. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1157-1305. Saunders Elsevier, Edinburgh.

Ramachandran S, Manohar BM (1996) Is horse susceptible to Rift Valley fever? *Centaur* XIII: 19-20.

Turell MJ, Kay BH (1998) Susceptibility of selected strains of Australian mosquitoes (Diptera: Culicidae) to Rift Valley fever virus. *Journal of Medical Entomology* 35: 132-135.

Wilson ML (1994) Rift Valley fever virus ecology and the epidemiology of disease emergence. *Annals of the New York Academy of Sciences* 740: 169-180.

Wood OJ, Meegan JM, Morrill JC, Stephenson EH (1990) Rift Valley fever virus. In *Virus infections of ruminants* (eds. Dinter Z, Morein B) pp. 481-494. Elsevier Science Publishers, Amsterdam.

Yedloutschnig RJ, Dardiri AH, Walker JS (1981) The response of ponies to inoculation with Rift Valley fever virus. *Contributions to Epidemiology and Biostatics* 3: 68-71.

5.31 Schistosomiasis

5.31.1 Technical information

Background

Schistosomiasis is a serious zoonosis caused by trematodes of the genus *Schistosoma* in the family Schistosomatidae (Kassai 1999). The species that affect horses are *Schistosoma indicum, S. intercalatum, S. japonicum*, *S. mattheei, S. nasale* and *S. spindale*. These species are found in Africa, India, Pakistan, the Far East, east and southeast Asia (Kassai 1999) and their intermediate hosts are freshwater snails. None of these schistosome species nor their intermediate hosts is present in Australia (J. Walker, University of Sydney, pers. comm. May 2009).

Schistosomiasis is not an OIE-listed disease (OIE 2009).

Epidemiology

Unlike other trematodes, which are hermaphrodites, the sexes are separate in schistosomes. Schistosomes have an indirect life cycle and their intermediate hosts are freshwater snails of the species *Bulinus*, *Biomphalaria* and *Oncomelania* (Kassai 1999). All schistosomes are dependent on water as a medium for infection of both the intermediate and definitive hosts. Percutaneous infection can also occur. Transmission is seasonal as it is related to high rainfall and high temperature (Urquhart et al. 1996).

Clinical signs

The prepatent period is 30 days or longer. Schistosomes are found in the blood vessels of the alimentary tract and bladder. Clinical signs of infection are mainly a result of eggs becoming lodged in host tissues and the host's immunological response. Signs of acute infection include intermittent diarrhoea, anaemia, progressive weakness, weight loss and death. Chronic infection is often subclinical although heavier infections can cause anaemia, progressive weight loss, emaciation and death. Most infections in animals are of little or no pathogenic significance (Kassai 1999).

Diagnosis

Diagnosis is based on clinical and pathological findings, history of access to natural water sources and post-mortem findings. Demonstration of eggs in faeces or urine is definitive evidence of an existing infection. However, faecal egg counts have poor sensitivity (unreliable in mild and older infections) and are time consuming and laborious (Kassai 1999).

Conclusion

Schistosoma spp. that can infect horses are present in some approved countries. The intermediate hosts for these species are not present in Australia.

Schistosomiasis was not considered further in the IRA.

References

Kassai T (1999) Phylum: Platyhelminthes flatworms. In *Veterinary helminthology* pp. 3-50. Butterworth-Heinemann, Oxford.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Urquhart GM, Armour J, Duncan JL, Dunn AM, Jennings FW (1996) Veterinary helminthology. In *Veterinary parasitology* pp. 3-138. Blackwell Science, Oxford.

5.32 Screw-worm-fly myiasis

5.32.1 Technical information

Background

Two species of flies cause screw-worm-fly myiasis — New World screw-worm, *Cochlioma hominivorax* and Old World screw-worm, *Chrysomya bezziana*. Both species are members of the family Calliphoridae, subfamily Chrysomyinae. Screw-worms are the larvae of flies that feed on living flesh. 'New World' refers to the Americas and 'Old World' to Africa, Asia and Europe. *C. hominivorax* has never been reported in Canada and was eradicated from the United States with the last cases reported in 1982 (Branckaert et al. 1991; OIE 2008a). *C. bezziana* has not been reported in European countries and was last reported in the United Arab Emirates in cattle in 2000 (OIE 2008b). In Hong Kong, *C. bezziana* myiasis has been reported in dogs, cattle and pigs but not horses (FEHD 2006). It is postulated that the flies have been introduced into Hong Kong from southern China (FEHD 2006).

Both species of flies can affect all warm-blooded animals, including humans. Infections in birds are rare (CFSPH 2007).

C. hominivorax and *C. bezziana* have similar climatic requirements. Australia is the only continent with a suitable climate where screw-worm-fly has not established.

C. hominivorax and *C. bezziana* are multiple species OIE-listed diseases (OIE 2009b).

Epidemiology

Both *C. hominivorax* and *C. bezziana* are obligate parasites of warm-blooded animals. Adult females lay eggs in masses at wound margins or body orifices of living animals. Screw-worm-flies tend to be attracted to parts of the animal exposed by husbandry operations (e.g. castration and dehorning wounds, severed umbilical cords), where skin has been perforated and exudes blood, and to areas as small as a tick bite. Three instars of larval development follow in the living host tissue. Third stage larvae have heavy bands of backwardly directed thorn-like spines — hence the name 'screw-worm'.

Clinical signs

Screw-worm-fly myiasis produces a characteristic odour. Initially a small wound may be difficult to see due to fur or hair covering its location (e.g. prepuce, vulva, ear canal). Later, wounds become larger and a secondary strike may result in hundreds of larvae at different stages of development in the wound. Secondary infection and tissue necrosis follow in untreated cases, resulting in weight loss, debility and death.

Diagnosis

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

Conclusion

C. hominivorax and *C. bezziana* are not reported in horses in any approved country. However, *C. bezziana* has been reported in other animal species in some approved countries. The Code recommendations (OIE 2009a) include examination for infested wounds. Certification requirements, in accordance with the Code, will be included in Australia's quarantine measures.

Accordingly, no further analysis was necessary.

References

Branckaert RDS, Perlis A, Roland N, Gigli H, Criscuolo M, Cunningham EP, Kouba V, Qureshi AW, Phelan J, Lynnerup E, Richmond K (1991) New World screwworm response to an emergency. *World Animal Review* Special issue: online.

CFSPH (2007) Screwworm myiasis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 10 February 2009).

FEHD (2006) *Chrysomya bezziana*. The Government of Hong Kong, Food and Environmental Hygiene Department. http://www.fehd.gov.hk/safefood/pest-post-chrysomya.html (Accessed 24 February 2009).

OIE (2008a) List of countries by disease situation: N. w. screwworm (*C. hominivorax*). WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_status_lists&disease_type=Terrestrial&disease_id=25&empty=999999 (Accessed 17 February 2009).

OIE (2008b) List of countries by disease situation: O. w. screwworm (*C. bezziana*). WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_status_lists&disease_type=Terrestrial&disease_id=26&empty=999999 (Accessed 17 February 2009).

OIE (2009a) New World screwworm (*Cochloiomyia hominivorax*) and Old World screwworm (*Chrysomya bezziana*). Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.8.htm (Accessed 19 August 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

5.33 Surra

5.33.1 Technical information

Background

Surra is primarily a disease of camels, dogs, horses and water buffalo caused by the flagellate protozoan *Trypanosoma evansi*. Chronic wasting disease is most common in camels, whereas in dogs, horses and water buffalo the disease is usually acute and fatal if untreated (Losos 1980; Taylor and Authie 2004). Infection can persist in animals that recover from acute disease resulting in recurrent disease. Subclinical infection, reduced productivity or chronic disease occurs in all susceptible animals including cattle, goats, sheep, llamas, cats, pigs and elephants (Abo-Shehada et al. 1999; Radostits et al. 2007; Sellon 2007). Acute and chronic disease has been reported in donkeys and mules (Pathak et al. 1999; Tuntasuvan et al. 2003). Wallabies are susceptible to experimental infection and develop acute fatal disease (Reid et al. 2001b).

T. evansi is a member of the family Trypanosomatidae. It is morphologically identical to *T. equiperdum*, the cause of dourine, and to the slender form of *T. brucei brucei*, a cause of nagana (Stevens and Brisse 2004). Some authors consider *T. evansi* to be synonymous with *T. equiperdum* (Monzón and Russo 1997). Distinction of these trypanosomes at the species level remains uncertain despite phylogenetic studies (Brun et al. 1998; Haag et al. 1998; Claes 2003).

Adaption of *T. evansi* to mechanical transmission by biting flies outside of the tsetse fly zone has resulted in it being the most widely distributed pathogenic trypanosome. Surra occurs across Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East (Radostits et al. 2007; OIE 2008a). Significant livestock production and economic losses due to surra occur in endemic areas. Surra is endemic in camels in the United Arab Emirates; the last outbreak reported to the OIE was in 2004 (OIE 2009a).

Surra is a multiple species OIE-listed disease (OIE 2009c).

Epidemiology

T. evansi infection is spread mechanically by infected blood on mouthparts of biting flies, especially tabanid flies (Hoare 1972). In South America, vampire bats are also a potential mechanical vector (Luckins 1994).

Flies of *Tabanus* spp. are more efficient at transmitting *T. evansi* than *Haematopota* spp., *Chrysops* spp. or biting flies from the family Muscidae (e.g. *Stomoxys calcitrans*) (Kettle 1995). Horse flies or March flies (family Tabanidae) and stable flies (family Muscidae) are widespread throughout Australia. They are most abundant in coastal regions, along inland river systems, and in timbered areas of valleys and mountain ranges (Seddon and Albiston 1967a; Seddon and Albiston 1967b; Mackerras 1971).

A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host or transmit infection to an uninfected susceptible animal (Luckins 1999). Tabanids are able to transmit infection for up to six hours after an infective feed

(Hoare 1972; Losos 1980; Luckins 1999). Tabanids are efficient vectors because they are obligate blood feeders, have large mouthparts to trap blood and a painful bite. The painful bite stimulates defensive behaviour in animals and re-initiation of feeding, potentially on another animal (Foil 1989). Tabanids have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000). Studies have shown that a greater proportion of tabanids return to feed on the original animal rather than move to another (Foil 1983; Barros and Foil 2007). In one study, 5% of recaptured tabanids fed on two different horses 51.7 metres apart during a single interrupted feed (Foil 1983). In a repeated study on interrupted feeding, no tabanids were recaptured feeding on a horse located 50 metres from the original host. However, in this study the majority of flies that left the original horse during interrupted feeding were not recaptured. A distance of 200 metres between infected and susceptible animals was recommended as an effective margin to minimise the risks of mechanical transmission by tabanids (Barros and Foil 2007).

Reservoirs of infection can occur in all susceptible animals (including equids) with mild or subclinical infection. Horses that appear to have recovered from acute disease and have developed chronic disease, or have subclinical infection are also potential reservoirs of infection (Utami 1996; Tinson et al. 1998; Abo-Shehada et al. 1999). Donkeys with subclinical infection or chronic disease can act as reservoirs of infection (Taylor and Authie 2004).

Outbreaks of surra occur when susceptible naïve animals are introduced to an endemic area, or when an infected host is in contact with vectors and reservoir hosts (Sellon 2007). Transmission and infection with *T. evansi* occurs more during warmer and wetter months when tabanid populations are highest (Hoare 1972; Foil and Hogsette 1994). In horses, there is no known correlation between prevalence and age, breed or gender (Sellon 2007). Animals are more susceptible to developing surra if under stress such as malnutrition, pregnancy, regular strenuous exercise or aircraft travel (Luckins 1988; OIE 2008a).

Levels of parasitaemias in horses have not been accurately determined nor quantified in either experimental or natural infections. Trypanosomes can be detected in blood (buffy coat), oedema fluid or lymph nodes or during the initial phase of infection; however, this becomes increasingly difficult during later phases. After 25 days post-infection, *T. evansi* trypanosomes localise in extravascular tissues, including the central nervous system and may be detected in cerebrospinal fluid (Horchner et al. 1983).

Several reports suggest that transplacental transmission of *T. evansi* occurred in cases of natural infection in buffalo (Rao et al. 2001), cattle (Rajguru et al. 2000) and donkeys (Pathak et al. 1999). Trypanosomes were recovered either from aborted foetuses or clinically affected neonates. Donkey and buffalo females were also clinically affected. There are no reports of transplacental transmission in horses, although abortions and stillbirths can occur in pregnant mares with surra (Tuntasuvan et al. 2003).

Host specificity and pathogenicity of *T. evansi* varies in different geographical regions (Taylor and Authie 2004; OIE 2008a). In the United Arab Emirates, the disease is chronic in camels and infection is often subclinical. During an investigation into outbreaks of clostridial enterotoxaemias in camels, 50% of breeding camels were found to have concurrent *T. evansi* infection (Wernery et al. 1991). The last outbreak

of surra in camels in the United Arab Emirates was in 2004. An incidence of less than 2% in camels was reported at the time (OIE 2008b). Surra has been a notifiable disease in the United Arab Emirates since 2007 (OIE 2009a).

The only reported case of surra in a horse in the United Arab Emirates was in 1996 in a 16 year-old Arabian stallion used for dressage and show jumping events. The horse developed chronic ataxia and tested positive to serological tests. No parasites were seen on microscopic examination of blood. The horse was stabled in the same complex as ten retired racing camels and one other 14 year-old stallion. The horse had been at that stable complex for five years, during which time all camels became infected with *T. evansi*. The other horse did not develop surra or show any signs of infection with *T. evansi*; however, it was observed that the affected horse was bitten significantly more by the stable fly *Stomoxys calcitrans* (the vector identified in this case) than the other horse (Tinson et al. 1998).

Clinical signs

The incubation period of surra in horses is usually 1–2 weeks, but can be up to 60 days (Losos 1980; Geering et al. 1995; Sellon 2007). Acute disease is most common in horses, although chronic disease and subclinical infection can occur (Utami 1996; Abo-Shehada et al. 1999; Sellon 2007). Clinical signs include intermittent pyrexia (coinciding with intermittent parasitaemia), progressive anaemia, weight loss despite good appetite, rough dry coat, ventral oedema with oedematous plaques on the ventral abdomen, ecchymotic haemorrhages in the eye, petechiae on mucous membranes, progressive muscular weakness and ataxia (mostly in hindlimbs), followed by death. Terminal central nervous system signs also occur, such as paraplegia, paralysis, delirium and convulsion (Gardiner and Mahmoud 1992; Luckins 1998; Radostits et al. 2007). Abortion or stillbirth can occur if mares are infected during pregnancy (Tuntasuvan et al. 2003). Mortality rates in horses are high when the disease is introduced into a naïve population (Sellon 2007).

In untreated horses, clinical disease lasts up to 90 days with death usually occurring within a few days to a few months. Death occurs in the majority of horses with surra that are not treated. Horses that are treated and recover from acute disease can develop chronic intermittent pyrexia, concurrent peaks of parasitaemia and ataxia due to meningioencephalitis (Hoare 1972; Gardiner and Mahmoud 1992; Tinson et al. 1998; Radostits et al. 2007; Sellon 2007).

In camels, subclinical infection or chronic disease occurs more commonly and can last for years (Radostits et al. 2007).

Diagnosis

Clinical signs of surra in horses are not pathognomonic (Gardiner and Mahmoud 1992). A definitive diagnosis requires laboratory methods to detect the parasite. There are no prescribed or alternative diagnostic techniques recommended by the OIE for surra (OIE 2009b). While the OIE describes direct and indirect tests (OIE 2008a), the ability of those tests to detect infection varies depending on the stage of infection. Several indirect techniques to detect *T. evansi* antigens or antibodies are available, but have variable sensitivity and specificity (Monzon et al. 1995; Luckins 1999; Wernery et al. 2001).

Horses with acute disease are most likely to have a high level of parasitaemia during periods of pyrexia. In these cases, direct parasitological tests are recommended to

confirm a clinical diagnosis. Typical trypanosomes can be observed by direct examination of the buffy coat, or in some cases oedema fluid or lymph nodes (Murray et al. 1977; Radostits et al. 2007; Sellon 2007; OIE 2008a). Experimentally, trypanosomes have been observed in the blood of horses 1–10 days post-infection and in donkeys four days post-infection (Horchner et al. 1983; Pathak et al. 1999; Wernery et al. 2001). Trypanosomes have been detected in cerebrospinal fluid 25–35 days post-infection (Horchner et al. 1983). The microhaematocrit centrifugation technique is considered to be the most reliable direct test (Murray et al. 1977; OIE 2008a). It is most consistent if used to detect the presence of trypanosomes from 10 days post-infection (Wernery et al. 2001).

Horses with chronic disease or subclinical infection are unlikely to be diagnosed using direct methods (Reid et al. 2001a). Examination of thick blood smears or buffy coat samples, in addition to parasite concentration and inoculation of laboratory animals, is more likely to detect infection. The mouse inoculation test using a buffy coat sample is able to detect 1.25 *T. evansi* trypanosomes per 4 ml of blood. It is the most sensitive parasitological method of diagnosis (Reid et al. 2001a). It is not practical for routine screening because results are not available for up to six weeks (Wernery et al. 2001).

The antigen-detection enzyme-linked immunosorbent assay has proved unreliable with conflicting reports on sensitivity and specificity (Monzon et al. 1995; Luckins 1999). Sensitivity is greatest in horses 7–21 days post-infection when *T. evansi* antigens can be detected in the serum (even when parasites are unable to be found). Antigen continues to be detectable until treatment is effective (Monzon et al. 1995). An antigen-detection latex agglutination test (Suratex®) has been adapted for the diagnosis of *T. evansi* infection and is used for camels (Nantulya 1994). There has been no rigorous validation of Suratex® for use in other species.

Serum antibodies are first detectable 10–19 days post infection (Wernery et al. 2001) and can persist for up to 22.6 months in horses that have been successfully treated for surra (Monzon et al. 2003). The antibody-detection enzyme-linked immunosorbent assay has been validated for use in horses (Monzon 2000). It detects IgG antibodies and is considered to have a higher sensitivity (95.5%) and specificity (98%) than other serological tests (Davison et al. 1999; Monzon 2000; OIE 2008a).

Polymerase chain reaction amplification tests have been described for detecting *T. evansi* DNA. The sensitivity and specificity are greater than other tests; however, they have not been validated for use in horses and are considered too expensive for routine use (Wuyts et al. 1995; Sellon 2007; OIE 2008a).

Treatment

Five drugs (suramin, quinapyramine sulphate, diminazene aceturate, isometamidium and cymelarsan) have been used for the treatment and/or prevention of surra. Although used widely, there have been conflicting reports on efficacy and curative doses.

Treatment is most effective during early stages of infection. Persistent infection can occur in animals treated during later stages of infection (Luckins 1994; Wernery et al. 2001). This is most likely due to trypanosomes localising in extravascular tissues, including the cerebrospinal fluid, and evading exposure to trypanocidal drugs. Evasion of treatment by trypanosomes has also contributed to the development of drug resistance (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).

Adverse reactions to treatment vary from moderate to severe, and can occur in up to 50% of horses and mules (Tuntasuvan et al. 2003; Radostits et al. 2007). Diminazene aceturate has been reported to cause lip oedema, salivation, recumbency, restlessness and dyspnoea (Tuntasuvan et al. 2003). Quinapyramine sulphate causes adverse local reactions at the site of injection (Luckins 1994).

Existence of several antigenic types has restricted the development of a vaccine for surra. However, experimental studies on laboratory animals have developed a protective immunogen from *T. evansi* that may lead to a potential vaccine (Li et al. 2007).

Conclusion

Surra is present in some approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.33.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

This risk assessment is based on the assumption that a response consistent with AUSVETPLAN Disease Strategy Manual for Surra (Animal Health Australia 2005) would be implemented.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of surra being present in an imported horse.

- *T. evansi* has a wide distribution in the Middle East and is endemic in the camel population of the United Arab Emirates.
- Prevalence of 50% was reported in camels in the United Arab Emirates in 1991 (Wernery et al. 1991). In 2004, the disease incidence in camels was reported to be less than 2% (OIE 2008b).
- The only report of disease in a horse in the United Arab Emirates was in 1996. The horse was stabled in the same complex as camels (Tinson et al. 1998).
- Horses with surra may show no clinical signs of infection or may be latent carriers (Utami 1996; Abo-Shehada et al. 1999). There is no known correlation between prevalence and age, breed or gender (Sellon 2007).
- The incubation period of surra in horses is generally 1–2 weeks but can be up to 60 days (Geering et al. 1995; Losos 1980; Sellon 2007).
- Persistent infection can occur in horses post-treatment due to trypanosomes localising in extravascular tissues and evading exposure to the trypanocidal drug (Luckins 1994; Radostits et al. 2007).
- Transmission and infection with *T. evansi* occurs seasonally during the warmer and wetter months when tabanid populations are highest (Hoare 1972; Foil and Hogsette 1994).

Based on these considerations, the likelihood of release of surra associated with horses from a country where the disease is present was estimated to be '*low*'.

Exposure assessment

The most likely pathway for exposure is importation of an infected horse into a region where biting flies are endemic and able to feed on the horse and transmit infection to other exposure groups.

Exposure groups are equids (including feral equids), other domestic species (including other non-ruminants, ruminants and feral animals) and wildlife.

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to surra via an infected imported horse.

- *T. evansi* has a wide mammalian host range (Radostits et al. 2007; Sellon 2007). In horses, there is no known correlation between prevalence and age, breed or gender (Sellon 2007).
- Transmission to susceptible animals occurs via biting flies, especially tabanids. Horse flies or March flies (family Tabanidae) and stable flies (family Muscidae) are widespread throughout Australia. They are most abundant in coastal regions, along inland river systems, and in timbered areas of valleys and mountain ranges (Seddon and Albiston 1967a; Seddon and Albiston 1967b; Mackerras 1971).
- A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids are able to transmit infection for up to six hours after an infective feed (Hoare 1972; Losos 1980; Luckins 1999).
- Intermittent parasitaemia occurs in infected animals, including animals with chronic disease or subclinical infection (Radostits et al. 2007). Trypanosomes localise in extravascular tissues after approximately five weeks post-infection (Horchner et al. 1983).

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with *T. evansi* was estimated to be '*moderate*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure involves consideration of the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*low*' combined with the likelihood of exposure estimated to be '*moderate*', the likelihood of release and exposure for surra was estimated to be '***low***'.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of a susceptible animal has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to surra is considered to be establishment and/or spread to populations of susceptible animals to more than one state through biting flies and the movement of infected horses.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to *T. evansi*.

- Susceptible animals include equids (including feral equids), other domestic species (including other non-ruminants, ruminants and feral animals) and wildlife. The disease is usually acute in dogs and horses (Losos 1980). In horses, there is no known correlation between prevalence and age, breed or gender (Sellon 2007).
- Horse flies or March flies (family Tabanidae) and stable flies (family Muscidae) are widespread throughout Australia. They are most abundant in coastal regions, along inland river systems, and in timbered areas of valleys and mountain ranges (Seddon and Albiston 1967a; Seddon and Albiston 1967b; Mackerras 1971).
- A feeding time of five seconds is sufficient for a fly to acquire or transmit infection (Luckins 1999). After feeding on an infected horse, tabanids are able to transmit infection to other susceptible animals for up to six hours after an infective feed (Hoare 1972; Losos 1980; Luckins 1999). Tabanids have been reported to feed on two different horses 51.7 m apart during a single interrupted feed (Foil 1983).
- Incubation period is generally 1–2 weeks (Sellon 2007) but can be up to 60 days (Losos 1980; Geering et al. 1995), clinical signs of disease are not pathognomonic and subclinical infection occurs in susceptible animals (Utami 1996; Abo-Shehada et al. 1999; Sellon 2007); therefore detection of infection is likely to be delayed.
- Movement of incubating or subclinically infected animals and mechanical vectors could spread the disease before detection.
- Given the widespread movement of susceptible animals within and between states, infection is most likely to spread to more than one state before detection.
- Movement of infected animals will contribute to the spread of infection. Infection in wild and feral animals would contribute to its uncontrolled and potentially undetected spread.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of surra was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of surra for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Infection with *T. evansi* causes acute and chronic disease, loss of productivity and possibly death in a wide range of susceptible animals including cattle, goats, sheep, water buffalo, camels, llamas, cats, dogs, donkeys, horses, mules, pigs and elephants. Naïve animals are more likely to develop acute disease (Sellon 2007).
- Acute fatal disease occurs most commonly in dogs, donkeys, horses, and water buffalo (Losos 1980; Taylor and Authie 2004).
- Death occurs in the majority of horses with surra that do not receive treatment (Radostits et al. 2007). There is no known correlation between prevalence and age, breed or gender (Sellon 2007).
- Adverse reactions to treatment can occur in up to 50% of horses and mules, and vary from moderate to severe (Tuntasuvan et al. 2003; Radostits et al. 2007). Clinical signs range from local reactions at the site of injection to systemic reactions including lip oedema, salivation, recumbency, restlessness and dyspnoea (Luckins 1994; Tuntasuvan et al. 2003).
- Persistent infection can occur following treatment, either due to drug resistance or trypanosomes evading exposure to trypanocidal drugs by localising in extravascular tissues (Luckins 1994; Radostits et al. 2007). This results in chronic intermittent pyrexia, concurrent peaks of parasitaemia and ataxia (Tinson et al. 1998; Radostits et al. 2007; Sellon 2007).

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- Wallabies have been shown experimentally to be susceptible to infection and develop acute fatal disease (Reid et al. 2001b). As *T. evansi* has a wide mammalian host range, other Australian wildlife species are potentially susceptible to infection and fatal disease.
- Surra is not considered to have any direct effects on the non-living environment.

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the state level. The effect

on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

Indirect effects

The effects on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- Surra is nationally notifiable in Australia (DAFF 2008).
- If surra were to be identified in Australia, the current policy as outlined in AUSVETPLAN Disease Strategy Manual for surra is to eradicate where practicable. If eradication is not a viable option, a control program would be established to slow the spread of disease and reduce the effect on trade (Animal Health Australia 2005).
- Surra is scheduled as Category 4 under Australia's Emergency Animal Disease Response Agreement (EADRA) for cost-sharing arrangements (Animal Health Australia 2001). Should it be activated, EADRA states that costs of a response would be covered by government and relevant industries by contributions of 20% and 80%, respectively (Animal Health Australia 2001). However, currently the horse industry is not a signatory to this Agreement. Other animal industries, such as those associated with cattle, are signatories to the agreement.
- Eradication or control measures would include quarantine and movement controls; treatment or selective destruction and disposal of affected animals; tracing, surveillance and zoning; control of biting flies and measures to prevent flies from biting susceptible animals; and a public awareness campaign to facilitate cooperation from industry and the community (Animal Health Australia 2005).
- Control measures for surra could result in widespread use of trypanocidal drugs and the development of drug resistance (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007). Increased use of insecticides could contribute to the development of insecticidal resistance.
- Control measures would affect all industry sectors associated with susceptible animals, including cattle, goats, sheep, water buffalo, camels, llamas, cats, dogs, donkeys, horses, mules, pigs, elephants, wallabies and potentially other native wildlife species.
- If surra were to become established, periodic outbreaks would occur and ongoing control programs, including zoning, may be required (Animal Health Australia 2005).

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- Surra is nationally notifiable in Australia. If it were detected in Australia, the policy as outlined in AUSVETPLAN Disease Strategy Manual for surra (Animal Health Australia 2005) is eradication where practicable, recognising that in some

species the disease is insidious and that at the point of detection, infection may be well established, both spatially and in a variety of wild and domestic animal species.

- Domestic trade or industry sectors associated directly or indirectly with all susceptible animals would experience productivity losses, disruption to sales (including supply and demand for animals, animal feed and agricultural products), increased costs and operational procedures associated with managing disease outbreaks and implementing eradication or control measures.
- Quarantine and movement controls would be imposed on susceptible species. Movements of animals to sale and slaughter would be affected. Horse, dog and livestock events and would be disrupted.
- Following detection of surra in one state or territory in Australia, other states may close their borders to susceptible animals and products until the extent of the outbreak was ascertained.
- If surra became established and a zoning program was implemented, there would be domestic market access losses for affected animal industries located within infected zones.

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the regional level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the state level* (national effect score D in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- If surra were detected in Australia, there would be immediate disruption to exports of susceptible animals e.g. horses, cattle, sheep, cats and dogs (Animal Health Australia 2005).
- If surra could not be eradicated, new technical requirements such as zoning may be necessary to maintain export market access for affected industry sectors (Animal Health Australia 2005). Loss of export markets would be ongoing for affected animal industries located within infected zones.
- If zoning was not feasible, losses in export market access would apply to all affected animal industries in Australia.

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- Wallabies have been shown experimentally to be susceptible to infection and develop acute fatal disease (Reid et al. 2001b), therefore threatened and endangered wildlife are potentially susceptible to infection and fatal disease.

- Increased use of insecticides to control biting flies could have an effect on a range of insect species and disrupt the food source of wildlife, lead to environmental contamination (including water sources) and resistance to insecticides.
- Widespread use trypanocides could lead to drug resistance (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Disruption of horse, dog and livestock events would have social and economic consequences for local communities. Horse and greyhound racing contribute significantly to government revenue.
- Where susceptible animals are important to the local economy, there would be reduced viability of communities within affected regions.
- There could be losses to tourism industries associated with susceptible animals and wildlife, including zoos.

Based on these considerations, the effect of the establishment and/or spread of surra in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is 'E', the overall effect associated with the outbreak scenario was considered to be '*moderate*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*moderate*') which results in '***moderate***' likely consequences.

Risk estimation

Risk estimation is the integration of likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of surra introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure (‘***low***’) is combined with the likely consequences of establishment and/or spread (‘***moderate***’) which results in a risk estimation of **LOW**.

Conclusion

The unrestricted risk associated with surra is determined to be **LOW**. The unrestricted risk estimate exceeds Australia’s ALOP and, therefore, risk management is deemed necessary.

A summary of the risk assessment for surra is shown in Figure 5.7 and Table 5.7

Figure 5.7 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for surra.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Low*
Likelihood of *exposure*	Likelihood of *exposure*	*Moderate*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**LOW**

Table 5.7 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for surra.

References

Abo-Shehada MN, Anshassi H, Mustafa G, Amr Z (1999) Prevalence of surra among camels and horses in Jordan. *Preventive Veterinary Medicine* 38: 289-293.

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

Animal Health Australia (2005) *Disease strategy: surra (version 3.0)*. Primary Industries Ministerial Council, Canberra.

Barros ATM, Foil LD (2007) The influence of distance on movement of tabanids (Diptera: Tabanidae) between horses. *Veterinary Parasitology* 144: 380-384.

Boid R, Hunter AG, Jones TW, Ross CA, Sutherland D, Luckins AG (1996) Trypanosomosis research at the Centre for Tropical Veterinary Medicine (CTVM) 1970 to 1995. *Tropical Animal Health and Production* 28: 5-22.

Brun R, Hecker H, Lun ZR (1998) *Trypanosoma evansi* and *T. equiperdum*: distribution, biology, treatment and phylogenetic relationship (a review). *Veterinary Parasitology* 79: 95-107.

Chippaux J-P, Bouchite B, Demanou M, Morlais I, Le Goff G (2000) Density and dispersal of the loaiasis vector *Chrysops dimidiata* in southern Cameroon. *Medical and Veterinary Entomology* 14: 339-344.

Claes F (2003) *Characterization of pathogenic Tryanosoma spp.: how does Trypanosoma equiperdum fit into the Trypanozoon group?* Katholieke Universiteit Leuven, Leuven.

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

Davison HC, Thrusfield MV, Muharsini S, Husein A, Partoutomo S, Rae PF, Masake R, Luckins AG (1999) Evaluation of antigen detection and antibody detection tests for *Trypanosoma evansi* infections of buffaloes in Indonesia. *Epidemiology and Infection* 123: 149-155.

Foil L (1983) A mark-recapture method for measuring effects of spatial separation of horses on tabanid (Diptera) movement between hosts. *Journal of Medical Entomology* 20: 301-305.

Foil LD (1989) Tabanids as vectors of disease agents. *Parasitology Today* 5: 88-96.

Foil LD, Hogsette JA (1994) Biology and control of tabanids, stable flies and horn flies. *Revue Scientifique et Technique de l'Office International des Epizooties* 13: 1125-1128.

Gardiner PR, Mahmoud MM (1992) Salivarian trypanosomes causing disease in livestock outside sub-Saharan Africa. In *Parasitic protozoa* (eds. Kreier JP, Baker JR) pp. 277-314. Academic Press, San Diego.

Geering WA, Forman AJ, Nunn MJ (1995) Surra. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 380-384. Australian Government Publishing Service, Canberra.

Gillingwater K, Buscher P, Brun R (2007) Establishment of a panel of reference *Trypanosoma evansi* and *Trypanosoma equiperdum* strains for drug screening. *Veterinary Parasitology* 148: 114-121.

Haag J, O'huigin C, Overath P (1998) The molecular phylogeny of trypanosomes: evidence for an early divergence of the Salivaria. *Molecular and Biochemical Parasitology* 91: 37-49.

Hoare CA (1972) Subgenus *Trypanozoon*. In *The trypanosomes of mammals: a zoological monograph* pp. 554-604. Blackwell Scientific Publications, Oxford.

Horchner F, Schonefeld A, Wust B (1983) Experimental infection of horses with *Trypanosoma evansi*: I. parasitological and clinical results. *Annales de la Société Belge de Médecine Tropicale* 63: 127-135.

Kettle DS (1995) Tabanidae (horseflies, deer flies, clegs). In *Medical and veterinary entomology* (ed. Kettle DS) pp. 211-224. CAB International, Wallingford.

Li S-Q, Fung M-C, Reid SA, Inoue N, Lun Z-R (2007) Immunization with recombinant beta-tubulin from *Trypanosoma evansi* induced protection against *T. evansi*, *T. equiperdum* and *T. b. brucei* infection in mice. *Parasite Immunology* 29: 191-199.

Losos GJ (1980) Diseases caused by *Trypanosoma evansi*: a review. *Veterinary Research Communications* 4: 165-181.

Luckins AG (1988) *Trypanosoma evansi* in Asia. *Parasitology Today* 4: 137-142.

Luckins AG (1994) Equine trypanosomiasis. *Equine Veterinary Education* 6: 259-262.

Luckins AG (1998) Epidemiology of surra: unanswered questions. *Journal of Protozoology Research* 8: 106-119.

Luckins AG (1999) Equine trypanosomiases: a review. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden O.R.) pp. 237-242, R & W Publications (Newmarket) Limited, Newmarket.

Mackerras IM (1971) The Tabanidae (Diptera) of Australia: V. subfamily Tabaninae, tribe Tabanini. *Australian Journal of Zoology* Suppl. 4: 1-54.

Monzon CM (2000) Validation of an indirect immunoenzyme assay for the detection of antibodies against *Trypanosoma evansi* in horses in Argentina. *Revue Scientifique et Technique de l'Office International des Epizooties* 19: 810-818.

Monzon CM, Jara A, Nantulya VM (1995) Sensitivity of antigen ELISA test for detecting *Trypanosoma evansi* antigen in horses in subtropical area of Argentina. *The Journal of Parasitology* 81: 806-808.

Monzon CM, Mancebo OA, Russo AM (2003) Antibody levels by indirect ELISA test in *Trypanosoma evansi* infected horses following treatment with quinapyramine sulphate. *Veterinary Parasitology* 1111: 59-63.

Monzón CM, Russo AM (1997) Epidemiological review of equine trypanosomosis in Argentina. Proceedings of the first internet conference on Salivarian trypanosomes. http://www.fao.org/docrep/W5781E/w5781e03.htm#epidemiological%20review%20of%20equine%20trypanosomosis%20in%20argentina (Accessed 24 December 2003).

Murray M, Murray PK, McIntyre WIM (1977) An improved parasitological technique for the diagnosis of African trypanosomiasis. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 71: 325-326.

Nantulya VM (1994) Suratex®: a simple latex agglutination antigen test for diagnosis of *Trypanosoma evansi* infections (surra). *Tropical Medicine and Parasitology* 45: 9-12.

OIE (2008a) *Trypanosoma evansi* infections (including surra). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.17_TRYPANO.pdf (Accessed 1 September 2009).

OIE (2008b) United Arab Emirates / 2004: annual animal disease status. Handistatus II: prototype. http://www.oie.int/hs2/zi_pays.asp?c_pays=5 (Accessed 5 June 2009).

OIE (2009a) Animal health situation: United Arab Emirates. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=country_status&year=2008 (Accessed 29 May 2009).

OIE (2009b) List of tests for international trade. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/0.02_PRESCRIBED_TESTS_2008.pdf (Accessed 3 September 2009).

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pathak KML, Bhatnagar CS, Yadav MP (1999) Diagnostic techniques in donkeys infected with *Trypanosoma evansi*. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden O.R.) pp. 315-317, R & W Publications (Newmarket) Limited, Newmarket.

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1483-1540. Saunders Elsevier, Edinburgh.

Rajguru DN, Ali MS, Joshi SA, Swami SB, Saleem M (2000) Observations on *Trypanosoma evansi* infection in neonatal calves. *Indian Veterinary Journal* 77: 996-997.

Rao PPR, Devi VR, Srilatha C, Kavitha K (2001) Transplacental transmission of trypanosomes in a buffalo calf. *Indian Veterinary Journal* 78: 849-850.

Reid SA, Husein A, Copeman DB (2001a) Evaluation and improvement of parasitological tests for *Trypanosoma evansi* infection. *Veterinary Parasitology* 102: 291-297.

Reid SA, Husein A, Partoutomo S, Copeman DB (2001b) The susceptibility of two species of wallaby to infection with *Trypanosoma evansi*. *Australian Veterinary Journal* 79: 285-288.

Seddon HR, Albiston HE (1967a) Stable fly. In *Diseases of domestic animals in Australia: part 2. arthropod infestations (flies, lice and fleas)* pp. 10-11. Department of Health, Canberra.

Seddon HR, Albiston HE (1967b) Tabanid (March-) flies. In *Diseases of domestic animals in Australia: part 2. arthropod infestations (flies, lice and fleas)* p. 33. Department of Health, Canberra.

Sellon DC (2007) Miscellaneous parasitic diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 473-480. Saunders Elsevier, St. Louis.

Sheppard DC, Wilson B, Hawkins JA (1973) A device for self-marking of Tabanidae. *Environmental Entomology* 2: 960-961.

Stevens JR, Brisse S (2004) Systematics of trypanosomes of medical and veterinary importance. In *The trypanosomiases* (eds. Maudlin I, Holmes PH, Miles MA) pp. 1-23. CABI Publishing, Oxfordshire.

Taylor K, Authie M-L (2004) Pathogenesis of animal trypanosomiasis. In *The trypanosomiases* (eds. Maudlin I, Holmes PH, Miles MA) pp. 331-353. CABI Publishing, Oxfordshire.

Tinson A, Gorde A, Herlekar S (1998) Clinical surra in a UAE Arabian horse. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 1-7, R & W Publications (Newmarket) Limited, Newmarket.

Tuntasuvan D, Jarabrum W, Viseshakul N, Mohkaew K, Borisutsuwan S, Theeraphan A, Kongkanjana N (2003) Chemotherapy of surra in horses and mules with diminazene aceturate. *Veterinary Parasitology* 110: 227-233.

Utami S (1996) Survey of trypanosome species in cattle, buffaloes and horses in three districts of Lombok. In *Proceedings of a seminar in diagnostic techniques for Trypanasoma evansi in Indonesia,* pp. 54-57, Research Institute for Veterinary Science, Bogor.

Wernery U, Seifert HS, Billah AM, Ali M (1991) Predisposing factors in enterotoxemias of camels (*Camelus dromedarius*) caused by *Clostridium perfringens* type A. *Revue d'Élevage et de Medecine Véterinaire des Pays Tropicaux* 44: 147-152.

Wernery U, Zachariah R, Mumford JA, Luckins T (2001) Preliminary evaluation of diagnostic tests using horses experimentally infected with *Trypanosoma evansi*. *The Veterinary Journal* 161: 287-300.

Wuyts N, Chokesajjawatee N, Sarataphan N, Panyim S (1995) PCR amplification of crude blood on microscope slides in the diagnosis of *Trypanosoma evansi* infection in dairy cattle. *Annales de la Société Belge de Médecine Tropicale* 75: 229-237.

5.34 Taylorella asinigenitalis

5.34.1 Technical information

Background

Taylorella asinigenitalis was identified in 1997 as a second species within the genus *Taylorella* after it was isolated from the genital tracts of donkeys in the United States (Jang et al. 2001).

Previously, only *T. equigenitalis,* the causative agent of contagious equine metritis (CEM), was identified. Colony morphology and growth rates of these species were too similar to be differentiated by standard bacteriological isolation so species typing was required (Duquesne et al. 2007). The isolation and identification of *T. asinigenitalis* from a stallion in Sweden in 2005, the first natural case of infection in a horse, highlighted the need for identification at a species level (Baverud et al. 2006).

Subsequently, an isolate was reported in a horse in Spain in 2006, and 24 isolates from horses were reported in France between 1999 and 2005 (Roest 2007). None of these isolates have been associated with clinical disease in horses. It has not been reported in Australia.

T. asinigenitalis is not an OIE-listed disease (OIE 2009).

Epidemiology

The primary form of disease transmission is thought to be similar to CEM, via natural service or artificial insemination (Timoney 1996). Infection could occur through contaminated semen, and through mechanical spread of disease by contaminated equipment or poor breeding practices. Stallions and carrier mares, should they exist, may act as reservoirs of *T. asinigenitalis*. Stallions, because they inseminate numerous mares, could play a more important role in transmission.

As *T. asinigenitalis* is considered to be a contagious venereal disease of equids, in horses not used for breeding, or where there is no opportunity to mate, the risk of disease transmission would be minimal.

Stallions and potentially clinically recovered mares may harbour *T. asinigenitalis* for extended periods — possibly for years after initial infection — whether or not clinical signs of disease, or reduced fertility, are apparent.

Vertical spread of *T. asinigenitalis* has not been reported; however, it is likely that it would be similar to *T. equigenitalis*. If there was disease in foals it is likely to be acquired either *in utero* or at the time of parturition (Timoney and Powell 1982).

Clinical signs

T. asinigenitalis is widely regarded as non-pathogenic. There is only a single study in which clinical disease was experimentally produced with a mare developing mild endometritis (Katz et al. 2000). It is highly unlikely that *T. asinigenitalis* would cause infertility in mares.

Diagnosis

Diagnosis of *T. asinigenitalis* is principally confirmed by culture of the organism from a series of urogenital swabs of mares or stallions.

In contrast to *T. equigenitalis*, *T. asinigenitalis* has been isolated more frequently in the proximal, rather than the distal, reproductive tract in mares (Timoney pers. comm. 2007).

Stallions do not show serological evidence of infection (Timoney 1996). However, complement fixing antibody has been demonstrated in naturally infected donkey jacks infected with *T. asinigenitalis* (Jang et al. 2001; Roest 2007), suggesting a humoral response is present in some male equids.

Conclusion

T. asinigenitalis is present in some approved countries and there are no recommendations in the Code. A risk assessment was undertaken.

5.34.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of *T. asinigenitalis* being present in an imported horse.

- There have been no reported clinical cases of disease in horses infected naturally with *T. asinigenitalis*.
- Breeding stallions are the most likely source of infection.
- False negative test results can occur.

Based on these considerations, the likelihood of release of *T. asinigenitalis* associated with horses from a country where the disease is present was estimated to be '*very low*'.

Exposure assessment

The single most likely pathway is direct transmission through coitus.

The exposure group considered is equids (including feral equids).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to *T. asinigenitalis* via an imported horse.

- The disease is a contagious venereal disease and transmitted during mating.
- Subclinical or latent carrier stallions, and possibly mares, may harbour the pathogen without detection.
- Imported infected horses could remain infectious for long periods, increasing the likelihood of contact with other horses to transmit the pathogen.

Based on these considerations, the likelihood of susceptible animals being exposed to *T. asinigenitalis* via an infected imported horse was estimated to be '*high*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*very low*' combined with the likelihood of exposure estimated to be '*high*', the likelihood of release and exposure for *T. asinigenitalis* was estimated to be ***'very low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to *T. asinigenitalis* is considered to be establishment and/or spread to populations of susceptible animals within multiple states/territories through transmission by coitus.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to *T. asinigenitalis*.

- Disease transmission from subclinical or latent carrier stallions to susceptible horses could readily occur.
- Subclinical or latent carrier stallions could infect a number of susceptible mares before the disease would be detected.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of *T. asinigenitalis* was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of *T. asinigenitalis* for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- Stallions are subclinically infected or latent carriers for *T. asinigenitalis*; therefore there are no health implications for these animals.
- Only experimental infection with *T. asinigenitalis* has been reported to cause clinical disease in one study (Katz et al. 2000) in which a mare developed mild endometritis.

Based on these considerations, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- There are no reported effects of *T. asinigenitalis* on the living or non-living environment.

Based on this consideration, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- *T. asinigenitalis* is not a notifiable disease in Australia, there is no AUSVETPLAN Disease Strategy Manual for *T. asinigenitalis* and the disease is not scheduled in Australia's Emergency Animal Disease Response Agreement.
- If *T. asinigenitalis* was introduced into Australia it would be unlikely that eradication and control would be implemented.
- If *T. asinigentitalis* established in Australia it would require additional testing of horses to differentiate from *T. equigenitalis*.

Based on these considerations, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- Horse industries would not be greatly affected as *T. asinigenitalis* is considered to be much less pathogenic than *T. equigenitalis.*
- The effect on fertility of a mare should it get infected, is likely to be minimal. Therefore the effect on breeding, related industries and domestic trade in horses would be minimal.

Based on these considerations, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *minor at the local level* (national effect score B in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- Due to its similarity to CEM there could be a temporary loss of access to export markets and reduced consumer demand.
- An outbreak or the establishment of *T. asinigenitalis* would result in increased export costs due to additional veterinary and testing fees to differentiate it from *T. equigenitalis*.

Based on these considerations, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- There are no reported indirect effects of *T. asinigenitalis* on the environment.

Based on this consideration, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- There could be some very small reductions in rural and regional economic viability and associated industries; however, this would most likely be short term.

Based on this consideration, the effect of the establishment and/or spread of *T. asinigenitalis* in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is 'C', the overall effect associated with the outbreak scenario is considered to be '*very low*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*very low*') which results in ***'very low***' likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with

release, exposure, establishment and/or spread of *T. asinigenitalis* introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure (‘***very low’***) is combined with the likely consequences of establishment and/or spread (‘***very low***’), resulting in a risk estimation of **NEGLIGIBLE**.

Conclusion

The unrestricted risk associated with *T. asinigenitalis* is determined to be **NEGLIGIBLE**. As the unrestricted risk estimate achieves Australia’s ALOP, no risk management is considered necessary.

A summary of the risk assessment for *T. asinigenitalis* is shown in Figure 5.8 and Table 5.8.

Figure 5.8 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for *T. asinigenitalis*.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Very low*
Likelihood of *exposure*	Likelihood of *exposure*	*High*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Very low*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Very low***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**NEGLIGIBLE**

Table 5.8 Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for *T. asinigenitalis*.

References

Baverud V, Nystrom C, Johansson KE (2006) Isolation and identification of *Taylorella asinigenitalis* from the genital tract of a stallion, first case of a natural infection. *Veterinary Microbiology* 116: 294-300.

Duquesne F, Pronost S, Laugier C, Petry S (2007) Identification of *Taylorella equigenitalis* responsible for contagious equine metritis in equine genital swabs by direct polymerase chain reaction. *Research in Veterinary Science* 82: 47-49.

Jang SS, Donahue JM, Arata AB, Goris J, Hansen LM, Earley DL, Vandamme PAR, Timoney PJ, Hirsh DC (2001) *Taylorella asinigenitalis* sp. nov., a bacterium isolated from the genital tract of male donkeys (*Equus asinus*). *International Journal of Systematic and Evolutionary Microbiology* 51: 971-976.

Katz JM, Evans LE, Hutto DL, Schroeder-Tucker LC, Carew AM, Donahue JM, Hirsh DC (2000) Clincal, bacteriologic, serologic, and pathologic features of infections with atypical *Taylorella equigenitalis* in mares. *Journal of the American Veterinary Medical Association* 216: 1945-1948.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Roest HJ (2007) *Taylorella asinigenitalis*: what approach should governments take? In *First international conference on contagious equine metritis, 11-12 July, Lelystad, The Netherlands,* pp. 1-15, Central Institute for Animal Disease Control, The Netherlands.

Timoney PJ (1996) Contagious equine metritis. *Comparative Immunology, Microbiology and Infectious Diseases* 19: 199-204.

Timoney PJ, Powell DG (1982) Isolation of the contagious equine metritis organism from colts and fillies in the United Kingdom and Ireland. *The Veterinary Record* 111: 478-482.

5.35 Trichinellosis

5.35.1 Technical information

Background

Trichinellosis is a serious zoonotic disease caused by ingestion of raw or undercooked meat containing larvae of *Trichinella* spp., family Trichinellidae (Dupouy-Camet 2006). Primarily an infection of pigs, *Trichinella* spp. are maintained in a sylvatic cycle among carnivores with cannibalistic and scavenger behaviour (Pozio 2001) and infect a wide range of mammals and birds (Soulsby 1982). *Trichinella* spp. in animals, and trichinellosis in humans have been reported in Africa, the Americas, Asia, Europe, New Zealand and Oceania. Only in France and Italy has human trichinellosis been attributed to the consumption of infected horse meat (Pozio et al. 2001; Pozio 2001).

There are seven species and three genotypes of *Trichinella* (Murrell et al. 2000), three of which have been identified in horses (Pozio 2001), these are: *T. spiralis*, *T. britovi* and *T. murrelli*. In the European Union, horse meat infected with *T. spiralis* has affected over 3000 humans in 13 outbreaks of trichinellosis. (Boireau et al. 2000). *T. britovi* and *T. murrelli*, both parasites of sylvatic carnivores, have also been identified in patients who acquired trichinellosis in outbreaks associated with horse meat (Pozio et al. 2001). *T. pseudospiralis* has been reported in birds and native animals in Tasmania (Obendorf et al. 1990; Obendorf and Clarke 1992). Domestic pork, wild boar, horse and crocodile meat exported from Australia is tested using the digestion method (Gamble et al. 2000) and *T. spiralis* has never been detected (P. Vanderlinde, Australian Quarantine and Inspection Service, pers. comm. March 2009).

Trichinellosis is a notifiable disease in Australia and control measures include general surveillance and precautions at the border (OIE 2009a).

Trichinellosis (*T. spiralis*) is a multiple species OIE-listed disease (OIE 2009b).

Epidemiology

The life cycle of *Trichinella* spp. is initiated when larvae encysted in muscles are ingested. In the intestine of the host, larvae develop to adult stage and mate. Over several weeks, the female hatches the eggs and the newborn larvae enter the bloodstream of the host via the lymphatics. Larvae are distributed throughout the body, localising primarily in the diaphragm, tongue, larynx, eye, masticatory and intercostal muscles, where they become encapsulated and survive many years until ingested by another host (Soulsby 1982). The highest muscle burden in naturally infected horses has been found in tongue, cheek and masseter muscles (Pozio et al. 1998).

There is no evidence of *Trichinella* transmission between horses. Infection is most likely to occur via ingestion of feed or pastures contaminated with infected rodent carcasses or pork scraps; or via ingestion of infected flesh from pigs and wild carnivores. Evidence for the latter is supported by the practice of using carnivore carcasses to fatten horses before slaughter and by the identification in horse meat of

T. britovi and *T. murrelli* larvae — generally present in sylvatic carnivores (Pozio et al. 2001).

The horse is considered an aberrant host for *T. spiralis.* Transmission to humans of *T. spiralis* is via ingestion of infected raw, or undercooked, horse meat (Murrell et al. 2004). Epidemiological investigations of human outbreaks have shown that they occurred because of inadequate veterinary controls at the slaughterhouse. Occurrence of trichinellosis in humans is related to cultural food practices, including the consumption of raw or undercooked meat. The presence of the parasite in domestic or wild animals is not sufficient for infection to occur in humans (Pozio 2007). Worldwide, the prevalence of horse infection with *Trichinella* spp. is low with only 27 infections reported since 1975, 23 of which were in France and Italy and four in Mexico (Pozio et al. 2001; Pozio 2001).

Clinical signs

Infection in horses has not been associated with clinical signs of disease (Hill et al. 2007).

Diagnosis

The only reliable and recommended technique for detecting *Trichinella* spp. is digestion assays on meat (OIE 2008). There is no method for detecting infection in live horses (Pozio 2001).

Conclusion

Trichinellosis is present in approved countries. The Code has recommendations for fresh meat from pigs and horses. There are no recommendations in the Code for live animals other than pigs. A risk assessment was undertaken.

5.35.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of trichinellosis being present in an imported horse.

- The most likely route of infection in horses is via ingestion of feed or pastures contaminated with infected carcasses or flesh (Pozio et al. 2001).
- Worldwide, the prevalence of horse infection with *Trichinella* spp. is very low with only 27 infections reported since 1975 (Pozio 2001).

Based on these considerations, the likelihood of release of trichinellosis associated with horses from a country where the disease is present was estimated to be *'extremely low'*.

Exposure assessment

The most likely pathway for exposure is if an infected imported horse died or was slaughtered, and raw or poorly cooked meat from the carcass was consumed by humans or animals.

Exposure groups considered are equids (including feral equids) and domestic and wild carnivores and omnivores (including cats, dogs, dingoes, foxes, pigs, rodents and carnivorous wildlife).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to *Trichinella* spp. via an imported horse.

- Transmission of *Trichinella* spp. occurs when meat containing encysted larvae is ingested (Murrell et al. 1987; Murrell et al. 2004).
- There are no methods for detecting infection in live horses.

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with trichinellosis was estimated to be '*extremely low*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure involves consideration of the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*extremely low*' combined with the likelihood of exposure estimated to be '*extremely low*', the likelihood of release and exposure for trichinellosis is estimated to be ***'negligible'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to trichinellosis is considered to be no further establishment and/or spread to populations of susceptible animals.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to *Trichinella* spp.

- There is no evidence of *Trichinella* spp. transmission between horses.
- The most likely route of infection of exposure groups is if infected horse meat was consumed by feral pigs, rodents or wild carnivores. Infection could then spread from the sylvatic cycle to domestic pigs, especially domestic pigs kept under free range and backyard conditions (Pozio et al. 2001).
- Burial or cremation are the most frequent methods of carcass disposal in Australia and it is unlikely that an infected horse carcass would be consumed by carnivores.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of *Trichinella* spp. was estimated to be *'extremely low'*.

Risk estimation

If the risk assessment proceeded, no combination of any likelihoods or consequences would give a risk estimation above **NEGLIGIBLE**. The risk assessment therefore concluded at this point.

Conclusion

The overall risk associated with trichinellosis is determined to be **NEGLIGIBLE**. As the unrestricted risk estimate achieves Australia's ALOP, no risk management was considered necessary.

A summary of the risk assessment for trichinellosis is shown in Figure 5.9 and Table 5.9.

Figure 5.9 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for trichinellosis.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Extremely low*
Likelihood of *exposure*	Likelihood of *exposure*	*Extremely low*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Negligible***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Extremely low*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Not assessed*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Not assessed***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**NEGLIGIBLE**

Table 5.9 Combined release, exposure, and consequence assessments, resulting in an unrestricted risk estimate for trichinellosis.

References

Boireau P, Vallee I, Roman T, Perret C, Mingyuan L, Gamble HR, Gajadhar A (2000) *Trichinella* in horses: a low frequency infection with high human risk. *Veterinary Parasitology* 93: 309-320.

Dupouy-Camet J (2006) Trichinellosis: still a concern for Europe. *Euro Surveillance* 11: 5.

Gamble HR, Bessonov AS, Cuperlovic K, Gajadhar AA, van Knapen F, Noeckler K, Schenone H, Zhu X (2000) International Commission on Trichinellosis: recommendations on methods for the control of *Trichinella* in domestic and wild animals intended for human consumption. *Veterinary Parasitology* 93: 393-408.

Hill DE, Forbes L, Kramer M, Gajadhar A, Gamble HR (2007) Larval viability and serological response in horses with long-term *Trichinella spiralis* infection. *Veterinary Parasitology* 146: 107-116.

Murrell KD, Djordjevic M, Cuperlovic K, Sofronic L, Savic M, Djordjevic M, Damjanovic S (2004) Epidemiology of *Trichinella* infection in the horse: the risk from animal product feeding practices. *Veterinary Parasitology* 123: 223-233.

Murrell KD, Lichtenfels RJ, Zarlenga DS, Pozio E (2000) The systematics of the genus *Trichinella* with a key to species. *Veterinary Parasitology* 93: 293-307.

Murrell KD, Stringfellow F, Dame JB, Leiby DA, Duffy C, Schad GA (1987) *Trichinella spiralis* in an agricultural ecosystem. II. Evidence for natural transmission of *Trichinella spiralis spiralis* from domestic swine to wildlife. *The Journal of Parasitology* 73: 103-109.

Obendorf DL, Clarke KP (1992) *Trichinella pseudospiralis* infections in free living Tasmanian birds. *Journal of the Helminthological Society of Washington* 59: 144-147.

Obendorf DL, Handlinger JH, Mason RW, Clarke KP, Forman AJ, Hooper PT, Smith SJ, Holdsworth M (1990) *Trichinella pseudospiralis* infection in Tasmanian wildlife. *Australian Veterinary Journal* 67: 108-110.

OIE (2008) Trichinellosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.16_TRICHINELLOSIS.pdf (Accessed 1 September 2009).

OIE (2009a) Disease control measures: trichinellosis. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=control&disease_type=Terrestrial&disease_id=27&selected_start_year=2008&selected_report_period=1 (Accessed 14 October 2009).

OIE (2009b) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pozio E (2001) New patterns of *Trichinella* infection. *Veterinary Parasitology* 98: 133-148.

Pozio E (2007) World distribution of *Trichinella* spp. infections in animals and humans. *Veterinary Parasitology* 149: 3-21.

Pozio E, Celano GV, Sacchi L, Pavia C, Rossi P, Tamburrini A, Corona S, La Rosa G (1998) Distribution of *Trichinella spiralis* larvae in muscles from a naturally infected horse. *Veterinary Parasitology* 74: 19-27.

Pozio E, Tamburrini A, La Rosa G (2001) Horse trichinellosis, an unresolved puzzle. *Parasite* 8: S263-S265.

Soulsby EJL (1982) Helminths. In *Helminths, arthropods and protozoa of domesticated animals* pp. 7-335. Baillière Tindall, London.

5.36 Tuberculosis

5.36.1 Technical information

Background

Tuberculosis, caused by *Mycobacterium bovis*, is an infectious, chronic respiratory disease in cattle, water buffalo, pigs, deer, goats and a wide range of other animal species. *M. bovis* belongs to the *M. tuberculosis* complex of large gram negative acid fast bacilli which cause nodular granulomas or tubercles within the respiratory system and less commonly in other parts of the body (Oaks 2007). *M. caprae*, a pathogen of goats, and *M. pinnipedii*, a pathogen of fur seals and sea lions, were previously considered to be *M. bovis* (Aranaz et al. 2003; Cousins et al. 2003; OIE 2009a). *M. bovis* infections in horses are extremely rare. Tuberculosis in humans is typically caused by *M. tuberculosis* and *M. bovis* (Animal Health Australia 2007).

Bovine tuberculosis is widespread. Tuberculosis eradication campaigns in many countries have resulted in rare cases of clinical tuberculosis in cattle with *M. bovis* being restricted to particular zones. Bovine tuberculosis has been not been reported for several years from Denmark (1988), Finland (1982) and Norway (1986). Clinical disease was reported in Sweden in 2005 and is present in the rest of Europe (OIE 2009b).

In Australia, *M. bovis* was eradicated in 1997. It had been widespread in cattle and water buffalo. Prior to 1970, when the eradication campaign commenced, *M. bovis* was reported sporadically in other species in Australia, including a sheep, several horses, and a dog (Seddon 1965). Australia maintains its internationally recognised status as free from bovine tuberculosis through monitoring activities under the Tuberculosis Freedom Assurance Program (Animal Health Australia 2007).

M. avium subsp. *paratuberculosis*, which causes Johne's disease in ruminants, is present in Australia. Other mycobacteria are environmental organisms which cause sporadic infections in animals and humans, particularly *M. avium* subsp. *avium* (Oaks 2007). These and non-pathogenic mycobacteria have a worldwide distribution, including Australia, and may interfere with diagnosis of tuberculosis.

This chapter will only consider *M. bovis* infection as it is exotic to Australia.

Bovine tuberculosis is an OIE-listed disease of cattle (OIE 2009c).

Epidemiology

Baccilli are shed in the air by coughing and *M. bovis* is most commonly transmitted to cattle by aerosol. However, infection by ingestion of contaminated material also occurs, especially under intensive animal management, such as housing or high stocking rates (OIE 2009a). Infection via venereal transmission, *in utero* or through skin abrasions is uncommon (Beveridge 1983). Reservoirs of *M. bovis* in wildlife, such as brush-tailed possums in New Zealand, badgers in Ireland and the United Kingdom and wild ruminants in Africa, Canada and the United States, present a risk of infection to domestic animals (OIE 2009a).

In horses, disease caused by *M. bovis* is rare, even in countries with high rates of infection in other species, due to limited exposure to infection and possibly innate resistance (Oaks 2007). Transmission to horses can occur after very close exposure to infected cattle (Radostits et al. 2007) or grazing contaminated pasture (Seddon 1965). A single case of tuberculosis in a horse was attributed to rearing a foal on cow's milk (Gilruth 1911). With the exception of one confirmed case of *M. bovis* in a mare in Spain, all reported infections of horses with mycobacteria in the last 50 years were caused by *M. avium* complex and most reports of *M. bovis* infection of horses over the last 100 years were not confirmed by bacteriology (Monreal et al. 2001). Based on observations in humans, it is speculated that horses with *M. avium* complex infections may be immunodeficient (Oaks 2007). Transmission of *M. bovis* from horses to other species has not been reported.

Clinical signs

M. bovis infection in horses is associated with osteomyelitis of the cervical vertebrae which may restrict neck movement. Other signs include polyuria, coughing, lymph node enlargement, nasal discharge and intermittent pyrexia. Tubercles in horses caused by *M. bovis* are usually located in bone, intestinal wall, mesenteric lymph nodes and spleen. Two cases of cervical osteomyelitis in horses were also associated with infection with *M. avium* complex (Seddon 1965; Radostits et al. 2007).

Diagnosis

Ante-mortem diagnosis of *M. bovis* infection in horses using the intradermal skin test, the prescribed test for international trade, is unreliable because up to 70% of clinically normal horses may have positive results (Konyha and Kreier 1971). A review of tests for tuberculosis in non-bovine species did not include horses (Cousins and Florisson 2005). The presence of acid fast organisms from granulomatous lesions is characteristic of mycobacterial infection, but identification of *M. bovis* requires culture on selective media and biochemical testing which may take several weeks. Polymerase chain reaction tests on clinical samples can rapidly distinguish *M. bovis* from *M. avium* and *M. tuberculosis* (Monreal et al. 2001; OIE 2008; OIE 2009a).

Conclusion

Tuberculosis due to *M. bovis* is present in approved countries but is unlikely to be present in or transmitted by horses.

Tuberculosis was not considered further in the IRA.

References

Animal Health Australia (2007) *Bovine tuberculosis case response manual*. Primary Industries Ministerial Council, Canberra.

Aranaz A, Cousins D, Mateos A, Dominguez L (2003) Elevation of *Mycobacterium tuberculosis* subsp. *caprae* Aranaz *et al.* 1999 to species rank as *Mycobacterium caprae* comb. nov., sp. nov. *International Journal of Systematic and Evolutionary Microbiology* 53: 1785-1789.

Beveridge WIB (1983) Bovine tuberculosis. In *Bacterial diseases of cattle, sheep and goats* pp. 155-170. Australian Bureau of Animal Health, Canberra.

Cousins DV, Bastida R, Cataldi A, Quse V, Redrobe S, Dow S, Duignan P, Murray A, Dupont C, Ahmed N, Collins DM, Butler WR, Dawson D, Rodriguez D, Loureiro J, Romano MI, Alito A, Zumarraga M, Bernardelli A (2003) Tuberculosis in seals caused by a novel member of the *Mycobacterium tuberculosis* complex: *Mycobacterium pinnipedii* sp. nov. *International Journal of Systematic and Evolutionary Microbiology* 53: 1305-1314.

Cousins DV, Florisson N (2005) A review of tests available for use in the diagnosis of tuberculosis in non-bovine species. *Revue Scientifique et Technique de l'Office International des Epizooties* 24: 1039-1059.

Gilruth JA (1911) Two cases of equine tuberculosis (mother and daughter). *The Veterinary Journal* 67: 358-363.

Konyha LD, Kreier JP (1971) The significance of tuberculin tests in the horse. *The American Review of Respiratory Disease* 103: 91-99.

Monreal L, Segura D, Segalés J, Garrido JM, Prades M (2001) Diagnosis of *Mycobacterium bovis* infection in a mare. *The Veterinary Record* 149: 712-714.

Oaks JL (2007) Mycobacterial infections. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 296-300. Saunders Elsevier, St. Louis.

OIE (2008) Avian tuberculosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.03.06_AVIAN_TB.pdf (Accessed 2 September 2009).

OIE (2009a) Bovine tuberculosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.04.07_BOVINE_TB.pdf (Accessed 1 September 2009).

OIE (2009b) List of countries by disease situation: bovine tuberculosis. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=disease_status_lists&disease_type=Terrestrial&disease_id=32&empty=999999 (Accessed 19 June 2009).

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with bacteria IV. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1007-1156. Saunders Elsevier, Edinburgh.

Seddon HR (1965) Tuberculosis. In *Diseases of domestic animals in Australia: part 5. bacterial diseases* (eds. Seddon HR, Albiston HE) pp. 185-228. Department of Health, Canberra.

5.37 Vesicular stomatitis

5.37.1 Technical information

Background

Vesicular stomatitis (VS) virus is a single-stranded RNA virus in the genus Vesiculovirus of the family Rhabdoviridae (Tordo et al. 2005). Two serologically distinct serotypes exist that are associated with outbreaks in the United States — Indiana serotype (with three subtypes) and New Jersey serotype (McCluskey and Mumford 2000; OIE 2008a). New Jersey is the more frequent cause of outbreaks of VS in the United States (Rainwater-Lovett et al. 2007) and responsible for over 80% of clinical cases in endemic areas (Rodriguez 2002).

VS is characterised by vesicular lesions on the tongue, oral mucous membranes, mammary glands, external genitalia and coronary bands (McCluskey and Mumford 2000). It is responsible for substantial loss of productivity and is clinically indistinguishable from foot-and-mouth disease (FMD) except for its occurrence in horses (Letchworth et al. 1999). As such, VS causes anxiety for owners and losses associated with quarantine in FMD-free areas and complicates FMD control in affected regions (Rodriguez 2002).

VS is zoonotic and can cause a temporary debilitating disease in humans (Letchworth et al. 1999). During a VS outbreak in Colorado in 1982, antibody prevalence in farming families and occupationally exposed subgroups was 13%, with relatively close direct contact required for infection to occur (Reif et al. 1987).

VS was described in South Africa in the late 19th century and in France in the 1910s. The disease is now limited to the Americas (OIE 2008a). The last outbreak of VS in Canada was reported in 1949 (OIE 2008b) and in the United States in 2009 (Promed Mail 2009). VS has never been reported in Australia (OIE 2008b).

Outbreaks in the United States mainly affect horses and cattle, although VS viruses have reportedly caused disease in a range of other species including other equids, pigs, llamas and humans. Serum antibody titres have been detected in many other wild and domestic species including ducks, turkeys, coyotes, dogs, cotton rats, wood rats, deer mice, goats, elk, deer, pronghorn antelope and raccoons (McCluskey and Mumford 2000). Experimental infection has been reported in other mammals such as opossums, rats and other laboratory animals (Schmitt 2002).

VS is a multiple species OIE-listed disease (OIE 2009a).

Agent properties

VS virus is inactivated by exposure to 58 °C for 30 minutes (Shahan 1946), although it is stable for prolonged periods at low temperatures (Galasso 1967). It is reported to be stable within a pH range of four to in excess of ten (Fong and Madin 1954). VS virus is rapidly inactivated by sunlight and common disinfectants (Bridges et al. 1997).

Epidemiology

VS is endemic, and occurs seasonally (early summer to the first frost), from northern South America (Columbia, Venezuela, Ecuador and Peru) to southern Mexico and on Ossabaw Island, Georgia, in the eastern United States where the virus has been isolated repeatedly from feral and domestic pigs, and phlebotamine sandflies (McCluskey and Mumford 2000; Stallknecht 2000; Rodriguez 2002).

In Argentina, Brazil and the south-western United States — particularly Arizona, Colorado, New Mexico, Texas and Utah — the disease occurs sporadically (McCluskey and Mumford 2000). Outbreaks tend to occur at approximately ten year intervals (Rodriguez 2002), although in recent years outbreaks have been more frequent (in 2004, 2005, 2006 and 2009).

The epidemiology of VS is not well understood. Viral reservoirs, amplification hosts, and natural modes of transmission are unclear (Cornish et al. 2001). Vesicular fluids contain extremely high concentrations (in excess of 10^8 $TCID_{50}$/ml) of virus (Clarke et al. 1996) and susceptible animals may be exposed to these fluids by direct contact and contact with contaminated fomites (McCluskey and Mumford 2000; Stallknecht et al. 2001). VS is shed from lesions into saliva and contaminates water troughs and probably feed and feed troughs (Letchworth 1996). The virus may gain access to a vertebrate host via minor abrasions or trauma to skin or mucosal surfaces. Experimental studies have found that viral application to superficial breaks or direct inoculation of oral mucous membranes is more effective in producing lesions and results in more frequent and higher titres of viral shedding compared to oral inoculation to unbroken surfaces (Howerth et al. 2006).

Insects have been implicated as both mechanical and biological vectors. The virus has been isolated from many insect species such as black flies, culicoides, house flies, eye gnats, mosquitoes and sand flies (Rodriguez 2002). Transmission of virus from infected black flies and sand flies to susceptible vertebrates has also been demonstrated experimentally (Comer et al. 1990; Mead et al. 1999).

Others have presented arguments against insect transmission of VS virus (Webb and Holbrook 1989; Schmidtmann et al. 1999). Viral multiplication is believed to remain localised, with lesions generally restricted to one anatomic area in the affected animal. Viraemias are not detected and new lesions do not tend to occur following initial detection of clinical disease (McCluskey and Mumford 2000), thus domestic animals do not appear to act as amplifying hosts for VS virus. Viraemia (after experimental infection) has been reported in rodents (Cornish et al. 2001), and it is suggested that deer mice and/or other native American rodents may be involved in the epidemiology.

There are currently two hypotheses used to explain the cyclic nature of epidemics of VS in south-western United States (Rainwater-Lovett et al. 2007). Either VS is endemic in reservoir species and periodically infects domestic animals or each VS outbreak is an introduction into the United States from endemic areas. Phylogenetic studies support the latter hypothesis and suggest that each VS outbreak in the south-western United States is caused by a novel introduction of virus from endemic areas of Mexico. VS has not established in this region of the United States, possibly due to the lack of vectors and reservoirs for long-term maintenance (Rainwater-Lovett et al. 2007). However, the VS virus isolated from animals in the 2006 outbreak in the United States was closely related to viruses isolated in region in the 2005 outbreak,

thought to be due to overwintering of the 2005 VS viruses in the area (USDA:APHIS 2007).

Virus shedding from an active lesion is thought to stop 6–7 days after lesion formation (McCluskey and Mumford 2000). Persistence or shedding of infective VS virus from recovered animals is not known to occur, nor has a mechanism for persistence or latency been determined for any rhabdoviruses (McCluskey and Mumford 2000).

Viral multiplication is believed to remain localised, with lesions generally restricted to one anatomic area in the affected animal (McCluskey and Mumford 2000). High titres of virus occur at the edges of damaged tissues shortly after infection, but a sustained high-titre viraemia is not produced in naturally infected vertebrates (Schmidtmann et al. 1999).

The spread of VS during an outbreak is irregular. Not all susceptible groups of animals become infected and spread does not follow human or animal routes. Outbreaks seem to follow natural features such as valleys and rivers (Letchworth 1996). During outbreaks, a majority of positive identified premises are not contiguous with other positive premises (McCluskey et al. 1999).

In the 1995 outbreak of VS in the United States, positive premises had a quarantine zone (ten miles diameter) placed around the premises. During the 1997 outbreak, positive premises were quarantined but ten mile quarantine zones were not implemented (McCluskey et al. 1999). The United States considered that this quarantine zone was not required as there was insufficient evidence that the spread of disease from infected to adjacent premises occurred. The spread of VS was deemed to be more sporadic, characteristic of insect transmission, so a greater emphasis was placed on insect control (Cunningham 1997). However, some states in the United States require horses from VS-affected states not to have been within ten miles of a VS-infected premises within the previous 30 days (Zimmel 2006).

During VS outbreaks horses are commonly subclinically infected (Mumford et al. 1998b; Mumford et al. 1998a). Oral shedding may occur in horses without detectable oral lesions (Howerth et al. 2006). Generally 10–15% of animals show clinical signs of disease in an affected herd, although disease incidence can vary widely. Mortality is close to zero in horses (OIE 2008a). Horses may be more susceptible to infection and development of clinical disease than cattle: the infection rate on selected farms in one outbreak was 44.7% for horses and 4.5% for cattle, and in a retrospective study, seroprevelance among horses was more than double that of cattle a year after an outbreak (McCluskey and Mumford 2000). In 1995 during an epidemic of VS in the United States, it was determined that at least one animal on 41% of 890 premises investigated in six states was infected. Of the infected animals, horses were identified on 78% of the 362 'infected' premises, and cattle were identified on the remaining 22% (Bridges et al. 1997). Outbreaks may occur on individual isolated premises or as premises clusters (McCluskey and Mumford 2000).

The 2006 United States Animal Health Report states the following figures for VS outbreaks in 2004, 2005 and 2006 (USDA:APHIS 2007):

- 2004: three states; 294 quarantined positive premises; 405 positive horses and 63 positive cattle

- 2005: nine states; 445 quarantined positive premises; 584 positive horses and 202 positive cattle
- 2006: one state; 13 quarantined positive premises; 17 positive horses and 12 positive cattle.

Clinical signs

The incubation period of VS is 1–3 days followed by the development of blanched areas and macules. These become 2–4 cm grey–red vesicles on lips, gums, palate and upper tongue that may coalesce into a large vesicle (Letchworth 1996). Vesicle eruption may also occur on the coronary band, mammary gland of lactating mares, prepuce and sometimes on the ears and ventral abdomen. Vesicles rupture soon after formation, releasing straw-coloured fluid and leaving erosions (Letchworth 1996).

Infected horses can be depressed and listless, with a transient pyrexia that resolves when vesicles rupture. Horses can be reluctant to eat or drink with weight loss due to oral lesions. They can drip saliva, have fetid mouth odour and may grit their teeth. Bleeding, respiratory distress and dysphagia can occur due to inflammation and oedema of the turbinates, nasopharynx and larynx. Lesions of the coronary band can lead to hoof deformity and sloughing of the hoof, potentially requiring euthanasia (Letchworth 1996).

If secondary bacterial infection does not occur, oral erosions heal in 3–21 days by granulation, while udder and foot lesions heal more slowly (Letchworth 1996).

Diagnosis

VS cannot be confirmed based on clinical signs alone. If horses are not involved, early laboratory diagnosis of VS is required to differentiate it from other vesicular diseases such as FMD, vesicular exanthema of swine and swine vesicular disease.

VS virus can be readily isolated and viral RNA can be detected from epithelial tissue and vesicular fluid by conventional and real-time reverse transcriptase polymerase chain reaction (RT-PCR). The preferred immunological methods for identifying viral antigens are the enzyme-linked immunosorbent assay (ELISA), the complement fixation test (CFT) and fluorescent antibody staining, while the virus neutralisation (VN) test is more time consuming (OIE 2008a). For clinical samples, real-time RT-PCR may be more sensitive than virus isolation or CFT (Letchworth 1996).

If sample collection for identification of virus is not possible, paired sera from the same animal, collected 1–2 weeks apart can be used to detect a change in antibody titre (OIE 2008a). Prescribed tests for international trade by the OIE are liquid-phase blocking ELISA (LP-ELISA), competitive ELISA (C-ELISA), VN and CFT. The OIE Manual states that the LP-ELISA is the method of choice for the detection and quantification of antibodies to VS serogroup viruses (OIE 2008a). The ELISA and VN test are preferable for identification and quantification of specific antibodies in serum, while the CFT may be used for quantification of early antibodies (OIE 2008a).

The C-ELISA can be used as a screening test. The C-ELISA and VN test are of comparable sensitivity with the C-ELISA being less serotype-specific than the VN test and a capture ELISA that specifically detects IgM (mcELISA) has been developed by the United States Department of Agriculture (McCluskey and Mumford 2000). C-ELISA and mcELISA can detect antibody within 5–6 days of infection, and

CFT and VN test 1–3 days later. CFT titres are maximal two weeks following exposure. Antibody titres determined by mcELISA decline more rapidly than CFT titres, and both are at undetectable levels by 110 days following exposure. In contrast, VN antibody titres continue to rise and may be detectable for 1–3 years post-infection, thus providing the ability to identify recent infections. It is common to have variable antibody responses among individual affected animals (McCluskey and Mumford 2000).

Immunology

Serotype-specific responses are mounted after infection with a single serotype of VS (Katz et al. 1997). It is likely that a protective response to one serotype will not provide cross-protection against the other (McCluskey et al. 1999).

McCluskey and Mumford (2000) suggest that horses with circulating antibody may be susceptible to reinfection because exposure and subsequent infection are generally localised to epithelial layers, or because variability among strains within serotypes may produce differences in specificity in the immune response.

Vaccines are not commercially available in the United States, although an autogenous killed virus vaccine was approved for use in horses and cattle in select states during outbreaks in the south-western United States in 1963, 1985 and 1995. No efficacy data are available for these (McCluskey and Mumford 2000). Killed vaccines are manufactured in Colombia and Venezuela for the Indiana and New Jersey serotypes of VS (OIE 2008a).

Other vesicular diseases

If horses are not involved, VS is clinically indistinguishable from FMD, vesicular exanthema of swine and swine vesicular disease (OIE 2008a). The presence of a vesicular disease in cloven-hoofed animals must be regarded as suspicious of FMD and rapid laboratory confirmation is essential. Horses are not susceptible to FMD (Radostits et al. 2007) and there are no reports of equids involved in the epidemiology of FMD outbreaks.

Conclusion

VS is present in some approved countries and there are recommendations in the Code (OIE 2009b). Australia's current quarantine measures for VS differ to those in the Code. The Expert Panel thus considered that a risk assessment was appropriate.

5.37.2 Risk assessment

For details of the method used in this risk assessment see section 3.2 of chapter 3.

This risk assessment is based on the assumption that a response consistent with AUSVETPLAN Disease Strategy Manual for VS (ARMCANZ 1996) would be implemented.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of VS being present in an imported horse.

- VS outbreaks occur sporadically in the United States (Rodriguez 2002) and VS is endemic and occurs seasonally in limited areas of the United States (McCluskey and Mumford 2000).
- Subclinically infected horses are common during outbreaks (Mumford et al. 1998b; Mumford et al. 1998a) but latent infection is not known to occur (McCluskey and Mumford 2000).
- In outbreaks, disease incidence can vary widely (OIE 2008a).
- The epidemiology of VS in countries where the disease occurs is not well understood and a conservative approach is warranted.

Based on these considerations, the likelihood of release of VS associated with horses from a country where the disease is present was estimated to be '*low*'.

Exposure assessment

Direct contact and fomite transmission are equally likely exposure pathways with equivalent outcomes. Insects have been implicated as mechanical and biological vectors.

The exposure group considered is equids (including feral equids) as they are considered to be more susceptible to VS infection than other species (McCluskey and Mumford 2000).

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to VS via an imported horse.

- Oral shedding can occur in horses without detectable oral lesions (Howerth et al. 2006).
- Shedding of virus from an active lesion is thought to stop 6–7 days after lesion formation (McCluskey and Mumford 2000).
- VS virus is rapidly inactivated by sunlight and common disinfectants (Bridges et al. 1997), but the virus is stable in the environment for prolonged periods at low temperatures (Galasso 1967).
- Not all susceptible groups of animals become infected (Letchworth 1996) but horses are considered to be more susceptible to VS infection than other species (McCluskey and Mumford 2000).

Based on these considerations, the likelihood of susceptible animals being exposed to VS via an infected imported horse was estimated to be '*moderate*'.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*low*' combined with the likelihood of exposure estimated to be '*moderate*', the likelihood of release and exposure was estimated to be ***'low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario was determined by the extent of establishment and/or spread at detection. The most likely outbreak scenario following exposure to VS is considered to be establishment and/or spread to populations of susceptible animals (including equids, pigs and ruminants) within a district or region through direct contact and fomite transmission.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals to VS.

- Movement of incubating or subclinically infected animals, contaminated fomites and mechanical vectors could spread the disease before detection. In the 2005 outbreak of VS in the United States, nine states were affected (USDA:APHIS 2007).
- Outbreaks in the United States mainly affect horses and cattle (McCluskey and Mumford 2000). Animals, particularly cattle (or pigs), with clinical signs of a vesicular disease would probably be readily detected and control measures quickly implemented before establishment of VS in feral pig or wildlife populations.
- High titres of virus occur at the edges of damaged tissues shortly after infection, but a sustained viraemia is not produced in naturally infected vertebrates (Schmidtmann et al. 1999).
- Shedding of virus from an active lesion is thought to cease 6–7 days after lesion formation (McCluskey and Mumford 2000).
- The role of insects as biological vectors of disease is not well understood and the role of vertebrate viral reservoirs in the epidemiology of VS is also unclear. Although some insect species from which VS virus has been isolated occur in Australia, it is unknown if Australia has suitable reservoir hosts for establishment of virus in wildlife.
- Disease has not established outside the Americas.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of VS was estimated to be '*moderate*'.

Determination of the effects resulting from this outbreak scenario

Following estimation of establishment and/or spread of a disease agent is the determination of the effects (health, environmental and socioeconomic) resulting from that outbreak scenario. Adverse effects are evaluated in terms of seven (two direct and five indirect) criteria.

The following factors were considered relevant to a conclusion on the effects of the establishment and/or spread of VS for each criterion.

Direct effects

The effect on the life or health (including production effects) of susceptible animals

- VS is responsible for loss of productivity in cattle due to increased culling and mortality and reduced milk production in dairy herds and performance losses in affected stables (Goodger et al. 1985; Alderink 1984; Hayek et al. 1998).
- Morbidity in susceptible species may be high but most animals will recover.
- Lesions of the coronary band can lead to hoof deformity and sloughing of the hoof, potentially requiring euthanasia (Letchworth 1996).
- VS is zoonotic and can cause a temporary debilitating disease in humans that are exposed through occupations such as veterinarians (Letchworth et al. 1999).

Based on these considerations, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

The effect on the living environment, including life and health of wildlife, and any effects on the non-living environment

- In areas where VS is endemic, vertebrate wildlife have serological evidence of infection with the virus; however, clinical signs of disease are not reported. It is not known if Australian wildlife and insects are susceptible to infection with the virus, and it is considered that clinical disease is unlikely to be *discernible* in wildlife.

Based on this consideration, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

Indirect effects

The effect on new or modified eradication, control, monitoring or surveillance and compensation strategies or programs

- VS is nationally notifiable in Australia (DAFF 2008).
- If VS were to be identified in Australia, the current policy as outlined in AUSVETPLAN Disease Strategy Manual for VS (ARMCANZ 1996) is to eradicate VS, recognising that the virus may be transmitted by a variety of insect vectors and that the disease does not always follow predictable transmission and distribution patterns.

- VS is scheduled as Category 2 under the Australia's Emergency Animal Disease Response Agreement (EADRA) for cost-sharing arrangements (Animal Health Australia 2001). Should it be activated, EADRA states that costs of the response would be covered by government and relevant industries by contributions of 80% and 20% respectively (Animal Health Australia 2001). However, currently the horse industry is not a signatory to the agreement. Other animal industries, such as those associated with cattle and pigs, are signatories to the agreement.
- A combination of strategies will be employed, including judicious slaughter and disposal of clinically affected animals, quarantine and movement controls, tracing and surveillance, vector control, decontamination, epidemiological investigations, and a public awareness campaign.
- The presence of a vesicular disease in cloven-hoofed animals would initially be regarded as suspicious of FMD and differentiation of VS from FMD is required.
- In this outbreak scenario where VS has only limited spread, eradication would be possible. However, if the disease were to become established in vertebrate and/or invertebrate reservoir hosts, periodic outbreaks could occur.

Based on these considerations, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on domestic trade or industry, including changes in consumer demand and effects on other industries supplying inputs to, or using outputs from, directly affected industries

- VS is nationally notifiable in Australia. If it were detected in Australia, the current policy as outlined in AUSVETPLAN Disease Strategy Manual for VS (ARMCANZ 1996) is to eradicate VS, recognising that the virus may be transmitted by a variety of insect vectors and that the disease does not always follow predictable transmission and distribution patterns.
- As a result of the detection of VS, movement restrictions would be imposed on susceptible species and other potentially infected fomites.
- Movements of animals to sale and slaughter would be affected. Clinically affected cattle would not be accepted for slaughter for human consumption. Horse racing and other equestrian events would probably be prohibited.
- Following detection of VS in one state or territory of Australia, other states may close their borders to susceptible animals and products until the extent of the outbreak was ascertained.
- Public health perceptions and market fluctuations may reduce the value of cattle and pig industries.
- Supporting industries such as stockfeed manufacturers, veterinarians and farriers could also be affected. Additional labour would be involved on infected premises caring for sick animals (Hayek et al. 1998).

Based on these considerations, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on

directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on international trade, including loss of and restriction of markets, meeting new technical requirements to enter or maintain markets, and changes in international consumer demand

- The presence of a vesicular disease in cloven-hoofed animals would initially be regarded as suspicious of FMD. International trade in live susceptible animals and their products would be disrupted until VS was differentiated from FMD.
- The effects on international trade of a confirmed outbreak of VS in Australia would result in national disruption to exports of live animals, including horses, pigs, cattle and sheep and possibly markets for meat.
- If eradication were delayed, possibly because of establishment in feral or wild animals, the effect on live animal trade would be prolonged. The Code recommends that for the importation of animals from countries considered infected with VS, the animals should be held in quarantine and protected from insects for 30 days and be serologically negative and healthy at the time of shipment. Quarantine and protection from insects of feeder cattle prior to export may not be feasible in Australia. Zoning to assist in the international marketing of these animals may need to be adopted.
- If VS were to become established, recurrent outbreaks may result in periodic disruption to international trade.

Based on these considerations, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be significant at the state level. The effect on the national economy or the Australian community as a whole and not just on directly affected parties, corresponds to *minor at the national level* (national effect score E in Table 3.4).

The effect on the environment, including biodiversity, endangered species and the integrity of ecosystems

- VS is not considered to lead to any indirect effects on the environment.

Based on this consideration, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be *unlikely to be discernible at all levels* (national effect score A in Table 3.4).

The effect on communities, including reduced rural and regional economic viability and loss of social amenity, and any 'side effects' of control measures

- Disruption of horse events would have social consequences for people involved in these events.
- Slaughter of clinically affected animals would have emotional effects for people in communities.
- Where susceptible species were important to the local economy, the economic viability of communities within affected regions may be threatened due to loss of associated industries.

Based on these considerations, the effect of the establishment and/or spread of VS in Australia for this criterion was estimated to be significant at the local level. The effect on the national economy or the Australian community as a whole and not just on

directly affected parties, corresponds to *minor at the regional level* (national effect score C in Table 3.4).

Estimation of likely consequences

The measure of effect (A–G) obtained for each direct and indirect criterion (Table 3.4) was combined to give the overall effect of a disease agent. The rules (Figure 3.5) were used for the combination of direct and indirect effects.

Based on the rules described in Figure 3.5, that is, where the effect of a disease with respect to one or more criteria is 'E', the overall effect associated with the outbreak scenario is considered to be '*moderate*'.

The estimate of the overall effect associated with the outbreak scenario was combined with the likelihood of establishment and/or spread for the scenario using Table 3.5 to obtain an estimation of likely consequences.

The likelihood of establishment and/or spread ('*moderate*') is combined with the estimate of the overall effect of establishment and/or spread ('*moderate*') which results in ***'moderate***' likely consequences.

Risk estimation

Risk estimation is the integration of the likelihood of release and exposure and the likely consequences of establishment and/or spread to derive the risk associated with release, exposure, establishment and/or spread of VS introduced by the importation of horses into Australia.

Using Table 3.6, the likelihood of release and exposure ('***low***') is combined with the likely consequences of establishment and/or spread ('***moderate***'), resulting in a risk estimation of **LOW**.

Conclusion

The unrestricted risk associated with VS is determined to be **LOW**. The unrestricted risk estimate exceeds Australia's ALOP and, therefore, risk management is deemed necessary.

A summary of the risk assessment for VS is shown in Figure 5.10 and Table 5.10.

Figure 5.10 **Summary of the risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for VS.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Low*
Likelihood of *exposure*	Likelihood of *exposure*	*Moderate*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods (Table 3.3)	***Low***
Consequences assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Moderate*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Moderate*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**LOW**

Table 5.10 **Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for VS.**

References

Alderink FJ (1984) Vesicular stomatitis epidemic in Colorado: clinical observations and financial losses reported by dairymen. *Preventive Veterinary Medicine* 3: 29-44.

Animal Health Australia (2001) *Government and livestock industry cost sharing deed in respect of emergency animal disease responses*. No. 07/01 - 23.01.09, Australian Animal Health Council Limited, Canberra.

ARMCANZ (1996) *Disease strategy: vesicular stomatitis*. Agriculture and Resource Management Council of Australia and New Zealand, Canberra.

Bridges VE, McClusky BJ, Salman MD, Hurd HS, Dick J (1997) Review of the 1995 vesicular stomatitis outbreak in the western United States. *Journal of the American Veterinary Medical Association* 211: 556-560.

Clarke GR, Stallknecht DE, Howerth EW (1996) Experimental infection of swine with a sandfly (*Lutzomyia shannoni*) isolate of vesicular stomatitis virus, New Jersey serotype. *Journal of Veterinary Diagnostic Investigation* 8: 105-108.

Comer JA, Tesh RB, Modi GB, Corn JL, Nettles VF (1990) Vesicular stomatitis virus, New Jersey serotype: replication in and transmission by *Lutzomyia shannoni* (Diptera: Psychodidae). *The American Journal of Tropical Medicine and Hygiene* 42: 483-490.

Cornish TE, Stallknecht DE, Brown CC, Seal BS, Howerth EW (2001) Pathogenesis of experimental vesicular stomatitis virus (New Jersey serotype) infection in the deer mouse (*Peromyscus maniculatus*). *Veterinary Pathology* 38: 396-406.

Cunningham W (1997) Vesicular stomatitis looms again. Colorado Dairy Newsletter: Colorado State University. http://www.cvmbs.colostate.edu/ilm/proinfo/cdn/97archives.htm (Accessed 28 May 2009).

DAFF (2008) National list of notifiable animal diseases. Department of Agriculture, Fisheries and Forestry. http://www.daff.gov.au/animal-plant-health/pests-diseases-weeds/animal/notifiable (Accessed 9 January 2009).

Fong J, Madin SH (1954) Stability of vesicular stomatitis virus at varying hydrogen ion concentrations. *Proceedings of the Society for Experimental Biology and Medicine* 86: 676-678.

Galasso GJ (1967) Quantitative studies on the quality, effects of aggregation and thermal inactivation of vesicular stomatitis virus. *Archiv für die Gesamte Virusforschung* 21: 437-446.

Goodger WJ, Thurmond M, Nehay J, Mitchell J, Smith P (1985) Economic impact of an epizootic of bovine vesicular stomatitis in California. *Journal of the American Veterinary Medical Association* 186: 370-373.

Hayek AM, McCluskey BJ, Chavez GT, Salman MD (1998) Financial impact of the 1995 outbreak of vesicular stomatitis on 16 beef ranches in Colorado. *Journal of the American Veterinary Medical Association* 212: 820-823.

Howerth EW, Mead DG, Mueller PO, Duncan L, Murphy MD, Stallknecht DE (2006) Experimental vesicular stomatitis virus infection in horses: effect of route of inoculation and virus serotype. *Veterinary Pathology* 43: 943-955.

Katz JB, Ernisse KA, Landgraf JG, Schmitt BJ (1997) Comparative performance of four serodiagnostic procedures for detecting bovine and equine vesicular stomatitis virus antibodies. *Journal of Veterinary Diagnostic Investigation* 9: 329-331.

Letchworth GJ (1996) Vesicular stomatitis. In *Virus infections of equines* (ed. Studdert MJ) pp. 265-279. Elsevier Science Publishers, Amsterdam.

Letchworth GJ, Rodriguez LL, Barrera JDC (1999) Vesicular stomatitis. *The Veterinary Journal* 157: 239-260.

McCluskey BJ, Hurd HS, Mumford EL (1999) Review of the 1997 outbreak of vesicular stomatitis in the western United States. *Journal of the American Veterinary Medical Association* 215: 1259-1262.

McCluskey BJ, Mumford EL (2000) Vesicular stomatitis and other vesicular, erosive, and ulcerative diseases of horses. *Emerging Infectious Diseases* 16: 457-469.

Mead DG, Maré CJ, Ramberg FB (1999) Bite transmission of vesicular stomatitis virus (New Jersey serotype) to laboratory mice by *Simulium vittatum* (Diptera: Simuliidae). *Journal of Medical Entomology* 36: 410-413.

Mumford EL, McCluskey BJ, Traub-Dargatz JL, Schmitt B, Salman MD (1998a) Vesicular stomatitis: epidemiology of the 1995 and 1997 outbreaks in Colorado. In *Proceedings of the 44th annual convention of the AAEP, December 6-9, 1998, Baltimore, Maryland,* pp. 23-25, American Association of Equine Practitioners, Lexington.

Mumford EL, McClusky BJ, Traub-Dargatz JL, Schmitt BJ, Salman MD (1998b) Serologic evaluation of vesicular stomatitis virus exposure in horses and cattle in 1996. *Journal of the American Veterinary Medical Association* 213: 1265-1269.

OIE (2008a) Vesicular stomatitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.19_VESICULAR_STOMITIS.pdf (Accessed 1 September 2009a).

OIE (2008b) WAHID interface. World Animal Health Information Database: version 1.2. http://www.oie.int/wahid-prod/public.php?page=home (Accessed 14 November 2008).

OIE (2009a) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2009b) Vesicular stomatitis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.15.htm (Accessed 1 September 2009).

Promed Mail (2009) Vesicular stomatitis, equine - USA: (Texas). ProMED Mail. http://www.promedmail.org (Accessed 19 June 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia I. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1157-1305. Saunders Elsevier, Edinburgh.

Rainwater-Lovett K, Pauszek SJ, Kelley WN, Rodriguez LL (2007) Molecular epidemiology of vesicular stomatitis New Jersey virus from the 2004-2005 US outbreak indicates a common origin with Mexican strains. *Journal of General Virology* 88: 2042-2051.

Reif JS, Webb PA, Monath TP, Emerson JK, Poland JD, Kemp GE, Cholas G (1987) Epizootic vesicular stomatitis in Colorado, 1982: infection in occupational risk groups. *The American Journal of Tropical Medicine and Hygiene* 36: 177-182.

Rodriguez LL (2002) Emergence and re-emergence of vesicular stomatitis in the United States. *Virus Research* 85: 211-219.

Schmidtmann ET, Tabachnick WJ, Hunt GJ, Thompson LH, Hurd HS (1999) 1995 epizootic of vesicular stomatitis (New Jersey serotype) in the western United States: an entomologic perspective. *Journal of Medical Entomology* 36: 1-7.

Schmitt B (2002) Vesicular stomatitis. Veterinary Clinics of North America: Food Animal Practice 18: 453-459.

Shahan MS (1946) Effect of temperature, phenol, and crystal violet on vesicular stomatitis virus. *American Journal of Veterinary Research* 7: 27-31.

Stallknecht DE (2000) VSV-NJ on Ossabaw Island, Georgia: the truth is out there. *Annals of the New York Academy of Sciences* 916: 431-436.

Stallknecht DE, Perzak DE, Bauer LD, Murphy MD, Howerth EW (2001) Contact transmission of vesicular stomatitis virus New Jersey in pigs. *American Journal of Veterinary Research* 62: 516-520.

Tordo N, Benmansour A, Calisher C, Dietzgen RG, Fang RX, Jackson AO, Kurath G, Nadin-Davis S, Tesh RB, Walker PJ (2005) Rhabdoviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 623-643. Elsevier, San Diego.

USDA:APHIS (2007) *2006 United States animal health report*. No. 801, United States Department of Agriculture, Animal and Plant Health Inspection Service, Fort Collins.

Webb PA, Holbrook FR (1989) Vesicular stomatitis. In *The arboviruses: epidemiology and ecology* (ed. Monath TP) pp. 1-29. CRC Press, Boca Raton.

Zimmel DN (2006) Causes and symptoms of vesicular stomatitis. American Association of Equine Practitioners. http://www.aaep.org/health_articles_view.php?id=294 (Accessed 28 May 2009).

5.38 West Nile fever

5.38.1 Technical information

Background

West Nile virus (WNV), the aetiological agent of West Nile fever, is a member of the Japanese encephalitis (JE) virus serogroup of arthropod-borne flaviviruses in the family Flaviviridae (Thiel et al. 2005). This complex also includes, St Louis encephalitis virus and Murray Valley encephalitis virus (Gubler et al. 2007). Two lineages of WNV are described, based on nucleotide sequence data. Lineage I viruses have been isolated from different regions of Africa, Australia, Europe, India, the Middle East and the Western Hemisphere. Kunjin virus, a strain in Lineage I, is present in Australia (Hayes 2001). Lineage II viruses are from Africa and Madagascar (Gubler et al. 2007).

WNV was originally isolated from a febrile human patient in the West Nile province of Uganda in the 1930s. WNV was recognised in the Western Hemisphere for the first time in 1999. The virus had previously been found in Africa, western Asia, the Mediterranean region of Europe and the Middle East. WNV spread to the United States in 1999, and subsequently to Canada, Mexico (Blitvich et al. 2003; Loroño-Pino et al. 2003), parts of Central America (Cruz et al. 2005), South America (Mattar et al. 2005; Morales et al. 2006) and the Caribbean (Komar and Clark 2006), causing fatal neurological disease in humans, horses and birds (Steele et al. 2000; Komar and Clark 2006). There has been a single case of a horse imported into Australia in 2002 being diagnosed with WNV while in quarantine.

WNV is a multiple species OIE-listed disease (OIE 2009a). The Code recommends that import restrictions should not be imposed on dead-end hosts (i.e. animals that do not pass the virus on to vectors or other animals) such as horses (OIE 2009b).

Epidemiology

WNV has been isolated from numerous species of mammals, birds, reptiles, amphibians and arthropods. Vectors and host animals are reviewed by Hubalek and Halouzka (1999) who consider that wild birds are the principal hosts of WNV. Mosquito vectors (mostly *Culex* spp.) and wild birds maintain the virus in a natural transmission cycle, with humans and horses occasionally serving as dead-end hosts.

Although non-mosquito borne transmission of WNV has been reported (CDC 2002a; CDC 2002b; CDC 2002c; CDC 2002d; CDC 2002e; Banet-Noach et al. 2003), none of the reports related to horses.

All viruses isolated in the past decade during WNV outbreaks of human or avian disease belong to Lineage I. Horses develop low levels of viraemia, but clinical disease and mortalities do occur. Horses infected experimentally with WNV developed low level viraemias of short duration (Bunning et al. 2002). Due to the low levels of viraemia, horses are not considered important in the transmission of WNV.

Retrospective serology on horses has been carried out in regions in France and Italy where outbreaks have occurred (Autorino et al. 2002; Durand et al. 2002) and in these

areas, 30–50% of potentially exposed horses showed evidence of seroconversion. Investigations conducted after the first outbreak of WNV infection in the United States in 1999 revealed that approximately 20% in-contact animals were also seropositive to WNV (Castillo-Olivares and Wood 2004). Another study in the United States found that 18.5% of horses associated with naturally occurring clinical cases of WNV infection demonstrated seroconversion to WNV without displaying clinical signs of disease (Kleiboeker et al. 2004). Such exposure would depend on vector density, competence and feeding preferences, and on host factors such as housing, vaccination and exposure to other flaviviruses.

Case fatality rates in horses can vary from 20% to 50% (Cantile et al. 2000; Autorino et al. 2002). During a WNV outbreak in Italy in 1998, clinical disease was seen in 14 of 498 potentially exposed horses (Autorino et al. 2002); of these, six died or were euthanased (Cantile et al. 2000). Tests of sera from 282 horses in the affected area, for IgG, showed an overall seroprevalence of 38%.

Clinical signs

The incubation period in horses is 1–15 days. Neurological signs can appear abruptly and become progressive. The course of the disease — the combination, degree and length of neurological signs — is generally unpredictable. Approximately 30% of horses that recover from clinical signs can suffer from a relapse 7–10 days after appearing to recover from the disease (Long 2007).

Outbreaks of WNV have seen increasing numbers of both horses (10%) and humans (1%) presenting with neurological signs (Castillo-Olivares and Wood 2004). Clinical descriptions of disease in horses vary between reports, though most include muscle fasciculation, weakness, ataxia and proprioception deficits, and may progress to paralysis, recumbency, convulsions and death (Komar 2000). Blindness, somnolence, hyperaesthesia and sensitivity to sound may occur, though pyrexia is not always present (Snook et al. 2001). Horses showing clinical signs have experienced mortality rates of 57%, 42% and 38%, in the outbreaks in France in 2000, Italy in 1998 and the United States in 2000, respectively (Cantile et al. 2001; Murgue et al. 2001; Ostlund et al. 2001).

There is no effective treatment for WNV infection other than supportive depending on the severity of clinical signs.

Diagnosis

Clinical signs cannot be relied on for diagnosis, as they may not be sufficiently prominent to be easily detected, or may not be sufficiently specific to differentiate WNV infection from other causes of encephalitis in horses.

Methods for detecting virus in clinically affected animals may be directed at detecting either virus or antibody. Isolation of virus, polymerase chain reaction, and immunohistochemistry may be used to detect the presence of virus or viral antigen in tissue specimens.

Viraemia in humans and horses usually ceases soon after clinical signs appear, leaving only a small window of opportunity for isolating virus. The low levels of viraemia make it difficult to detect the virus. Therefore, failure to detect WNV or antigen in

samples obtained from a horse's central nervous system should not lead to a conclusion that infection is absent.

Antibody in equine sera can be detected using IgM capture enzyme-linked immunosorbent assay (ELISA), haemagglutination inhibition, IgG ELISA or a plaque reduction neutralisation test (PRNT). In regions where other flaviviruses of the JE serological complex occur, antibody testing may not differentiate WNV from other flavivirus infections, even where paired sera reveal rising titres. The IgM capture ELISA does not distinguish between antibodies due to WNV and some other viruses of the JE virus complex (Snook et al. 2001). Further confusion may occur when serological testing is conducted on vaccinated animals. Levels of IgM may be low or undetectable in some recently infected horses (OIE 2008). Samples that test antibody positive by IgM capture ELISA must undergo further testing using PRNT to confirm the antibody as being specific to WNV.

Immunology

Several types of effective vaccines are currently used in horses in endemic areas. These include inactivated vaccines, DNA vaccines, live attenuated vaccines and genetically modified vaccines (Seino et al. 2007). A formalin-inactivated whole virus vaccine with an adjuvant has been available since 2001, a recombinant canarypox-vectored vaccine since 2003, a DNA vaccine since 2005 and the latest, a chimaeric yellow fever viral vector vaccine since 2006 (Seino et al. 2007; OIE 2008).

Conclusion

West Nile fever is present in some approved countries and there are no recommendations in the Code for horses. Due to the serious nature of the disease for an individual horse and the potential for a clinical case to disrupt post-arrival quarantine (PAQ) a risk assessment was undertaken.

5.38.2 Risk assessment

For details of the method used in this risk assessment, see section 3.2 of chapter 3.

Release assessment

The following factors were considered relevant to an estimate of the likelihood of West Nile virus being present in an imported horse.

- Seroprevalence of WNV in endemic countries ranges from 18.5% to 50% (Autorino et al. 2002; Durand et al. 2002; Kleiboeker et al. 2004). However, this may be an overestimate of the true prevalence due to widespread vaccination and cross-reactions with other flaviviruses.
- Clinical signs of West Nile fever are unpredictable and variable, with over 18% of exposed horses not clinically affected (Kleiboeker et al. 2004).
- There is a potential for recrudescence of infection — the course of disease is variable and 30% of apparently recovered horses suffer from relapses.
- Of approximately 2500 horses imported into Australia from North America in the last ten years, there has been one case of WNV in PAQ.

Based on these considerations, the likelihood of release of WNV associated with horses from a country where the disease is present was estimated to be '*low*'.

Exposure assessment

Vector transmission of WNV from horses does not occur and therefore direct contact is considered the most likely exposure pathway.

Exposure groups are other equids (including feral equids) and wildlife (including wild birds) since these are the species that seroconvert and/or exhibit clinical signs of West Nile fever.

The following factors were considered relevant to an estimate of the likelihood of susceptible animals being exposed to WNV via an imported horse.

- Horses are unlikely to be involved in the transmission of WNV because they develop only low-level viraemias of short duration.
- Although transmission of WNV other than by vectors has been reported to occur, none of these reports has involved horses (CDC 2002a; CDC 2002b; CDC 2002c; CDC 2002d; CDC 2002e).

Based on these considerations, the likelihood of susceptible animals being exposed to an imported horse infected with WNV was estimated to be '*extremely low'*.

Estimation of the likelihood of release and exposure

Estimation of release and exposure considered the volume of trade in horses imported into Australia in one year.

The likelihood of release and exposure is estimated by combining the likelihood of release and the corresponding likelihood of exposure using the matrix of rules for combining descriptive likelihoods (Table 3.3).

With the likelihood of release estimated to be '*low*' combined with the likelihood of exposure estimated to be '*extremely low*', the likelihood of release and exposure for WNV was estimated to be ***'extremely low'***.

Consequence assessment

The consequence assessment describes the potential consequences associated with disease agent entry and exposure, and estimates the likelihood of them occurring.

This involves estimating the likelihood of establishment and/or spread of the disease agent for the most likely outbreak scenario, and determining the direct and indirect effects (health, environmental and socioeconomic) should this outbreak scenario occur. Combining the likelihood of establishment and/or spread for this outbreak scenario with the corresponding overall effect gives an estimation of likely consequences.

Likelihood of establishment and/or spread associated with the outbreak scenario

Once exposure of susceptible animals has occurred, a number of possible outbreak scenarios could follow, ranging from no spread to establishment of widespread disease.

The most likely outbreak scenario following exposure to WNV is considered to be no further establishment and/or spread to populations of susceptible animals or birds through direct contact with infected horses.

The following factors were considered relevant to an estimate of the likelihood of establishment and/or spread associated with exposure of susceptible animals and birds to WNV.

- Horses do not develop sufficiently high virus titres or duration of viraemia to infect other animals, humans or mosquitoes (Bunning et al. 2002).
- There are no reports of transmission from horses to any other animal or bird species or to humans.

Based on these considerations for the identified outbreak scenario, the likelihood of establishment and/or spread of WNV was estimated to be '*negligible*'.

Risk estimation

If the risk assessment proceeded, no combination of any likelihoods or consequences would give a risk estimation above **NEGLIGIBLE**. The risk assessment therefore concluded at this point.

Conclusion

The overall risk associated with West Nile fever is determined to be **NEGLIGIBLE**. As the unrestricted risk estimate achieves Australia's ALOP, no risk management is considered necessary.

The Expert Panel recognises best practice with respect to animal health and welfare. Due to the serious nature of West Nile fever, a clinical case during PAQ would raise animal welfare concerns for the affected horse, result in disease investigations that could delay the release of all horses in the PAQ facility and disrupt entry of subsequent consignments. The Expert Panel thus considers that vaccination of horses against West Nile virus, from countries where clinical disease is known to occur, facilitates trade and minimises the risk of the occurrence of serious clinical disease during quarantine.

A summary of the risk assessment for West Nile fever is shown in Figure 5.11 and Table 5.11.

Figure 5.11 **Summary of risk assessment pathways and assigned likelihoods to derive an estimate of the unrestricted risk for West Nile fever.**

Likelihood / Risk factor	Estimation / description	Likelihood
Release and exposure assessment		
Likelihood of *release*	Likelihood of *release*	*Low*
Likelihood of *exposure*	Likelihood of *exposure*	*Extremely low*
Likelihood of *release and exposure*	Estimated using the matrix for combining qualitative likelihoods using Table 3.3	***Extremely low***
Consequence assessment		
Likelihood of *establishment and/or spread*	Likelihood of *establishment and/or spread* associated with the identified outbreak scenario	*Negligible*
Overall effect of *establishment and/or spread*	Outbreak scenario effects (health, environmental and socioeconomic) of *establishment and/or spread* assessed using Table 3.4 and combined to estimate overall effect using Figure 3.5	*Not assessed*
Likely consequences	Estimated by combining the likelihood of *establishment and/or spread* (associated with the outbreak scenario) with the overall effect of *establishment and/or spread* using the matrix shown in Table 3.5 to obtain the likely consequences	***Not assessed***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	Estimated by combining the likelihood of *release and exposure* with the likely consequences of *establishment and/or spread* using the risk estimation matrix shown in Table 3.6 to obtain the unrestricted risk of *release, exposure, establishment and/or spread*	**NEGLIGIBLE**

Table 5.11 **Summary of the release, exposure and consequence assessments resulting in an unrestricted risk estimate for West Nile fever.**

References

Autorino GL, Battisti A, Deubel V, Ferrari G, Forletta R, Giovannini A, Lelli R, Murri S, Scicluna MT (2002) West Nile virus epidemic in horses, Tuscany region, Italy. *Emerging Infectious Diseases* 8: 1372-1378.

Banet-Noach C, Simanov L, Malkinson M (2003) Direct (non-vector) transmission of West Nile virus in geese. *Avian Pathology* 32: 489-494.

Blitvich BJ, Fernandez-Salas I, Contreras-Cordero JF, Marlenee NL, Gonzalez-Rojas JI, Komar N, Gubler DJ, Calisher CH, Beaty BJ (2003) Serologic evidence of West Nile virus infection in horses, Coahuila State, Mexico. *Emerging Infectious Diseases* 9: 853-856.

Bunning ML, Bowen RA, Cropp CB, Sullivan KG, Davis BS, Komar N, Godsey MS, Baker D, Hettler DL, Holmes DA, Biggerstaff BJ, Mitchell CJ (2002) Experimental infection of horses with West Nile virus. *Emerging Infectious Diseases* 8: 380-386.

Cantile C, Del Piero F, Di Guardo G, Arispici M (2001) Pathologic and immunohistochemical findings in naturally occurring West Nile virus infection in horses. *Veterinary Pathology* 38: 414-421.

Cantile C, Di Guardo G, Eleni C, Arispici M (2000) Clinical and neuropathological features of West Nile virus equine encephalomyelitis in Italy. *Equine Veterinary Journal* 32: 31-35.

Castillo-Olivares J, Wood J (2004) West Nile virus infection of horses. *Veterinary Research* 35: 467-483.

CDC (2002a) Intrauterine West Nile virus infection: New York, 2002. *Morbidity and Mortality Weekly Report* 51: 1135-1136.

CDC (2002b) Laboratory-acquired West Nile virus infections: United States, 2002. *Morbidity and Mortality Weekly Report* 51: 1133-1135.

CDC (2002c) Possible West Nile virus transmission to an infant through breast-feeding: Michigan, 2002. *Morbidity and Mortality Weekly Report* 51: 877-878.

CDC (2002d) Update: investigations of West Nile virus infections in recipients of blood transfusions. *Morbidity and Mortality Weekly Report* 51: 973-974.

CDC (2002e) Update: investigations of West Nile virus infections in recipients of organ transplantation and blood transfusion: Michigan, 2002. *Morbidity and Mortality Weekly Report* 51: 879.

Cruz L, Cardenas VM, Abarca M, Rodriguez T, Reyna RF, Serpas MV, Fontaine RE, Beasley DWC, Da Rosa APAT, Weaver SC, Tesh RB, Powers AM, Suarez-Rangel G (2005) Serological evidence of West Nile virus activity in El Salvador. *The American Journal of Tropical Medicine and Hygiene* 72: 612-615.

Durand B, Chevalier V, Pouillot R, Labie J, Marendat I, Murgue B, Zeller H, Zientara S (2002) West Nile virus outbreak in horses, southern France, 2000: results of a serosurvey. *Emerging Infectious Diseases* 8: 777-782.

Gubler DJ, Kuno G, Markoff L (2007) Flaviviruses. In *Fields virology* (eds. Knipe DM, Howley PM) pp. 1153-1252. Lippincott Williams & Wilkins, Philadelphia.

Hayes CG (2001) West Nile virus: Uganda, 1937, to New York City, 1999. *Annals of the New York Academy of Sciences* 951: 25-37.

Hubálek Z, Halouzka J (1999) West Nile fever: a re-emerging mosquito-borne viral disease in Europe. *Emerging Infectious Diseases* 5: 643-650.

Kleiboeker SB, Loiacono CM, Rottinghaus A, Pue HL, Johnson GC (2004) Diagnosis of West Nile virus infection in horses. *Journal of Veterinary Diagnostic Investigation* 16: 2-10.

Komar N (2000) West Nile viral encephalitis. *Revue Scientifique et Technique de l'Office International des Epizooties* 19: 166-176.

Komar N, Clark GG (2006) West Nile virus activity in Latin America and the Caribbean. *Revista Panamericana de Salud Pública/Pan American Journal of Public Health* 19: 112-117.

Long MT (2007) Flavivirus. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 198-206. Saunders Elsevier, St. Louis.

Loroño-Pino MA, Blitvich BJ, Farfán-Ale JA, Puerto FI, Blanco JM, Marlenee NL, Rosado-Paredes EP, García-Rejón JE, Gubler DJ, Calisher CH, Beaty BJ (2003) Serologic evidence of West Nile virus infection in horses, Yucatan State, Mexico. *Emerging Infectious Diseases* 9: 857-859.

Mattar S, Edwards E, Laguado J, González M, Alvarez J, Komar N (2005) West Nile virus antibodies in Colombian horses. *Emerging Infectious Diseases* 11: 1497-1498.

Morales MA, Barrandeguy M, Fabbri C, Garcia JB, Vissani A, Trono K, Gutierrez G, Pigretti S, Menchaca H, Garrido N, Taylor N, Fernandez F, Levis S, Enría D (2006) West Nile virus isolation from equines in Argentina, 2006. *Emerging Infectious Diseases* 12: 1559-1561.

Murgue B, Murri S, Zientara S, Durand B, Durand JP, Zeller H (2001) West Nile outbreak in horses in southern France, 2000: the return after 35 years. *Emerging Infectious Diseases* 7: 692-696.

OIE (2008) West Nile fever. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/Normes/mmanual/2008/pdf/2.01.20_WEST_NILE.pdf (Accessed 1 September 2009).

OIE (2009a) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

OIE (2009b) West Nile fever. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.16.htm (Accessed 19 August 2009).

Ostlund EN, Crom RL, Pederson DD, Johnson DJ, Williams WO, Schmitt BJ (2001) Equine West Nile encephalitis, United States. *Emerging Infectious Diseases* 7: 665-669.

Seino KK, Long MT, Gibbs EPJ, Bowen RA, Beachboard SE, Humphrey PP, Dixon MA, Bourgeois MA (2007) Comparative efficacies of three commercially available vaccines against West Nile virus (WNV) in a short-duration challenge trial involving an equine WNV encephalitis model. *Clinical and Vaccine Immunology* 14: 1465-1471.

Snook CS, Hyman SS, Del Piero F, Palmer JE, Ostlund EN, Barr BS, Desrochers AM, Reilly LK (2001) West Nile virus encephalomyelitis in eight horses. *Journal of the American Veterinary Medical Association* 218: 1576-1579.

Steele KE, Linn MJ, Schoepp RJ, Komar N, Geisbert TW, Manduca RM, Calle PP, Raphael BL, Clippinger TL, Larsen T, Smith J, Lanciotti RS, Panella NA, McNamara TS (2000) Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. *Veterinary Pathology* 37: 208-224.

Thiel HJ, Collett MS, Gould EA, Heinz FX, Houghton M, Meyer G, Purcell RH, Rice CM (2005) Flaviviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 981-998. Elsevier, San Diego.

6 Risk management

Risk management measures considered in this IRA report aim to reduce the likelihood that an imported horse would lead to the release, exposure, establishment and/or spread of disease agents of quarantine concern. The method for risk management used is consistent with the Code (OIE 2009b).

The Code states at Article 2.1.5. that:

> 'Risk management is the process of deciding upon and implementing measures to achieve the Member's appropriate level of protection.'

Australia has determined that to achieve its Appropriate Level of Protection (ALOP), the quarantine risk associated with imported animals must be at least 'very low'. In the risk assessment chapter, the unrestricted risk was estimated for each disease agent to ascertain whether it achieved Australia's ALOP. If the unrestricted risk estimate was 'very low' or 'negligible', it achieved Australia's ALOP and risk management was not required. If the unrestricted risk estimate did not achieve Australia's ALOP, risk management options to reduce the risk to an acceptable level, i.e. 'very low' or 'negligible', were considered.

For the diseases listed in Table 6.1, the unrestricted risk estimate did not achieve Australia's ALOP and risk management measures were considered necessary to reduce the risk to an acceptable level.

Disease	Unrestricted risk estimate
Equine influenza	Moderate
Equine piroplasmosis	Low
Lyme disease	Low
Surra	Low
Vesicular stomatitis	Low

Table 6.1 Diseases that did not achieve Australia's ALOP and required risk management.

For other diseases of quarantine concern, the Expert Panel considers the risk management measures in accordance with the Code recommendations, are appropriate. These are included in Australia's quarantine measures for the following: African horse sickness, anthrax, contagious equine metritis, dourine, epizootic lymphangitis, equid herpesvirus-1 (abortigenic and neurological strains), equine infectious anaemia, equine viral arteritis, equine viral encephalitides (Eastern, Western and Venezuelan equine encephalitis viruses), glanders, horse pox, Japanese encephalitis, rabies and screw-worm-fly myiasis.

Although assessed as achieving Australia's ALOP, it was the Expert Panel's view that two disease agents warranted further consideration. There are no recommendations in the Code for, and a lack of knowledge about the epidemiology of, Borna disease. Horses should not be sourced from premises where an emerging disease agent of potential quarantine concern is known to occur. This would apply to premises where clinical cases of Borna disease have occurred. The Code recommends for West Nile fever that import restrictions should not be imposed on dead-end hosts (i.e. animals that do not pass the virus on to vectors or other animals) such as horses. The Expert

Panel recognises best practice with respect to animal health and welfare. Due to the serious nature of West Nile fever, a clinical case during PAQ would raise animal welfare concerns for the affected horse, result in disease investigations that could delay the release of all horses in the PAQ facility and disrupt entry of subsequent consignments. The Expert Panel thus considers that vaccination of horses against West Nile virus, from countries where clinical disease is known to occur, facilitates trade and minimises the risk of the occurrence of serious clinical disease during quarantine.

The Code states at Article 2.1.6. that:

> 'Evaluating the efficacy of the options selected is an iterative process that involves their incorporation into the risk assessment and then comparing the resulting level of risk with that considered acceptable.'

The effect of measures was assessed as the change in the risk estimate, and expressed as 'restricted risk'. The restricted risk was derived using either a particular risk management measure or a combination of measures. Where the effect was to reduce the risk estimate to 'very low' or 'negligible', the measure, or combination of measures, was deemed acceptable.

The Code further states at Article 2.1.5 that:

> 'The objective is to manage risk appropriately to ensure that a balance is achieved between a country's desire to minimize the likelihood or frequency of disease incursions and their consequences and its desire to import commodities and fulfil its obligations under international trade agreements.'

In its consideration of risk management measures, the Expert Panel considered equivalent measures that would achieve Australia's ALOP and identified those that would be the least trade restrictive.

6.1 Risk management options

The Code states at Article 2.1.6 that:

> 'Option evaluation - the process of identifying, evaluating the efficacy and feasibility of, and selecting measures in order to reduce the risk associated with an importation in line with the Member's appropriate level of protection. The efficacy is the degree to which an option reduces the likelihood and/or magnitude of adverse health and economic consequences.'

The efficacy and feasibility of risk management measures that could be applied in this IRA report take into account that:

- the animals are live, which rules out quarantine treatments associated with non-viable product e.g. heat treatment
- some horses infected with disease agents do not show clinical signs of infection (i.e. they have a subclinical infection or latent carrier status) and therefore detection of disease through visual observation and examination is unreliable
- horses can be subclinically infected or latent carriers of some disease agents for varying periods of time, affecting the length of the quarantine period

- the sensitivity of some diagnostic tests is not always optimal (i.e. infection may not be detected)
- vaccinations and treatments are not always reliable in preventing disease or stopping shedding of disease agents
- vectors and/or iatrogenic factors can transmit disease agents to other horses.

Risk management options considered apply to horses imported on a permanent basis. For the temporary importation of horses, such as for racing or competition, risk management measures for some diseases could be amended, including those transmitted venereally (e.g. contagious equine metritis and equine viral arteritis) as the horses are held under quarantine surveillance, denied the opportunity to mate and must be re-exported. The following 14 potential risk management options (nine pre-entry measures and five post-arrival measures) that could be applied to horses were considered in this report.

6.1.1 Pre-entry measures

Approved country

Countries, administrative regions and territories from which Australia currently permits the importation of horses, are referred to in the IRA as approved countries and are listed in section 1.4.1.

For countries to be approved, Australia takes into consideration a number of criteria. These include the animal health status of the country, animal health legislation, the effectiveness of systems for control over certification of animals and products, the effectiveness of veterinary and laboratory services, and the standard of reporting of disease outbreaks to the OIE. Australia has issued 'Guidelines for the approval of countries to export animals (including fish) and their products to Australia' (see Animal Quarantine Policy Memorandum 1999/62).

This IRA considers the importation of horses from approved countries only. This provides a satisfactory level of assurance of the exporting countries' capacity for certifying to Australia's quarantine requirements.

Country or area freedom

Horses will only be imported from countries or defined areas, such as states or provinces, free of disease agents of quarantine concern. The horse must have been continuously resident and free of quarantine restriction in the country or defined area of export for not less than 60 days immediately before export. The 60 days residency requirement may be achieved in more than one approved country if specifically authorised by the Australian Quarantine and Inspection Service (AQIS).

Determining disease freedom must be to a standard consistent with that recommended in Article 1.4.6 in the Code (OIE 2009a), or to an equivalent standard for those diseases not listed by the Code. For Australian government authorities to be satisfied that a country or area is free of a given disease, they must have a knowledge of the Veterinary Authority (e.g. the government veterinary service or equivalent) of that country and be satisfied that the Veterinary Authority has the capacity for disease control, monitoring and surveillance, as appropriate for the disease agent. In some

cases, it might be necessary for the disease to be subject to compulsory reporting or disease investigation.

Premises status

Horses will only be imported from premises where disease agents of quarantine concern are not known to have occurred. The horse must have been resident for a specified period immediately before export on premises where no clinical, epidemiological or other evidence of a given disease has occurred for a specified period before export. For the disease status to be certified, the Veterinary Authority may need to undertake investigations, surveillance or monitoring. Certification for diseases that are not notifiable would need to be based on a declaration from a private veterinarian or from the vendor. The Expert Panel recognises that it is difficult to certify premises freedom for diseases that have a subclinical or carrier status, and those for which diagnostic tests have variable or limited sensitivity, or where diseases are not officially notifiable. However, horses should not be sourced from premises where a disease agent of quarantine concern, or an emerging disease agent of potential quarantine concern, for example Borna disease, is known to occur.

Vaccination

Vaccination of horses before export may assist to reduce the likelihood of release for some disease agents. Vaccine production should be consistent with methods and quality management standards recommended in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals (OIE 2009c), hereafter referred to as 'the OIE Manual', for OIE-listed diseases, or to an equivalent standard for those diseases not listed by the Code.

Pre-export quarantine

Pre-export quarantine (PEQ) of horses may assist to reduce the likelihood of release for some disease agents. PEQ allows separation of horses from other animals and the opportunity to apply biosecurity measures to prevent transmission of disease. Horses can be monitored during PEQ for clinical signs of disease, and tested and/or treated for disease agents of quarantine concern.

The PEQ facility must be located within 250 km of the point of export, and must be approved by AQIS and the Veterinary Authority of the country of export, before commencement of the PEQ period. Verification visits by Australian government authorities may be necessary for initial and ongoing approval of the PEQ facility.

The PEQ facility must be located in an approved country, or defined area of an approved country, where no clinical, epidemiological or other evidence of disease agents of quarantine concern are present, and that can be supervised by the Veterinary Authority. Biosecurity measures should be implemented in the PEQ facility to prevent the entry of disease agents and transmission of disease within the facility. Measures include, but are not limited to, two secure stock-proof fences at least five metres apart or a physical barrier providing equivalent security to isolate horses in PEQ; control of movement of horses and personnel in and out of the facility; vector control; hygienic operating practices to prevent transmission of infection via fomites or iatrogenically; disinfection and decontamination, and ensuring horses do not have the opportunity to mate and are not subjected to reproductive manipulation.

The PEQ period will commence from the time the last horse in the consignment has entered the facility and all horses have been examined by the Official Veterinarian. All equipment used in feeding, handling and treating horses in PEQ must be new or cleaned and disinfected before entry and use in PEQ. All feed to be used during PEQ and transport to Australia must enter the PEQ facility before commencement of PEQ. All bedding to be used during PEQ must enter the PEQ facility before commencement of PEQ.

Provided all other quarantine requirements are met, only horses that are inspected and deemed to be clinically healthy and free from signs of infectious disease in the 24 hours before leaving the PEQ facility for the port of export, and at the time of loading, would be exported. AQIS must be notified, before export, of all treatments administered to the horse during PEQ and the reason for the removal of any horse from PEQ.

Diagnostic testing

Diagnostic testing of horses before export may assist to reduce the likelihood of release for some disease agents. Testing would need to be conducted using methods described and recommended in the OIE Manual (OIE 2009c) where such descriptions exist, and at a laboratory meeting the standards recommended by the OIE (OIE 2008). The level of risk reduction provided by testing would depend on the availability and sensitivity of tests, and on sampling and other operational procedures. All testing must be conducted at a laboratory approved and monitored by the Veterinary Authority of the country of export, with all results attached to the health certification.

Preventive treatment(s)

Preventive treatments, such as anthelmintics, parasiticides and insect repellents, before export may assist to reduce the likelihood of release for some disease agents.

Inclusion of general risk management measures of thorough examination and treatment for external parasites was considered appropriate. The Expert Panel similarly considered that anthelmintic treatment was an appropriate risk management measure for internal parasites. Parasite resistance to treatments was not considered.

Treatments must not be administered before or during PEQ that could adversely affect the sensitivity of diagnostic tests used for risk management. All treatments during PEQ must be administered under the control of the Veterinary Authority, and details and records of such treatments available for inspection.

Documentation

Each horse, other than unweaned foals under six months of age travelling with their dam, must travel with an original international veterinary certificate consistent with the Code signed by an Official Veterinarian of the country of export (OIE 2009d). Certification of PEQ measures applied to unweaned foals under six months of age is to be attached to the veterinary certificate of the foal's dam.

Transport measures

Measures applied during the transport of horses from the PEQ facility in the exporting country to the post-arrival quarantine (PAQ) facility in Australia, including transit and transhipment at approved ports, may assist to reduce the likelihood of release for

some disease agents. Risk management measures for the transport of horses from approved countries are described in chapter 7.

6.1.2 Post-arrival measures

Post-arrival quarantine

Quarantine isolation of horses when they arrive in Australia may assist to reduce the likelihood of release for some disease agents of quarantine concern and the exposure of susceptible Australian animals to these agents. PAQ allows isolation and separation of imported horses from the Australian animal population. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake. Horses can be monitored during PAQ for clinical signs of disease, tested and/or treated for disease agents of quarantine concern. AQIS must be notified immediately if there is any evidence of disease during PAQ, and a thorough investigation must be conducted.

Biosecurity measures should be implemented in PAQ facilities to prevent the transmission of infection within the facility and release of disease agents from the facility. Measures include, but are not limited to, two secure stock-proof fences at least five metres apart or a physical barrier providing equivalent security to prevent horses in PAQ having contact with animals outside the facility; control of movement of horses and personnel in and out of the facility; vector control; hygienic operating practices to prevent transmission of infection via fomites or iatrogenically; disinfection and decontamination, and ensuring horses do not have the opportunity to mate.

All equipment used in feeding, handling and treating horses must remain in PAQ or be cleaned and disinfected before removal from PAQ. Provided all quarantine requirements are met, only horses that are deemed to be healthy and free from disease agents of quarantine concern will be released after completion of PAQ.

Diagnostic testing

Diagnostic testing of horses during PAQ may assist to reduce the likelihood of release for some disease agents of quarantine concern. The testing would need to be conducted using methods described and recommended in the OIE Manual (OIE 2008). The level of risk reduction provided by testing would depend on the availability and sensitivity of tests, and on sampling and other operational procedures.

Preventive treatment(s)

Use of preventive treatments, such as parasiticides and insect repellents, during PAQ may assist to reduce the likelihood of release for some disease agents and vectors, and the likelihood of exposure for susceptible Australian animals. Some therapeutic treatments can adversely affect the sensitivity of diagnostic tests for disease agents of quarantine concern. If required during PAQ, they must not be administered until after samples have been collected for diagnostic testing. Therapeutic treatments for diseases not of quarantine concern may be administered to horses during PAQ only after consultation with Australian government authorities.

Quarantine surveillance

Following the completion of PAQ, official quarantine surveillance, such as at quarantine approved premises, may assist to reduce the likelihood of exposure for some disease agents. Horses can be held under quarantine surveillance for diseases of quarantine concern that have limited ability for direct transmission (e.g. only via mating) and if samples for diagnostic testing could not be collected before completion of PAQ (e.g. pregnant mares need to be tested for contagious equine metritis after foaling).

Directions can be given regarding the housing, treatment, examination and diagnostic testing of horses. AQIS must be notified immediately if there is any evidence of disease during quarantine surveillance and a thorough investigation conducted. Once all requirements of quarantine surveillance are met, the horse would be released without restriction into the general horse population.

Horses can also be held under quarantine surveillance as a risk management measure for diseases that have limited ability for transmission (e.g. only via mating or vectors) and are imported for specific purposes, such as temporarily for competition.

Contingency measures

In the event that an imported horse does not meet Australia's quarantine requirements, or during PAQ fails a test and/or shows signs of disease, then that horse, and any or all horses in the PAQ facility, may be:

- detained in quarantine for observation and subjected to additional testing and/or treatment prescribed by Australian government authorities at the importer's expense
- released from the PAQ facility and held under quarantine surveillance with directions, at the importer's expense
- exported at the importer's expense
- destroyed without recompense.

References

OIE (2008) *OIE quality standard and guidelines for veterinary laboratories: infectious diseases*. Office International des Epizooties, Paris.

OIE (2009a) Animal health surveillance. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.1.4.htm (Accessed 14 August 2009a).

OIE (2009b) Import risk analysis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.2.1.htm (Accessed 14 September 2009b).

OIE (2009c) Manual of diagnostic tests and vaccines for terrestrial animals 2009. World Organisation for Animal Health (OIE).

http://www.oie.int/eng/normes/mmanual/A_summry.htm (Accessed 14 September 2009c).

OIE (2009d) Trade measures, import/export procedures and veterinary certification. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_titre_1.5.htm (Accessed 8 September 2009d).

6.2 Risk management options for specific diseases

6.2.1 Equine influenza

The unrestricted risk for equine influenza (EI) associated with the importation of horses was estimated to be **MODERATE**. Risk management options that could be applied to achieve Australia's appropriate level of protection (ALOP) were considered by Biosecurity Australia.

The likelihood of release of EI was estimated to be '*moderate*' and the likelihood of exposure of EI was estimated to be '*high*'. The following options were considered as risk management measures to reduce the likelihood of release and/or exposure for EI.

Pre-entry measures

Country or area freedom

- Equine influenza virus (EIV) circulates in most countries with substantial equine populations (OIE 2008a).
- EI is not notifiable in all approved countries (OIE 2008c).
- Prior exposure to endemic disease and/or vaccination in all approved countries (except New Zealand) means that it is difficult for a country to demonstrate disease freedom. Vaccination can mask clinical signs of disease.
- The Code states that the infective period of EI is 21 days (OIE 2009) .

Based on this information, country freedom was considered a risk management option for EI.

Consideration of country freedom from EI may be given if the disease is notifiable, no clinical, epidemiological or other evidence of EI has been reported in the previous 12 months, and vaccination of the general horse population is not permitted. Supporting documentation must be provided. If vaccination for EI is permitted, means of differentiation of vaccinated from previously infected horses must be in place. Documentation of adequate surveillance to demonstrate freedom from disease should be available for inspection.

Country freedom alone would be sufficient to achieve Australia's ALOP, subject to the horse having been continuously resident and free of quarantine restriction in the country for at least 21 days immediately before export. EIV is highly contagious and if it were introduced to a largely naïve horse population where vaccination is not routinely practised, 21 days allows time for disease to be detected.

Premises status

- EIV circulates in most countries with substantial equine populations (OIE 2008a).
- Vaccination is practised in many EI-endemic countries. Horses that have been vaccinated and/or previously exposed to EIV can shed virus without showing clinical signs of disease, making detection on a premises difficult.
- EI is not notifiable in all approved countries (OIE 2008c).
- Horses can visit multiple premises within a relatively short period.
- Congregation of horses from multiple premises, such as for competition, breeding and sale, increases the risk of exposure to EIV. Virus could circulate in such environments where horses are well vaccinated.
- The Code states that the infective period of EI is 21 days (OIE 2009).

Based on this information, premises status was considered a risk management option for EI. Due to the difficulty in detecting clinical signs of disease in vaccinated and/or previously exposed horses, a requirement for horses to have been resident on premises for 21 days where no clinical, epidemiological or other evidence of EI has occurred during the previous 30 days was considered appropriate for populations of vaccinated horses.

Due to subclinical infection being common in horse populations where EI is endemic and where vaccination is practised, premises status alone was not considered sufficient to reduce the likelihood of release.

Vaccination

- Available vaccines vary widely in both preparation (live, killed, subunit, vectored) and antigen type (viral strain or genetic material used in preparation).
- Vaccination requirements imposed by various authorities differ in timing and vaccine specification, creating uncertainty when assessing vaccine related immunity.
- Vaccination has been reported to reduce the incidence and severity of clinical signs (Powell et al. 1995) and the duration of clinical disease (Morley et al. 1999).
- Vaccination can significantly reduce the duration and extent of viral shedding. Viral shedding can occur in vaccinated horses in the absence of clinical signs of disease (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).
- Since 2004, the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition has recommended that vaccine manufacturers update the American lineage H3N8 component of vaccines to an A/eq/South Africa 03-like virus (other viruses such as A/eq/Wisconsin/03, A/eq/Ohio/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are also suitable) (OIE 2008b).

Based on this information, vaccination was considered a risk management option for EI.

The Expert Panel adopted the OIE Code recommendations relating to EI vaccination and determined that horses should be vaccinated according to the manufacturer's recommendations with a vaccine complying with standards described in the OIE Manual between 21 and 90 days before assembly for export with either a primary course or a booster.

When available in the country of export, a vaccine containing strains recommended by the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition should be used.

Due to variable immune responses of horses to vaccination and the potential for subclinical infection in vaccinated horses, vaccination alone was not considered sufficient to reduce the likelihood of release.

Pre-export quarantine

- EI has an incubation period of 1–4 days (Park et al. 2004).
- EIV is highly contagious and endemic in horse populations in most countries (OIE 2008a). Verifiable and effective biosecurity measures must be in place to prevent entry of virus into pre-export quarantine (PEQ).
- EIV persists in moist, partially protected conditions (e.g. transport vehicles, tack, veterinary equipment and clothing). It persists on hard, nonporous surfaces such as stainless steel and plastic for 24–48 hours, but for less than 8–12 hours on porous surfaces such as cloth and paper (Bean et al. 1982).
- Spread of droplet-borne EIV from an infected horse is reported over at least 32 metres (Miller 1965).
- Infections, especially in previously exposed or vaccinated horses, can be sub-clinical. Vaccinated horses can shed virus without clinical signs (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).
- EIV infection does not result in a carrier state.
- Naïve experimentally infected equids show clinical signs of disease as early as two days after infection, lasting up to ten days, and virus is shed from within 48 hours of infection for up to seven days (Soboll et al. 2003; Heldens et al. 2004; Crouch et al. 2004; Edlund Toulemonde et al. 2005).
- EIV may be able to circulate undetected in PEQ despite isolation measures.

Based on this information, PEQ was considered a risk management option for EI. A PEQ period of at least 14 days was considered adequate by the Expert Panel to provide the opportunity for isolation from the domestic horse population and observation (including monitoring of rectal temperatures).

The Expert Panel noted that many outbreaks, in both fully susceptible and vaccinated horse populations overseas, indicate that direct contact with infected horses and contaminated fomites is the primary means of spread of infection of EI. Experience with EI both overseas and in the 2007 Australian outbreak shows that EI can be controlled and eradicated using standard quarantine and control of movements of

horses and fomites. Although a range of views exist, the Expert Panel concluded that aerosol and windborne spread is unlikely to occur over a distance greater than 100 metres, particularly from contiguous vaccinated populations of horses in the exporting country. To minimise the potential of aerosol spread of EIV to horses in PEQ, for the duration of PEQ, horses must not be held, housed or exercised within 100 metres of other equids not of equivalent health status. Equivalent biosecurity measures, such as impervious barriers between horses in PEQ and other equids, may be authorised by AQIS.

Because of the highly contagious nature of EIV, strict biosecurity procedures must be in place to prevent the entry of EIV into PEQ. These include control of entry and exit of personnel, equipment and other animals, decontamination procedures for personnel and equipment entering the PEQ facility, control of exercise and other areas, and prevention of horses in PEQ having contact with horses outside PEQ.

Due to the highly contagious nature of EI and its presence in approved countries, PEQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- All ages and breeds of equids can be infected with EIV experimentally (Nyaga et al. 1980).
- Clinical signs of disease, such as coughing and nasal discharge, in vaccinated and previously infected horses can be difficult to discern. Mild clinical signs in infected vaccinated horses can make diagnosis difficult on clinical signs alone (Hannant and Mumford 1996).
- In Hong Kong in 1992, EIV infection remained unrecognised among imported vaccinated horses that underwent a 14-day post-arrival quarantine period (Powell et al. 1995). Retrospective investigation suggested EIV escaped quarantine and infection occurred in a large proportion of the vaccinated local population; clinical signs of disease were not reported in Hong Kong until 25 to 32 days after importation (Powell et al. 1995).
- Vaccination can reduce the duration and extent of viral shedding (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).
- Following experimental infection, previously naïve horses shed virus from within 48 hours of infection for up to seven days; the duration of viral shedding in vaccinated horses was up to four days (Soboll et al. 2003; Heldens et al. 2004; Crouch et al. 2004; Edlund Toulemonde et al. 2005). Vaccination can reduce the window of opportunity for detection of the virus.
- Pyrexia is typically the first sign of EI, peaking at 48 to 96 hours after infection (Landolt et al. 2007). Monitoring of rectal temperatures is useful in early detection of EI, including in vaccinated horses.
- Definitive diagnosis of EI is achieved by detecting virus or viral product from nasopharyngeal swabs. Swabs should be sufficiently long to pass through the ventral meatus into the nasopharynx and should be transferred to transport media

and transported on ice (OIE 2008a). Swabs should be collected within 24 hours of the onset of pyrexia (Hannant and Mumford 1996).

- Quantitative polymerase chain reaction (PCR) tests have been developed that allow rapid detection and quantification of viral RNA in swab material. Detection of virus by real-time reverse transcriptase (RT)-PCR has been shown to be faster, less labour-intensive and more sensitive than isolation of virus in eggs or cell culture (Quinlivan et al. 2005).
- Quantitative RT-PCR has been shown to be more sensitive than the ELISA. It is able to detect EIV earlier and for longer than the ELISA or virus isolation in eggs, even if no clinical signs of EI are present (Bryant et al. 2010).
- RT-PCR is more sensitive than rapid antigen detection kits in detecting infection, particularly when only a small amount of virus is shed, and a negative result from a rapid antigen detection kit does not preclude the possibility of infection with EIV (Quinlivan et al. 2004; Yamanaka et al. 2008).
- Viral isolation can take days to weeks to complete. Its value is limited by low sensitivity and by the need for maintenance of conditions optimal for viral preservation during collection and transport.
- Serological tests can be performed on paired sera to demonstrate a rise in EI-specific antibody concentration, with the first sample being taken as early in the course of infection as possible and the second approximately two weeks later (OIE 2008a).
- Serological results can be difficult to interpret, especially in vaccinated horses. Some serological techniques can be used to differentiate between the immune response of infected and vaccinated animals, but only when the vaccine is not a whole virus vaccine preparation.
- In countries where vaccination is routine, most vaccines in commercial use are not marker vaccines and so immune response to vaccination cannot be differentiated serologically from the humoral response to acute infection.
- Blocking ELISA can be used alongside HI testing to differentiate infected from vaccinated animals in horses vaccinated only with the canarypox recombinant vaccine (EI Epidemiology Support Group 2009).

Based on this information, diagnostic testing was considered a risk management option for EI. For diagnostic testing to be useful, it needs to be accompanied by isolation from the domestic horse population from the time of testing until export to prevent potential subsequent transmission of EIV to horses being tested.

Before export, horses should be observed for clinical signs of disease and their rectal temperatures should be monitored. Any cases of pyrexia (persistent rectal temperatures of 38.5 ºC or higher), other signs of infectious respiratory disease, or instances where the temperature is unable to be taken should be investigated.

Based on potential spread of EI within isolated groups of horses, the Expert Panel determined that there is a need to monitor for EIV by PCR testing during a pre-export isolation period, with nasopharyngeal samples for PCR testing being taken four to six

days after commencement of isolation and again during the four days before leaving the PEQ facility.

In case serological testing is later required, reference serum samples should be taken close to the time of commencement of isolation and stored until the horse has completed isolation after arrival in Australia.

Due to the potential for subsequent transmission of EIV to horses after sampling or for horses to be incubating EIV at the time of sampling, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- Antiviral agents may ameliorate clinical signs of disease but probably do not eliminate virus.

Based on this information, preventive treatment was not considered a risk management option for EI.

Post-arrival measures

Post-arrival quarantine

- EI has an incubation period of 1–4 days (Park et al. 2004).
- Undetected circulation of EIV in PEQ, or exposure to EIV before export or during transport could result in importation of infected horses. EIV could circulate undetected in PAQ.
- EIV is highly contagious. Verifiable and effective biosecurity measures must be in place to prevent escape of virus from PAQ into the Australian horse population.
- EIV persists in moist, partially protected conditions (e.g. vehicles for transporting horses, tack, veterinary equipment and clothing). It persists on hard, nonporous surfaces such as stainless steel and plastic for 24–48 hours, but for less than 8–12 hours on porous surfaces such as cloth and paper (Bean et al. 1982).
- Spread of droplet-borne EIV from an infected horse is reported over at least 32 metres (Miller 1965).
- Vaccination can significantly reduce both the clinical signs of EI and the extent of viral shedding in both horses and ponies, however, viral shedding can occur in vaccinated horses in the absence of clinical signs of disease (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).
- EIV infection does not result in a carrier state.
- Immunity to EIV infection is influenced by a horse's exposure to vaccine antigens and/or circulating strains of virus. A horse exposed only to a particular EI antigen may have relatively less effective immunological protection to challenge by another strain of virus displaying altered antigenic properties.
- Vaccine heterogenicity to the challenge strain may contribute to vaccine breakdown (Daly et al. 2003; Park et al. 2004). Antigenic drift was suggested as a

major contributing factor in an EI outbreak in vaccinated horses in the United Kingdom in 1989 (Binns et al. 1993) and in Croatia in 2004 (Barbic et al. 2009).

- Antigenic drift of strains and their sub-lineages can occur in different regions and continents. Antigenically and genetically distinct American and European variants of H3N8 EIV are recognised (Daly et al. 1996).
- European strains of EIV have been isolated in North America and *vice versa* (Mumford 1999)
- Florida sublineage clade 1 viruses appear to predominate in North America and clade 2 viruses predominate in Europe (Bryant et al. 2009).

Based on this information, PAQ was considered a risk management option for EI. A PAQ period of at least 14 days was considered necessary by the Expert Panel to provide the opportunity for isolation of imported horses from the susceptible domestic horse population and observation (including monitoring of rectal temperatures).

If all horses originate from a single PEQ facility or more than one PEQ facility in the same country or region, potential exposure to unfamiliar strains of virus on arrival in PAQ is less likely to occur, resulting in decreased potential for infection. The Expert Panel thus determined that a PAQ period of at least 14 days could be considered in these circumstances.

Virus strains of heterogeneous antigenicity circulating in different regions were considered an increased risk if horses from different regions are commingled during transport or on arrival in PAQ. The Expert Panel considered an increased PAQ period of at least 21 days is necessary to provide extended opportunity to detect virus if present when mixing horses from different regions.

The PAQ period commences from the time of entry into the PAQ facility of the last horse of the PAQ intake. Any commingling can result in staggered intake of horses into PAQ and thus staggered potential introduction of EIV; horses from earlier consignments will then be held for a longer period. The period of intake of consignments into the PAQ facility should be kept to a minimum.

The Expert Panel considered that any single consignment must not be split between PAQ facilities on arrival in Australia. If an outbreak of EI were to occur in a consignment, having the consignment restricted to a single PAQ facility means infection can be managed more effectively. It also provides for potential continuation of horse imports into alternative uninfected PAQ facilities.

The Expert Panel noted that many outbreaks, in both fully susceptible and vaccinated horse populations overseas, indicate that direct contact with infected horses and contaminated fomites is the primary means of spread of infection of EI. Experience with EI both overseas and in the 2007 Australian outbreak shows that EI can be controlled and eradicated using standard quarantine and control of movements of horses and fomites. Although a range a views exist, the Expert Panel concluded that aerosol and windborne spread is unlikely to occur over a distance greater than 100 metres, particularly from vaccinated populations of horses. To minimise the potential of aerosol spread of EIV to horses outside PAQ, for the duration of PAQ, horses must not be held, housed or exercised within 100 metres of other equids not of equivalent health status.

Because of the highly contagious nature of EIV, strict biosecurity procedures must be maintained from the time horses arrive in Australia and for the duration of PAQ to prevent the release of EIV. This includes controls of entry and exit of personnel, equipment and other animals, decontamination of vehicles for transporting horses, decontamination procedures for personnel and equipment leaving the PAQ facility, and prevention of horses in PAQ having contact with horses outside of PAQ.

Due to the highly contagious nature of EI, PAQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- All ages and breeds of equids can be infected with EIV experimentally (Nyaga et al. 1980).
- Clinical signs of disease, such as coughing and nasal discharge, in vaccinated and previously infected horses can be difficult to discern. Mild clinical signs in infected vaccinated horses can make diagnosis difficult on clinical signs alone (Hannant and Mumford 1996).
- In Hong Kong in 1992, EIV infection remained unrecognised among imported vaccinated horses that underwent a 14-day post-arrival quarantine period (Powell et al. 1995). Retrospective investigation suggested EIV escaped quarantine and infection occurred in a large proportion of the vaccinated local population; clinical signs of disease were not reported in Hong Kong until 25 to 32 days after importation (Powell et al. 1995).
- Vaccination can reduce the duration and extent of viral shedding (Chambers et al. 2001; Lunn et al. 2001; Townsend et al. 2001; Crouch et al. 2004; Heldens et al. 2004; Crouch et al. 2005; Edlund Toulemonde et al. 2005; Daly et al. 2007; Minke et al. 2007).
- Following experimental infection, previously naïve horses shed virus from within 48 hours of infection for up to seven days; the duration of viral shedding in vaccinated horses was up to four days (Soboll et al. 2003; Heldens et al. 2004; Crouch et al. 2004; Edlund Toulemonde et al. 2005). Vaccination can reduce the window of opportunity for detection of the virus.
- Pyrexia is typically the first clinical sign of EI, peaking at 48 to 96 hours after infection (Landolt et al. 2007). Monitoring of rectal temperatures is useful in early detection of EI infection, including in vaccinated horses.
- Definitive diagnosis of EI is achieved by detecting virus or viral product from nasopharyngeal swabs. Swabs should be sufficiently long to pass through the ventral meatus into the nasopharynx and should be transferred to transport media and transported on ice (OIE 2008a). Swabs should be collected within 24 hours of the onset of pyrexia (Hannant and Mumford 1996).
- Quantitative PCR tests have been developed that allow rapid detection and quantification of viral RNA in swab material. Detection of virus by RT-PCR has been shown to be faster, less labour-intensive and more sensitive than isolation of virus in eggs or cell culture (Quinlivan et al. 2005).

- Quantitative RT-PCR has been shown to be more sensitive than the ELISA. It is able to detect EIV earlier and for longer than the ELISA or virus isolation in eggs, even if no clinical signs of EI are present (Bryant et al. 2010).
- RT-PCR is more sensitive than rapid antigen detection kits in detecting infection, particularly when only a small amount of virus is shed, and a negative result from a rapid antigen detection kit does not preclude the possibility of infection with EIV (Quinlivan et al. 2004; Yamanaka et al. 2008).
- Viral isolation can take days to weeks to complete. Its value is limited by low sensitivity and by the need for maintenance of conditions optimal for viral preservation during collection and transport.
- Serological tests can be performed on paired sera to demonstrate a rise in EI-specific antibody concentration, with the first sample being taken as early in the course of infection as possible and the second approximately two weeks later (OIE 2008a).
- Serological results can be difficult to interpret, especially in vaccinated horses. Some serological techniques can be used to differentiate between the immune response of infected and vaccinated animals, but only when the vaccine is not a whole virus vaccine preparation.
- In countries where vaccination is routine, most vaccines in commercial use are not marker vaccines so immune response to vaccination cannot be differentiated serologically from the humoral response to acute infection.
- Blocking ELISA can be used alongside HI testing to differentiate infected from vaccinated animals in horses vaccinated only with the canarypox recombinant vaccine (EI Epidemiology Support Group 2009).

Based on this information, diagnostic testing was considered a risk management option for EI. For diagnostic testing to be useful, it needs to be accompanied by isolation to prevent potential transmission of EIV from imported horses to the domestic horse population.

During a post-arrival isolation period, horses should be observed for clinical signs of disease and their rectal temperatures should be monitored. Any cases of pyrexia (persistent rectal temperatures of 38.5 °C or higher), other signs of infectious respiratory disease or instances where the temperature is unable to be taken should be investigated by a veterinarian and AQIS should be informed. Nasopharyngeal samples should be taken and tested for influenza A virus. Temperature records must be available for inspection by AQIS.

Based on potential spread of EI within isolated groups of horses, the Expert Panel determined that there is a need to monitor for EIV by PCR testing during a post-arrival isolation period, with nasopharyngeal samples for PCR testing being taken near the time of arrival in Australia (to detect a horse infected at the end of PEQ or infected during transit) and near the end of the isolation period (to prevent an infected horse being released from isolation).

If all horses originate from a single PEQ facility, the Expert Panel determined that nasopharyngeal samples for PCR testing be taken four to six days after arrival in Australia and within four days of release from isolation.

The Expert Panel determined that for horses originating from multiple PEQ facilities (within or from different regions) an additional nasopharyngeal sample for PCR testing is required on entry into isolation. Early detection of EIV in a consignment may allow subsequent consignments joining that PAQ to be postponed. This decreases numbers of horses and service personnel, reduces the amount of contaminated equipment in the infected PAQ facility, and promotes more rapid resolution of the incident.

In case serological testing is later required, reference serum samples should be taken on entry into isolation and stored.

Due to the potential for imported horses to be incubating EIV at the time of sampling or horses infected with EIV contacting the general horse population while undergoing diagnostic testing, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- Antiviral agents may ameliorate clinical signs of disease but probably do not eliminate virus.

Based on this information, preventive treatment was not considered an effective risk management option for EI.

Conclusion

The unrestricted risk associated with EI was estimated to be **MODERATE**.

Other than country freedom, no single risk management option reduced the unrestricted risk sufficiently to achieve Australia's ALOP. However, the combination of premises status, pre-export and post-arrival diagnostic testing, vaccination, PEQ and PAQ would reduce the likelihood of release for EI from '*moderate*' to '*very low*'.

This would reduce the likelihood of release and exposure to '***very low***' and the restricted risk to at least **VERY LOW**, thereby achieving Australia's ALOP. A summary of the effect of risk management measures for EI is set out in Table 6.2

Quarantine measures for equine influenza

To achieve Australia's ALOP with respect to the risk of EI in imported horses, the following quarantine measures are to be applied to all horses and foals, except that foals under six months of age do not require vaccination against EI.

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine influenza has occurred during the previous 12 months, vaccination against equine influenza is not practised, and the disease is compulsorily notifiable.

OR

For all horses including unweaned foals under six months of age, except where otherwise specified:

For 21 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine influenza has occurred during the previous 30 days.

AND

The horse (other than foals under six months of age) has been vaccinated against equine influenza 21–90 days before commencement of PEQ with either a primary course or a booster according to the manufacturer's recommendations using a vaccine that complies with the standards described in the OIE Manual.

NOTE: Vaccines used must contain the following or equivalent strains of equine influenza virus in accordance with the recommendations of the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition[16]:

an A/eq/South Africa/4/2003 (H3N8)-like virus (American lineage)[17].

AND

The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids not of equivalent health status.

AND

For the duration of PEQ the horse has not been held, housed or exercised within 100 metres of other equids not of equivalent health status, unless specifically authorised by AQIS.

AND

Nasopharyngeal samples have been taken from the horse four to six days after commencement of PEQ and during the four days before leaving the PEQ facility and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

A reference serum sample must be taken from the horse during the four days after commencement of PEQ and stored in the exporting country in a laboratory approved and monitored by the Veterinary Authority until completion of PAQ.

AND

For the duration of PEQ the rectal temperature of the horse has been taken and recorded twice daily at least eight hours apart. If the temperature was 38.5 °C or higher on two consecutive recordings, or other signs of infectious respiratory disease are present, a nasopharyngeal sample, has been taken and tested for influenza A virus and AQIS has been notified within 48 hours. If the temperature has not been taken for any reason on two consecutive occasions, AQIS has been notified within 48 hours and

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[16] Vaccines containing both American and European lineage (A/eq/Newmarket/2/93) are acceptable.

[17] A/eq/Ohio/2003, A/eq/Wisconsin/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are acceptable as A/eq/South Africa/4/2003-like viruses: Other strains (A/eq/Newmarket/1/93 or A/eq/Kentucky/94-like virus) are acceptable until such time as vaccine containing updated strains are available in the country of export.

a clinical examination by a registered veterinarian performed. Temperature records will be kept until completion of PAQ.

AND

For horses originating from a single PEQ facility:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

nasopharyngeal samples must be taken from the horse four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities within the same region:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples, must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities NOT within the same region:

The horse must be held in PAQ for at least 21 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

A single consignment must not be split between PAQ facilities on arrival in Australia.

AND

For the duration of PAQ, the horse must not be held, housed or exercised within 100 metres of other equids not of equivalent health status.

AND

A reference serum sample must be taken from the horse within 24 hours of arrival into the PAQ facility and stored at the National Animal Serum Bank at the Australian Animal Health Laboratory.

AND

For the duration of PAQ the rectal temperature of the horse must be taken and recorded twice daily at least eight hours apart. If the temperature is 38.5 °C or higher on two consecutive recordings or other signs of infections respiratory are present, a nasopharyngeal sample must be taken and tested for influenza A virus and AQIS notified. If the temperature cannot been taken for any reason on two consecutive occasions, AQIS must be notified and a clinical examination by a registered veterinarian performed. Temperature records must be made available for inspection by AQIS.

Requirements for PEQ include:

The PEQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status unless specifically authorised by AQIS.

All personnel entering the PEQ facility during PEQ must shower and change clothing on entry. Alternatively, they may shower off-site and must have no contact with horses or horse facilities between showering and entering the PEQ facility. Outer clothing used in the PEQ facility should be freshly laundered or dedicated to the facility and stored on site or disposable. Footwear used in the PEQ facility should be cleaned and disinfected before entry or dedicated to the facility and stored on site, or disposable covering should be used over existing footwear.

All equipment used in feeding, handling and treating the horse in PEQ must be new or cleaned and disinfected with a product effective against equine influenza virus before use and must be used only in the PEQ facility for the duration of PEQ.

Horses in PEQ must not access any areas used by other horses unless specifically authorised by AQIS.

Vehicles for transporting horses from the PEQ facility to the place of export must be cleaned and disinfected with a product effective against equine influenza virus.

Requirements for PAQ include:

The PAQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status.

All personnel entering the PAQ facility during PAQ must wear dedicated or disposable outer clothing and dedicated, cleaned and disinfected or disposable footwear. All personnel must shower and change outer clothing before leaving the PAQ facility. Outer clothing and footwear used within the PAQ facility must be cleaned to the satisfaction of AQIS before removal from the facility.

All equipment used in feeding, handling and treating the horse in PAQ must either be cleaned and disinfected with a product effective against equine influenza virus to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.

Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.

Likelihood / Risk factor	**Unrestricted**	**Restricted**
Release and exposure assessment		
Likelihood of *release*	*Moderate*	*Very low*
Likelihood of *exposure*	*High*	*High*
Likelihood of *release and exposure*	***Moderate***	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	*High*	*High*
Overall effect of *establishment and/or spread*	*Moderate*	*Moderate*
Likely consequences	***Moderate***	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	**MODERATE**	**VERY LOW**

Table 6.2 Summary of unrestricted risk assessment and restricted risk estimations when risk management measures have been applied for EI.

References

Barbic L, Madic J, Turk N, Daly J (2009) Vaccine failure caused an outbreak of equine influenza in Croatia. *Veterinary Microbiology* 133: 164-171.

Bean B, Moore BM, Sterner B, Peterson LR, Gerding DN, Balfour HH Jr (1982) Survival of influenza viruses on environmental surfaces. *The Journal of Infectious Diseases* 146: 47-51.

Binns MM, Daly JM, Chirnside ED, Mumford JA, Wood JM, Richards CM, Daniels RS (1993) Genetic and antigenic analysis of an equine influenza H3 isolate from the 1989 epidemic. *Archives of Virology* 130: 33-43.

Bryant NA, Paillot R, Rash AS, Medcalf E, Montesso F, Ross J, Watson J, Jeggo M, Lewis NS, Newton JR, Elton DM (2010) Comparison of two modern vaccines and previous influenza infection against challenge with an equine influenza virus from the Australian 2007 outbreak. *Veterinary Research* 41: 1-23.

Bryant NA, Rash AS, Russell CA, Ross J, Cooke A, Bowman S, MacRae S, Lewis NS, Paillot R, Zanoni R, Meier H, Griffiths LA, Daly JM, Tiwari A, Chambers TM, Newton JR, Elton DM (2009) Antigenic and genetic variations in European and North American equine influenza virus strains (H3N8) isolated from 2006 to 2007. *Veterinary Microbiology* 138: 41-52.

Chambers TM, Holland RE, Tudor LR, Townsend HGG, Cook A, Bogdan J, Lunn DP, Hussey S, Whitaker-Dowling P, Youngner JS, Sebring RW, Penner SJ, Stiegler GL (2001) A new modified live equine influenza virus vaccine: phenotypic stability, restricted spread and efficacy against heterologous virus challenge. *Equine Veterinary Journal* 33: 630-636.

Crouch CF, Daly J, Hannant D, Wilkins J, Francis MJ (2004) Immune responses and protective efficacy in ponies immunised with an equine influenza ISCOM vaccine containing an 'American lineage' H3N8 virus. *Vaccine* 23: 418-425.

Crouch CF, Daly J, Henley W, Hannant D, Wilkins J, Francis MJ (2005) The use of a systemic prime/mucosal boost strategy with an equine influenza ISCOM vaccine to induce protective immunity in horses. *Veterinary Immunology and Immunopathology* 108: 345-355.

Daly JM, Lai ACK, Binns MM, Chambers TM, Barrandeguy M, Mumford JA (1996) Antigenic and genetic evolution of equine H3N8 influenza A viruses. *Journal of General Virology* 77: 661-671.

Daly JM, Sindle T, Tearle J, Barquero N, Newton JR, Corning S (2007) Equine influenza vaccine containing older H3N8 strains offers protection against A/eq/South Africa/4/03 (H3N8) strain in a short-term vaccine efficacy study. *Equine Veterinary Journal* 39: 446-450.

Daly JM, Yates PJ, Browse G, Swann Z, Newton JR, Jessett D, Davis-Poynter N, Mumford JA (2003) Comparison of hamster and pony challenge models for evaluation of effect of antigenic drift on cross protection afforded by equine influenza vaccines. *Equine Veterinary Journal* 35: 458-462.

Edlund Toulemonde C, Daly J, Sindle T, Guigal PM, Audonnet JC, Minke JM (2005) Efficacy of a recombinant equine influenza vaccine against challenge with an American lineage H3N8 influenza virus responsible for the 2003 outbreak in the United Kingdom. *The Veterinary Record* 156: 367-371.

EI Epidemiology Support Group (2009) *Equine influenza 2007: the Australian experience*. Animal Health Australia, Canberra.

Hannant D, Mumford JA (1996) Equine influenza. In *Virus infections of equines* (ed. Studdert MJ) pp. 285-293. Elsevier Science Publishers, Amsterdam.

Heldens JGM, Pouwels HGW, van Loon AAWM (2004) Efficacy and duration of immunity of a combined equine influenza and equine herpesvirus vaccine against challenge with an American-like equine influenza virus (A/equi-2/Kentucky/95). *The Veterinary Journal* 167: 150-157.

Landolt GA, Townsend HGG, Lunn DP (2007) Equine influenza infection. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 124-134. Saunders Elsevier, St. Louis.

Lunn DP, Hussey S, Sebring R, Rushlow KE, Radecki SV, Whitaker-Dowling P, Youngner JS, Chambers TM, Holland RE, Horohov DW (2001) Safety, efficacy, and immunogenicity of a modified-live equine influenza virus vaccine in ponies after induction of exercise-induced immunosuppression. *Journal of the American Veterinary Medical Association* 218: 900-906.

Miller WMC (1965) Equine influenza: further observations on the 'coughing' outbreak, 1965. *The Veterinary Record* 77: 455-456.

Minke JM, Edlund Toulemonde C, Coupier H, Guigal P-M, Dinic S, Sindle T, Jessett D, Black L, Bublot M, Pardo MC, Audonnet J-C (2007) Efficacy of a canarypox-vectored recombinant vaccine expressing the hemagglutinin gene of equine influenza H3N8 virus in the protection of ponies from viral challenge. *American Journal of Veterinary Research* 68: 213-219.

Morley PS, Townsend HGG, Bogdan JR, Haines DM (1999) Efficacy of a commercial vaccine for preventing disease caused by influenza virus infection in horses. *Journal of the American Veterinary Medical Association* 215: 61-66.

Mumford JA (1999) Control of influenza from an international perspective. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden O.R.) pp. 11-24, R & W Publications (Newmarket) Limited, Newmarket.

Nyaga PN, Wiggins AD, Priester WA (1980) Epidemiology of equine influenza, risk by age, breed and sex. *Comparative Immunology, Microbiology and Infectious Diseases* 3: 67-73.

OIE (2008a) Equine influenza. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.05.07_EQ_INF.pdf (Accessed 1 September 2009).

OIE (2008b) Expert Surveillance Panel on equine influenza vaccines: conclusions and recommendations. *OIE Bulletin* 2: 42-45.

OIE (2008c) WAHID interface. World Animal Health Information Database: version 1.2. http://www.oie.int/wahid-prod/public.php?page=home (Accessed 14 November 2008).

OIE (2009) Equine influenza. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.12.7.htm (Accessed 19 August 2009).

Park AW, Wood JLN, Daly JM, Newton JR, Glass K, Henley W, Mumford JA, Grenfell BT (2004) The effects of strain heterology on the epidemiology of equine influenza in a vaccinated population. *Proceedings of the Royal Society of London B* 271: 1547-1555.

Powell DG, Watkins KL, Li PH, Shortridge KF (1995) Outbreak of equine influenza among horses in Hong Kong during 1992. *The Veterinary Record* 136: 531-536.

Quinlivan M, Cullinane A, Nelly M, van Maanen K, Heldens J, Arkins S (2004) Comparison of sensitivities of virus isolation, antigen detection, and nucleic acid amplification for detection of equine influenza virus. *Journal of Clinical Microbiology* 42: 759-763.

Quinlivan M, Dempsey E, Ryan F, Arkins S, Cullinane A (2005) Real-time reverse transcription PCR for detection and quantitative analysis of equine influenza virus. *Journal of Clinical Microbiology* 43: 5055-5057.

Soboll G, Horohov DW, Aldridge BM, Olsen CW, McGregor MW, Drape RJ, Macklin MD, Swain WF, Lunn DP (2003) Regional antibody and cellular immune responses to equine influenza virus infection, and particle mediated DNA vaccination. *Veterinary Immunology and Immunopathology* 94: 47-62.

Townsend HGG, Penner SJ, Watts TC, Cook A, Bogdan J, Haines DM, Griffin S, Chambers T, Holland RE, Whitaker-Dowling P, Youngner J, Sebring R (2001) Efficacy of a cold-adapted, intranasal, equine influenza vaccine: challenge trials. *Equine Veterinary Journal* 33: 637-643.

Yamanaka T, Tsujimura K, Kondo T, Matsumura T (2008) Evaluation of antigen detection kits for diagnosis of equine influenza. *The Journal of Veterinary Medical Science* 70: 189-192.

6.2.2 Equine piroplasmosis

The unrestricted risk for equine piroplasmosis associated with the importation of horses was estimated to be **LOW**. Risk management options that could be applied to achieve Australia's appropriate level of protection (ALOP) were considered by Biosecurity Australia.

The likelihood of release of equine piroplasmosis was estimated to be '*moderate*' and the likelihood of exposure of equine piroplasmosis was estimated to be '*moderate*'. The following options were considered as risk management measures to reduce the likelihood of release and/or exposure for equine piroplasmosis.

Pre-entry measures

Country or area freedom

- The causative organisms, *Babesia caballi* and *Theileria equi* are transmitted primarily by ixodid ticks. Prevalence of equine piroplasmosis is higher in tropical and subtropical regions (Radostits et al. 2007) and is affected by host and vector densities, and management procedures.
- Equine piroplasmosis affects equids on all continents except Australia (de Waal 1992).
- The volume of international movement of horses by air transport is significant.
- Horses visiting multiple countries within a relatively short period occurs regularly (Ellis and Watkins 2004).
- Equine piroplasmosis has an incubation period of up to 30 days (Rothschild and Knowles 2007).
- Given the movement of horses, the disease status for equine piroplasmosis of the country of export is not always relevant to the exposure history of a horse (Friedhoff and Soulé 1996).

Based on this information, country or area freedom was considered a risk management option for equine piroplasmosis. Australia may consider an approved country free of equine piroplasmosis if no clinical, epidemiological or other evidence of equine piroplasmosis has been reported in the previous two years. Country freedom alone would be sufficient to achieve Australia's ALOP, subject to the horse having been continuously resident and free of quarantine restriction in the country for at least 60 days immediately before export.

Premises status

- Outbreaks of equine piroplasmosis have occurred when an infected horse has been introduced to a property and the infection has been spread iatrogenically to other resident horses (Callow 1984; Gerstenberg et al. 1999).
- Control measures are directed primarily against the importation of infected horses (Friedhoff et al. 1990).
- Horses infected with *T. equi* or *B. caballi* can be subclinically infected or latent carriers (Radostits et al. 2007).

- Equine piroplasmosis has an incubation period of up to 30 days (Rothschild and Knowles 2007).

Based on this information, premises status was considered a risk management option for equine piroplasmosis. A requirement for horses to have been resident for at least 60 days (i.e. two incubation periods) immediately before commencement of pre-export quarantine (PEQ) on premises where no evidence of equine piroplasmosis has been reported during the 60 days immediately before export was considered appropriate.

Due to the high incidence of subclinical infections and the existence of a carrier state, premises status alone was not considered sufficient to reduce the likelihood of release.

Vaccination

- There are no vaccines against *B. caballi* or *T. equi* (CFSPH 2008).

Based on this information, vaccination was not considered a risk management option for equine piroplasmosis.

Pre-export quarantine

- Equine piroplasmosis has an incubation period of up to 30 days (Rothschild and Knowles 2007).
- The majority of horses infected with equine piroplasmosis remain subclinical (Rothschild and Knowles 2007) and there are latent carriers for life.
- A horse would be protected from infection in PEQ if there was no exposure to ticks or opportunity for iatrogenic spread.

Based on this information, PEQ was considered a risk management option for equine piroplasmosis. A PEQ period equal to the incubation period of 30 days would reduce the likelihood of release of a clinically affected horse; however most infections are subclinical and therefore a PEQ period of this length is not justified. Consequently, the length of PEQ will not be determined by the incubation period. Iatrogenic transmission of equine piroplasmosis is possible via infected blood. Hygienic operating practices would need to be applied during PEQ to prevent iatrogenic transmission. A minimum 14-day PEQ period was considered sufficient for tick control, diagnostic testing and monitoring for clinical signs of disease.

Due to the long incubation period, subclinical infections and carrier state, PEQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- Antibody titres can be detected at 7–11 days, and peak at 45 days post-infection (Tenter and Friedhoff 1986).
- The indirect fluorescent antibody test (IFAT) and the competitive enzyme-linked immunosorbent assay (C-ELISA) have replaced the complement fixation test as the prescribed tests for international trade by the OIE (OIE 2008). The IFAT is more sensitive than the C-ELISA (Ogunremi et al. 2008).

- In a recent outbreak in the United States, the C-ELISA test failed to detect horses found to be positive on polymerase chain reaction (PCR), IFAT and complement fixation tests (OIE 2009).
- Treatment with imidocarb can temporarily clear *B. caballi* and *T. equi* from the blood, resulting in false negative PCR (Butler et al. 2008) and CFT (Kuttler et al. 1988) results.
- There can be operator and laboratory variation in the interpretation of IFAT results and false negatives can occur due to subjective interpretation of fluorescence (Brüning 1996).

Based on this information, diagnostic testing was considered a risk management option for equine piroplasmosis. To be useful, and to prevent potential transmission of equine piroplasmosis to horses being tested, diagnostic testing needs to be accompanied by isolation from the domestic horse population, hygienic practices to prevent iatrogenic transmission, and protection from exposure to ticks, for at least seven days before the time of testing, and until export. To detect recently infected horses, diagnostic testing using the IFAT would be most effective if horses were tested at least seven days after the commencement of isolation, and the risk of infection during isolation was managed. To manage the risk of false negative test results, horses must not be treated with imidocarb or other anti-babesial agents, in the 42 days immediately before commencement of isolation. In addition, horses must not have had a positive test for equine piroplasmosis (*B. caballi* or *T. equi)* from any laboratory in the 60 days immediately before export. To manage the risk of false negative results due to variation between laboratories, testing is to be conducted in the country of export at a government approved laboratory. Where this is not possible in the country of export, a laboratory in a third country would need to be recognised by the exporting country's competent authority.

Due to the imperfect sensitivity, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- There is no pharmacological treatment that will eliminate equine piroplasmosis in horses and there are potential significant adverse reactions associated with treatment (Butler et al. 2008).
- *B. caballi* and *T. equi* can be transmitted iatrogenically by needles, surgical instruments, administration of contaminated blood transfusions or failure to properly sterilise equipment that contacts equine blood, including stomach tubes and dental instruments.
- Visual inspection of horses for ticks and treatment of horses with a parasiticide effective against ticks would reduce the risk of transmission before export.

Based on this information, preventive treatment against ticks was considered a risk management option for equine piroplasmosis.

Due to the risk of iatrogenic transmission, preventive treatment against ticks alone was not considered sufficient to reduce the likelihood of release.

Post-arrival measures

Preventive treatment(s)

- There is no pharmacological treatment that will eliminate equine piroplasmosis in horses and there are potential significant adverse reactions associated with treatment (Butler et al. 2008).
- *B. caballi* and *T. equi* can be transmitted iatrogenically by needles, surgical instruments, administration of contaminated blood transfusions or failure to properly sterilise equipment that contacts equine blood, including stomach tubes and dental instruments.
- Visual inspection of horses for ticks and treatment of horses with a parasiticide effective against ticks would reduce the risk of transmission.

Based on this information, preventive treatment against ticks was considered a risk management option for equine piroplasmosis.

Due to the risk of iatrogenic transmission, preventive treatment against ticks alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- Antibody titres can be detected at 7–11 days, and peak at 45 days post-infection (Tenter and Friedhoff 1986).
- The indirect fluorescent antibody test (IFAT) and the competitive enzyme-linked immunosorbent assay (C-ELISA) have replaced the complement fixation test as the prescribed tests for international trade by the OIE (OIE 2008). The IFAT is more sensitive than the C-ELISA (Ogunremi et al. 2008).

Based on this information diagnostic testing was considered a risk management option for equine piroplasmosis if ticks are detected on horses arriving in Australia. The IFAT would be most effective if horses were tested 11 days after detection of ticks and preventive treatment with a parasiticide effective against ticks.

Due to the imperfect sensitivity, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Conclusion

The unrestricted risk associated with equine piroplasmosis was estimated to be **LOW**.

Other than country freedom, no single risk management option reduced the unrestricted risk sufficiently to achieve Australia's ALOP. However, the combination of premises status, PEQ, diagnostic testing, and inspection and treatment for ticks pre-export and post-arrival would reduce the likelihood of release from '*moderate*' to '*very low*'. This would reduce the likelihood of release and exposure to '***very low***' and the restricted risk to **VERY LOW**, thereby achieving Australia's ALOP.

For the temporary importation of horses seropositive for equine piroplasmosis, a risk assessment was undertaken in 1998 (AQIS 1999) and the quarantine measures from this report will be applied.

A summary of the effect of risk management measures for equine piroplasmosis is set out in Table 6.3.

Quarantine measures for equine piroplasmosis

To achieve Australia's ALOP with respect to the risk of equine piroplasmosis in permanently imported horses, the following quarantine measures are to be applied to all horses, including unweaned foals under six months of age.

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous two years and the disease is compulsorily notifiable.

OR

For all horses including unweaned foals under six months of age:

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous 60 days.

AND

The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

AND

On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

AND

If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

AND

During PEQ there has been no opportunity for iatrogenic transmission.

AND

Blood samples have been taken from the horse not less than seven days after commencement of PEQ and tested using an indirect fluorescent antibody test for *Babesia caballi* and *Theileria equi* as described in the OIE Manual for equine piroplasmosis with negative results in each case. If there is no approved laboratory in the country of export, testing in another country must be undertaken in a laboratory recognised by the Veterinary Authority of the country of export.

AND

The horse has not been treated with imidocarb, or other anti-babesial agents active against *B. caballi* and *T. equi*, for at least 60 days before commencement of PEQ.

AND

The horse has not been tested with any test for equine piroplasmosis (*B. caballi* or *T. equi*) with a positive result for at least 60 days before export.

AND

Within 24 hours of arrival at the PAQ facility, the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

AND

If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks and all horses in the facility must be tested for equine piroplasmosis at least 11 days after treatment for ticks.

Likelihood / Risk factor	Unrestricted	Restricted
Release and exposure assessment		
Likelihood of *release*	*Moderate*	*Very low*
Likelihood of *exposure*	*Moderate*	*Moderate*
Likelihood of *release and exposure*	***Low***	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	*Moderate*	*Moderate*
Overall effect of *establishment and/or spread*	*Moderate*	*Moderate*
Likely consequences	***Moderate***	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	**LOW**	**VERY LOW**

Table 6.3 Summary of unrestricted risk assessment and restricted risk estimations when risk management measures have been applied for equine piroplasmosis.

References

AQIS (1999) *Final import risk analysis report on the temporary importation of horses that are serologically positive for equine piroplasmosis for competition, exhibition or racing purposes*. Australian Quarantine and Inspection Service, Canberra.

Brüning A (1996) Equine piroplasmosis an update on diagnosis, treatment and prevention. *British Veterinary Journal* 152: 139-151.

Butler CM, Nijhof AM, van der Kolk JH, de Haseth OB, Taoufik A, Jongejan F, Houwers DJ (2008) Repeated high dose imidocarb dipropionate treatment did not eliminate *Babesia caballi* from naturally infected horses as determined by PCR-reverse line blot hybridization. *Veterinary Parasitology* 151: 320-322.

Callow LL (1984) Equine babesiosis in Australia. In *Protozoal and rickettsial diseases* pp. 165-167. Australian Government Publishing Service, Canberra.

CFSPH (2008) Equine piroplasmosis. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 28 January 2009).

de Waal DT (1992) Equine piroplasmosis: a review. *British Veterinary Journal* 148: 6-14.

Ellis P, Watkins KL (2004) International movement of athletic horses - quarantine and regulatory controls. In *Equine sports medicine and surgery* (eds. Hinchcliff KW, Geor RJ, Kaneps AJ) pp. 1227-1238. Elsevier, Amsterdam.

Friedhoff KT, Soulé C (1996) An account on equine babesiosis. *Revue Scientifique et Technique de l'Office International des Epizooties* 15: 1191-1201.

Friedhoff KT, Tenter AM, Muller I (1990) Haemoparasites of equines: impact on international trade of horses. *Revue Scientifique et Technique de l'Office International des Epizooties* 9: 1187-1194.

Gerstenberg C, Allen WR, Phipps LP (1999) Mechanical transmission of *Babesia equi* infection in a British herd of horses. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden OR) pp. 217-222, R & W Publications (Newmarket) Limited, Newmarket.

Kuttler KL, Goff WL, Gipson CA, Blackburn BO (1988) Serologic response of *Babesia equi*-infected horses as measured by complement-fixation and indirect fluorescent antibody tests. *Veterinary Parasitology* 26: 199-205.

Ogunremi O, Halbert G, Mainar-Jaime R, Benjamin J, Pfister K, Lopez-Rebollar L, Georgiadis MP (2008) Accuracy of an indirect fluorescent-antibody test and of a complement-fixation test for the diagnosis of *Babesia caballi* in field samples from horses. *Preventive Veterinary Medicine* 83: 41-51.

OIE (2008) Manual of diagnostic tests and vaccines for terrestrial animals 2008. World Organisation for Animal Health (OIE). http://www.oie.int/eng/normes/mmanual/A_summry.htm (Accessed 11 August 2008).

OIE (2009) Equine piroplasmosis, United States of America. World Animal Health Information System (WAHIS). http://www.oie.int/wahis/public.php?page=single_report&pop=1&reportid=8173 (Accessed 1 July 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1483-1540. Saunders Elsevier, Edinburgh.

Rothschild CM, Knowles DP (2007) Equine piroplasmosis. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 465-473. Saunders Elsevier, St. Louis.

Tenter AM, Friedhoff KT (1986) Serodiagnosis of experimental and natural *Babesia equi* and *B. caballi* infections. *Veterinary Parasitology* 20: 49-61.

6.2.3 Lyme disease

The unrestricted risk for Lyme disease associated with the importation of horses was estimated to be **LOW**. Risk management options that could be applied to achieve Australia's appropriate level of protection (ALOP) were considered by Biosecurity Australia.

The likelihood of release of Lyme disease was estimated to be '*moderate*' and the likelihood of exposure of Lyme disease was estimated to be '*low*'. The following options were considered as risk management measures to reduce the likelihood of release and/or exposure for Lyme disease.

Pre-entry measures

Country or area freedom

- Lyme disease is the most commonly reported tick-borne disease in humans in Asia, Europe and the United States (Steere et al. 2004).
- The geographic distribution of *Borrelia burgdorferi* senso lato (s.l.) infection in horses corresponds closely to the distribution of Lyme disease in humans (Burgess 1988).

Based on this information, country or area freedom was considered a risk management option for Lyme disease. Australia may consider an approved country free of Lyme disease if no clinical, epidemiological or other evidence of Lyme disease has been reported in the previous two years. Country freedom alone would be sufficient to achieve Australia's ALOP, subject to the horse having been continuously resident and free of quarantine restriction in the country for at least 60 days immediately before export.

Premises status

- Identification of infected premises would be difficult because infections are difficult to diagnose in horses.
- The incubation period of Lyme disease in horses is unknown but is from several months to years in other species.
- Infected horses can be subclinical and infection may persist for life (Chang et al. 2005).
- The geographic distribution of *B. burgdorferi* s.l. infection in horses corresponds closely to the distribution of Lyme disease in humans (Burgess 1988).

Based on this information, premises status was considered a risk management option for Lyme disease. Due to the potentially long incubation period and possible maintenance of *B. burgdorferi* s.l in ticks, a requirement for horses to have been continuously resident on premises for at least 60 days where no evidence of Lyme disease has occurred in any species, including humans, during the 90 days before export, was considered appropriate.

Due to subclinical infections and the difficulties in diagnosing infections, premises status alone was not considered sufficient to reduce the likelihood of release.

Vaccination

- There is no vaccine against *B. burgdorferi* s.l available for horses.

Based on this information, vaccination was not considered a risk management option for Lyme disease.

Pre-export quarantine

- The incubation period of Lyme disease in horses is unknown but is from several months to years in other species.
- A horse would be protected from infection in pre-export quarantine (PEQ) if there was no opportunity for iatrogenic spread or for exposure to ticks.

Based on this information, PEQ was considered a risk management option for Lyme disease. As most infections are subclinical and the incubation period in horses is unknown, a PEQ period for the purpose of detecting clinical signs of disease is not appropriate. A minimum 14-day PEQ period was considered sufficient for tick control and monitoring for clinical signs of disease.

Due to the unknown incubation period and the occurrence of latent carriers, PEQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- Diagnosis is complicated by the range of clinical signs of disease and the possibility of co-infection with other disease agents (Foley et al. 2004).
- Serological diagnosis is complicated by the long incubation period, presence of latent infections, cross-reactions with other spirochaetes, and persistence of antibody titres for months or years (CFSPH 2005).
- Seronegative cases been reported in infected humans following early antibiotic treatment (Dattwyler 1988).

Based on this information, diagnostic testing was not considered a risk management option for Lyme disease.

Preventive treatment(s)

- There is no effective treatment for Lyme disease in horses (Divers 2007).
- Visual inspection of horses for ticks, and treatment of horses with a parasiticide effective against ticks would reduce the risk of transmission before export.
- Nonvector transmission has been demonstrated experimentally in other species (Burgess et al. 1986). In horses, *B. burgdorferi* s.l. has been isolated from blood, urine and synovial fluid (Burgess 1988; Madigan 1993; Manion et al. 1998), transplacental transmission has been reported (Burgess et al. 1988; Burgess 1988) and iatrogenic transmission is possible (Parker and White 1992).

Based on this information, preventive treatment against ticks was considered a risk management option for Lyme disease.

Due to the potential for nonvector transmission, preventive treatment against ticks alone was not considered sufficient to reduce the likelihood of release.

Post-arrival measures

Preventive treatment(s)

- There is no effective treatment for Lyme disease in horses (Divers 2007).
- Visual inspection of horses for ticks, and treatment of horses with a parasiticide effective against ticks would reduce the risk of transmission.
- Nonvector transmission has been demonstrated experimentally in other species (Burgess et al. 1986). In horses, *B. burgdorferi* s.l. has been isolated from blood, urine and synovial fluid (Burgess 1988; Madigan 1993; Manion et al. 1998), transplacental transmission has been reported (Burgess et al. 1988; Burgess 1988) and iatrogenic transmission is possible (Parker and White 1992).

Based on this information, preventive treatment against ticks was considered a risk management option for Lyme disease.

Due to the potential for nonvector transmission, preventive treatment against ticks alone was not considered sufficient to reduce the likelihood of release.

Conclusion

The unrestricted risk associated with Lyme disease was estimated to be **LOW**.

Other than country freedom, no single risk management option reduced the unrestricted risk sufficiently to achieve Australia's ALOP. However, the combination of premises status and inspection and treatment for ticks pre-export and post-arrival would reduce the likelihood of release from '*moderate*' to '*low*'.

This would reduce the likelihood of release and exposure to '***very low***' and the restricted risk to at least **VERY LOW**, thereby achieving Australia's ALOP. A summary of the effect of risk management measures for Lyme disease is set out in Table 6.4.

Quarantine measures for Lyme disease

To achieve Australia's ALOP with respect to the risk of Lyme disease in imported horses, the quarantine measures are to be applied to all horses, including unweaned foals under six months of age.

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Lyme disease has occurred during the previous two years.

OR

For all horses including unweaned foals under six months of age:

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of Lyme disease has occurred in any species during the previous 90 days.

AND

The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

AND

On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

AND

If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

AND

Within 24 hours of arrival at the PAQ facility the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS Veterinarian, and no ticks found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

AND

If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks.

Likelihood / Risk factor	Unrestricted	Restricted
Release and exposure assessment		
Likelihood of *release*	*Moderate*	*Low*
Likelihood of *exposure*	*Low*	*Low*
Likelihood of *release and exposure*	***Low***	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	*Moderate*	*Moderate*
Overall effect of *establishment and/or spread*	*Moderate*	*Moderate*
Likely consequences	***Moderate***	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	**LOW**	**VERY LOW**

Table 6.4 Summary of unrestricted risk assessment and restricted risk estimations when risk management measures have been applied for Lyme disease.

References

Burgess EC (1988) *Borrelia burdgorferi* infection in Wisconsin horses and cows. *Annals of the New York Academy of Sciences* 539: 235-243.

Burgess EC, Amundson TE, Davis JP, Kaslow RA, Edelman R (1986) Experimental inoculation of *Peromyscus* spp. with *Borrelia burgdorferi*: evidence of contact transmission. *American Journal of Tropical Medicine and Hygiene* 35: 355-359.

Burgess EC, Gendron-Fitzpatrick A, Mattison M (1988) Foal mortality associated with natural infection of pregnant mares with *Borrelia burgdorferi*. In *Equine infectious diseases V: proceedings of the fifth international conference, October 7-10, 1987, Lexington, Kentucky*, (ed. Powell DG) pp. 217-220, The University Press of Kentucky, Lexington.

Chang Y-F, Ku Y-W, Chang C-F, Chang C-D, McDonough SP, Divers T, Pough M, Torres A (2005) Antibiotic treatment of experimentally *Borrelia burgdorferi*-infected ponies. *Veterinary Microbiology* 107: 285-294.

Divers TJ (2007) Lyme disease. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 310-312. Saunders Elsevier, St. Louis.

Foley JE, Foley P, Brown RN, Lane RS, Dumler JS, Madigan JE (2004) Ecology of *Anaplasma phagocytophilum* and *Borrelia burgdorferi* in the western United States. *Journal of Vector Ecology* 29: 41-50.

Madigan JE (1993) Lyme disease (Lyme borreliosis) in horses. *Veterinary Clinics of North America: Equine Practice* 9: 429-434.

Manion TB, Khan MI, Dinger J, Bushmich SL (1998) Viable *Borrelia burgdorferi* in the urine of two clinically normal horses. *Journal of Veterinary Diagnostic Investigation* 10: 196-199.

Parker JL, White KK (1992) Lyme borreliosis in cattle and horses: a review of the literature. *The Cornell Veterinarian* 82: 253-274.

Steere AC, Coburn J, Glickstein L (2004) The emergence of Lyme disease. *Journal of Clinical Microbiology* 113: 1093-1101.

6.2.4 Surra

The unrestricted risk for surra associated with the importation of horses was estimated to be **LOW**. Risk management options that could be applied to achieve Australia's appropriate level of protection (ALOP) were considered by Biosecurity Australia.

The likelihood of release of surra was estimated to be *'low'* and the likelihood of exposure of surra was estimated to be *'moderate'*. The following options were considered as risk management measures to reduce the likelihood of release and/or exposure for surra.

Pre-entry measures

Country or area freedom

- The United Arab Emirates is the only approved country in which surra is present (OIE 2009a). Area freedom for surra in the United Arab Emirates has not been assessed by Australia.
- Surra is a multiple species OIE-listed disease (OIE 2009c) and there are no recommendations in the Code. Surra is compulsorily notifiable in some approved countries, including the United Arab Emirates (OIE 2009a).
- Adaption of *Trypanosoma evansi* to mechanical transmission by biting flies has resulted in it being widely distributed across Asia, Africa (north of the tsetse belt), Central and South America, and the Middle East (Luckins 1994; Radostits et al. 2007; OIE 2008).
- Tabanids have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000). A distance of 200 metres between infected and susceptible animals has been recommended as an effective margin to minimise risks of mechanical transmission by tabanids (Barros and Foil 2007).
- Infection with *T. evansi* causes acute and chronic disease, loss of productivity and possibly death in a wide range of susceptible animals including cattle, goats, sheep, water buffalo, camels, llamas, cats, dogs, donkeys, horses, mules, pigs, elephants, wallabies and potentially other wildlife species. Naïve animals are more likely to develop acute disease (Sellon 2007).
- The incubation period of surra in horses is usually 1–2 weeks, but can be up to 60 days, and clinical signs of disease are not pathognomonic (Losos 1980; Gardiner and Mahmoud 1992; Geering et al. 1995; Sellon 2007).
- Horses with surra may not show clinical signs of infection or may be latent carriers (Utami 1996; Abo-Shehada et al. 1999). Naïve animals are more likely to develop acute disease (Sellon 2007).

Based on this information, country freedom was considered a risk management option for surra. Australia may consider an approved country free of surra if no clinical, epidemiological or other evidence of surra has been reported in the previous 12 months. If a horse became infected just before entering an approved country free of surra, it is likely that infection would become apparent within 60 days, either in that

horse, or in other susceptible animals. Country freedom of approved countries alone would be sufficient to achieve Australia's ALOP.

Premises status

- A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids can feed on two different horses 51.7 metres apart during a single interrupted feed (Foil 1983) and have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000).
- A distance of 200 metres between infected and susceptible animals has been recommended as an effective margin to minimise the risks of mechanical transmission by tabanids (Barros and Foil 2007).
- The incubation period of surra in horses is usually 1–2 weeks, but can be up to 60 days, and clinical signs of disease are not pathognomonic (Losos 1980; Gardiner and Mahmoud 1992; Geering et al. 1995; Sellon 2007).
- Infection with *T. evansi* causes acute and chronic disease, loss of productivity and possibly death in a wide range of susceptible animals including cattle, goats, sheep, water buffalo, camels, llamas, cats, dogs, donkeys, horses, mules, pigs, elephants, wallabies and potentially other wildlife species. Naïve animals are more likely to develop acute disease (Sellon 2007).
- Horses with surra may not show clinical signs of infection or may be latent carriers (Utami 1996; Abo-Shehada et al. 1999). Naïve animals are more likely to develop acute disease (Sellon 2007).

Based on this information, premises status was considered a risk management option for surra. Due to the potentially long incubation period, a requirement for horses to have been continuously resident on premises for at least 60 days where no evidence of surra has occurred in any species during the 12 months before export was considered appropriate.

Due to the incidence of subclinical or latent infection, premises status alone was not considered sufficient to reduce the likelihood of release.

Vaccination

- Existence of several antigenic types has restricted the development of a vaccine for surra. Experimental studies on laboratory animals have developed a protective immunogen from *T. evansi* that may lead to a potential vaccine (Li et al. 2007).
- There are no vaccines against *T. evansi.*

Based on this information, vaccination was not considered a risk management option for surra.

Pre-export quarantine

- The incubation period of surra in horses is usually 1–2 weeks, but can be up to 60 days, and clinical signs of disease are not pathognomonic (Losos 1980; Gardiner and Mahmoud 1992; Geering et al. 1995; Sellon 2007).

- Horses with surra may not show clinical signs of infection or may be latent carriers (Utami 1996; Abo-Shehada et al. 1999). There is no known correlation between prevalence and age, breed or gender. Naïve animals are more likely to develop acute disease (Sellon 2007).
- *T. evansi* is transmitted by biting flies (Luckins 1994). A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids can travel up to 51.7 metres to feed on two different animals during a single interrupted feed (Foil 1983) and have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000).
- A distance of 200 metres between infected and susceptible animals has been recommended as an effective margin to minimise the risks of mechanical transmission by tabanids (Barros and Foil 2007).
- A wide range of animals are susceptible to surra and include cattle, goats, sheep, water buffalo, camels, llamas, cats, dogs, donkeys, horses, mules, pigs, elephants, wallabies and potentially other wildlife species (Sellon 2007).

Based on this information, pre-export quarantine (PEQ) was considered a risk management option for surra to isolate and separate horses from the domestic population of susceptible animals in which the disease is endemic. Most infections have an incubation period of 1–2 weeks, therefore a PEQ period of at least 21 days was considered appropriate to enable monitoring for clinical signs of surra and vector control. To help prevent infection during PEQ, the PEQ facility must be located and managed so that for the duration of PEQ other susceptible animals (not of equivalent health status) are not held, housed or exercised within 200 metres of horses in PEQ.

Due to the long incubation period in some cases (up to 60 days) and the occurrence of subclinical or latent infection, PEQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- The OIE describes direct and indirect tests for surra (OIE 2008); however, there are no prescribed or alternative diagnostic techniques recommended by the OIE for surra (OIE 2009b).
- The ability of direct and indirect tests to detect infection varies depending on the stage of infection. Direct parasitological tests are recommended to confirm a clinical diagnosis (Sellon 2007; Radostits et al. 2007). The microhaematocrit centrifugation technique is considered the most reliable direct test (Murray et al. 1977; OIE 2008) and is most consistent if used to detect the presence of trypanosomes from 10 days post-infection (Wernery et al. 2001).
- Detection of infection in animals with subclinical or latent infection is difficult. Direct techniques are unlikely to detect infection in horses with chronic disease, subclinical or latent infection (Reid et al. 2001). Several indirect techniques to detect *T. evansi* antigens or antibodies are available, but have variable sensitivity and specificity (Monzon et al. 1995; Luckins 1999; Wernery et al. 2001). An antibody-detection enzyme-linked immunosorbent assay (Ab-ELISA) was

validated for use in horses with a sensitivity of 95.5% and a specificity of 98% (Monzon 2000).

- Serum antibodies are first detectable 10–19 days post infection (Wernery et al. 2001) and can persist for up to 22.6 months in horses that have been successfully treated for surra (Monzon et al. 2003).
- Polymerase chain reaction amplification tests have been described for the detection of *T. evansi* DNA. While the sensitivity and specificity are greater than other tests, they have not been validated for use in horses (Wuyts et al. 1995; Sellon 2007; OIE 2008).
- There is no known correlation between prevalence of surra in horses and age, breed or gender (Sellon 2007).

Based on this information, diagnostic testing of all horses (including unweaned foals) was considered a risk management option for surra. For diagnostic testing to be useful, it needs to be accompanied by isolation from susceptible animals not of equivalent health status and protection from biting flies for at least ten days before samples are collected for testing until export, to prevent subsequent transmission of *T. evansi* to horses being tested. To detect both recently and chronically infected horses, testing using microhaematocrit centrifugation technique and Ab-ELISA, consistent with those described by the OIE, at no less than ten days following commencement of isolation was considered the most effective diagnostic measure.

Due to the limited and variable sensitivity of diagnostic tests and the occurrence of subclinical or latent infection, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- *T. evansi* is transmitted by biting flies (Luckins 1994). A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids can travel up to 51.7 metres to feed on two different animals during a single interrupted feed (Foil 1983) and have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000).
- There are conflicting reports on efficacy and curative doses of available pharmacological treatments for surra (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).
- Persistent infection can occur in animals treated during the later stages of infection (Luckins 1994; Wernery et al. 2001).
- There is evidence of resistance to trypanocidal drugs (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).
- Adverse reactions to treatment vary from moderate to severe and can occur in up to 50% of horses (Tuntasuvan et al. 2003; Radostits et al. 2007).
- Effectiveness and duration of surface insecticidal sprays and insect repellents when applied to stables or horses in repelling biting flies from biting horses is

largely unknown; however, it can assist in reducing the likelihood of a horse being bitten.

Based on this information, preventive treatment using trypanocidal drugs was not considered a risk management option for surra. Treatment has limited efficacy, especially when administered during the later stages of infection and causes moderate to severe adverse reactions in horses.

Treatment for the control of biting flies was considered a risk management option for surra. Keeping horses isolated in insect-screened stables and treating horses during isolation with insect repellent before leaving insect-screened stables would reduce the potential for horses being infected. Disinsection of vehicles for transporting horses to the port of export was also considered a risk management option for surra.

Due to the occurrence of subclinical or latent infection in horses and potential for resistance to insecticides and insect repellents, insect control alone was not considered sufficient to reduce the likelihood of release.

Post-arrival measures

Post-arrival quarantine

- The incubation period of surra in horses is usually 1–2 weeks but can be up to 60 days and clinical signs of disease are not pathognomonic (Losos 1980; Gardiner and Mahmoud 1992; Geering et al. 1995; Sellon 2007).
- Horses with surra may not show clinical signs of infection or may be latent carriers (Utami 1996; Abo-Shehada et al. 1999). There is no known correlation between prevalence and age, breed or gender (Sellon 2007).
- Horses are more susceptible to developing surra if stressed, such as a result of aircraft travel (OIE 2008).
- *T. evansi* is transmitted by biting flies (Luckins 1994). A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids can travel up to 51.7 metres to feed on two different animals during a single interrupted feed (Foil 1983) and have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000).

Based on this information, post-arrival quarantine (PAQ) was considered a risk management option for surra. If a horse has subclinical or latent infection that was not detected via observation or testing during PEQ, the stress of aircraft travel could trigger the onset of disease. A PAQ period of 14 days was considered appropriate to enable observation for clinical signs of surra for a usual incubation period. Due to the potentially long incubation period (up to 60 days) and the occurrence of subclinical or latent infection in horses, PAQ alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- *T. evansi* is transmitted by biting flies (Luckins 1994). A feeding time of five seconds is sufficient for a tabanid to acquire infection from an infected host, or transmit infection to a susceptible animal (Luckins 1999). Tabanids can travel up

to 51.7 metres to feed on two different animals during a single interrupted feed (Foil 1983) and have a home range over 6.4 km (Sheppard et al. 1973; Chippaux et al. 2000).

- There have been conflicting reports on efficacy and curative doses of available pharmacological treatments for surra (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).
- Persistent infection can occur in animals treated during the later stages of infection (Luckins 1994; Wernery et al. 2001).
- There is evidence of resistance to trypanocidal drugs (Boid et al. 1996; Gillingwater et al. 2007; Radostits et al. 2007).
- Adverse reactions to treatment vary from moderate to severe and can occur in up to 50% of horses (Tuntasuvan et al. 2003; Radostits et al. 2007).
- Effectiveness and duration of surface insecticidal sprays and insect repellents when applied to stables or horses in repelling biting flies from biting horses is largely unknown; however, it can assist in reducing the likelihood of a horse being bitten.

Based on this information, preventive treatment using trypanocidal drugs was not considered a risk management option for surra. Treatment has limited efficacy, especially when administered during the later stages of infection, and causes moderate to severe adverse reactions in horses.

Treatment for the control of biting flies was considered a risk management option for surra. Spraying stables with a residual insecticide immediately before horses are isolated and treatment of horses with an insect repellent would reduce the potential transmission of infection.

Due to the occurrence of subclinical or latent infection in horses and potential for resistance to insecticides and repellents, insect control alone was not considered sufficient to reduce the likelihood of release.

Conclusion

The unrestricted risk associated with surra was estimated to be **LOW**.

Other than country freedom, no single risk management option reduced the unrestricted risk sufficiently to achieve Australia's ALOP. However, the combination of premises status, PEQ, pre-export diagnostic testing, PAQ and pre-export and post-arrival preventive treatment (to prevent flies biting horses) would reduce the likelihood of release for surra from '*low*' to '*very low*'.

This would reduce the likelihood of release and exposure to '***very low***' and the restricted risk to at least **VERY LOW**, thereby achieving Australia's ALOP. A summary of the effect of risk management measures for surra is set out in Table 6.5.

Quarantine measures for surra

To achieve Australia's ALOP with respect to the risk of surra in imported horses, the following quarantine measures are to be applied to all horses, including unweaned foals under six months of age.

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred during the previous 12 months.

OR

For all horses including unweaned foals under six months of age:

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred in equids during the previous 12 months.

AND

For 60 days immediately before export the horse has not resided on premises in the country of export where there has been clinical, epidemiological or other evidence of surra in any species during the previous 12 months.

AND

The horse has been held in PEQ for at least 21 days immediately before export.

AND

The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of surra has occurred in equids for 12 months before export.

AND

During PEQ the horse has been isolated and not held, housed or exercised within 200 metres of ruminants or camelids.

AND

During PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting flies before leaving the stables.

AND

Blood samples have been taken from the horse not less than ten days after commencement of PEQ and tested using an antibody-detection enzyme-linked immunosorbent assay and microhaematocrit centrifugation technique as described in the OIE Manual for surra (*Trypanosoma evansi*) with negative results in each case.

AND

The horse has been treated with an insect repellent for protection from biting flies before leaving the stable and being loaded into the vehicle for transporting horses from the PEQ facility to the port of export, and after loading the vehicle was disinsected.

AND

The horse must be held in PAQ for at least 14 days.

AND

Stables at the PAQ facility must have been sprayed with a residual insecticide (e.g. synthetic pyrethroid) during the 24 hours before the horse arrives at the facility. For the duration of PAQ the horse must be treated with insect repellent according to manufacturer's recommendations for protection from biting flies.

Likelihood / Risk factor	**Unrestricted**	**Restricted**
Release and exposure assessment		
Likelihood of *release*	*Low*	*Very low*
Likelihood of *exposure*	*Moderate*	*Moderate*
Likelihood of *release and exposure*	***Low***	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	*Moderate*	*Moderate*
Overall effect of *establishment and/or spread*	*Moderate*	*Moderate*
Likely consequences	***Moderate***	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	**LOW**	**VERY LOW**

Table 6.5 Summary of unrestricted risk assessment and restricted risk estimations when risk management measures have been applied for surra.

References

Abo-Shehada MN, Anshassi H, Mustafa G, Amr Z (1999) Prevalence of surra among camels and horses in Jordan. *Preventive Veterinary Medicine* 38: 289-293.

Barros ATM, Foil LD (2007) The influence of distance on movement of tabanids (Diptera: Tabanidae) between horses. *Veterinary Parasitology* 144: 380-384.

Boid R, Hunter AG, Jones TW, Ross CA, Sutherland D, Luckins AG (1996) Trypanosomosis research at the Centre for Tropical Veterinary Medicine (CTVM) 1970 to 1995. *Tropical Animal Health and Production* 28: 5-22.

Chippaux J-P, Bouchite B, Demanou M, Morlais I, Le Goff G (2000) Density and dispersal of the loaiasis vector *Chrysops dimidiata* in southern Cameroon. *Medical and Veterinary Entomology* 14: 339-344.

Foil L (1983) A mark-recapture method for measuring effects of spatial separation of horses on tabanid (Diptera) movement between hosts. *Journal of Medical Entomology* 20: 301-305.

Gardiner PR, Mahmoud MM (1992) Salivarian trypanosomes causing disease in livestock outside sub-Saharan Africa. In *Parasitic protozoa* (eds. Kreier JP, Baker JR) pp. 277-314. Academic Press, San Diego.

Geering WA, Forman AJ, Nunn MJ (1995) Surra. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 380-384. Australian Government Publishing Service, Canberra.

Gillingwater K, Buscher P, Brun R (2007) Establishment of a panel of reference *Trypanosoma evansi* and *Trypanosoma equiperdum* strains for drug screening. *Veterinary Parasitology* 148: 114-121.

Li S-Q, Fung M-C, Reid SA, Inoue N, Lun Z-R (2007) Immunization with recombinant beta-tubulin from *Trypanosoma evansi* induced protection against *T. evansi*, *T. equiperdum* and *T. b. brucei* infection in mice. *Parasite Immunology* 29: 191-199.

Losos GJ (1980) Diseases caused by *Trypanosoma evansi*: a review. *Veterinary Research Communications* 4: 165-181.

Luckins AG (1994) Equine trypanosomiasis. *Equine Veterinary Education* 6: 259-262.

Luckins AG (1999) Equine trypanosomiases: a review. In *Equine infectious diseases VIII: proceedings of the eighth international conference, March 23-26, 1998, Dubai, United Arab Emirates,* (eds. Wernery U, Wade JF, Mumford JA, Kaaden O.R.) pp. 237-242, R & W Publications (Newmarket) Limited, Newmarket.

Monzon CM (2000) Validation of an indirect immunoenzyme assay for the detection of antibodies against *Trypanosoma evansi* in horses in Argentina. *Revue Scientifique et Technique de l'Office International des Epizooties* 19: 810-818.

Monzon CM, Jara A, Nantulya VM (1995) Sensitivity of antigen ELISA test for detecting *Trypanosoma evansi* antigen in horses in subtropical area of Argentina. *The Journal of Parasitology* 81: 806-808.

Monzon CM, Mancebo OA, Russo AM (2003) Antibody levels by indirect ELISA test in *Trypanosoma evansi* infected horses following treatment with quinapyramine sulphate. *Veterinary Parasitology* 1111: 59-63.

Murray M, Murray PK, McIntyre WIM (1977) An improved parasitological technique for the diagnosis of African trypanosomiasis. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 71: 325-326.

OIE (2008) *Trypanosoma evansi* infections (including surra). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.17_TRYPANO.pdf (Accessed 1 September 2009).

OIE (2009a) Animal health situation: United Arab Emirates. WAHID Interface: Animal Health Information. http://www.oie.int/wahis/public.php?page=country_status&year=2008 (Accessed 29 May 2009).

OIE (2009b) List of tests for international trade. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/0.02_PRESCRIBED_TESTS_2008.pdf (Accessed 3 September 2009).

OIE (2009c) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with protozoa. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1483-1540. Saunders Elsevier, Edinburgh.

Reid SA, Husein A, Copeman DB (2001) Evaluation and improvement of parasitological tests for *Trypanosoma evansi* infection. *Veterinary Parasitology* 102: 291-297.

Sellon DC (2007) Miscellaneous parasitic diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 473-480. Saunders Elsevier, St. Louis.

Sheppard DC, Wilson B, Hawkins JA (1973) A device for self-marking of Tabanidae. *Environmental Entomology* 2: 960-961.

Tuntasuvan D, Jarabrum W, Viseshakul N, Mohkaew K, Borisutsuwan S, Theeraphan A, Kongkanjana N (2003) Chemotherapy of surra in horses and mules with diminazene aceturate. *Veterinary Parasitology* 110: 227-233.

Utami S (1996) Survey of trypanosome species in cattle, buffaloes and horses in three districts of Lombok. In *Proceedings of a seminar in diagnostic techniques for Trypanasoma evansi in Indonesia,* pp. 54-57, Research Institute for Veterinary Science, Bogor.

Wernery U, Zachariah R, Mumford JA, Luckins T (2001) Preliminary evaluation of diagnostic tests using horses experimentally infected with *Trypanosoma evansi*. *The Veterinary Journal* 161: 287-300.

Wuyts N, Chokesajjawatee N, Sarataphan N, Panyim S (1995) PCR amplification of crude blood on microscope slides in the diagnosis of *Trypanosoma evansi* infection in dairy cattle. *Annales de la Société Belge de Médecine Tropicale* 75: 229-237.

6.2.5 Vesicular stomatitis

The unrestricted risk for vesicular stomatitis (VS) associated with the importation of horses was estimated to be **LOW**. Risk management options that could be applied to achieve Australia's appropriate level of protection (ALOP) were considered by Biosecurity Australia.

The likelihood of release of VS was estimated to be '*low*' and the likelihood of exposure of VS was estimated to be '*moderate*'. The following options were considered as risk management measures to reduce the likelihood of release and/or exposure for VS.

Pre-entry measures

Country or area freedom

- VS is limited to the Americas (OIE 2008a). VS outbreaks occur sporadically in the United States (Rodriguez 2002). In limited areas of the United States, VS is endemic and occurs seasonally (McCluskey and Mumford 2000).
- Horses visiting multiple countries within a relatively short period occurs regularly (Ellis and Watkins 2004).
- According to the Code, the incubation period for VS is considered to be 21 days (OIE 2009).
- Shedding of virus from an active lesion is thought to stop 6–7 days after lesions form (McCluskey and Mumford 2000).
- The OIE considers a country free from VS when VS is notifiable in the country and no clinical, epidemiological or other evidence of VS has been found during the past two years (OIE 2009).

Based on this information, country freedom was considered a risk management option for VS. Country freedom of approved countries alone, using the Code recommendation of a country free from VS, would be sufficient to achieve Australia's ALOP.

Premises status

- VS is compulsorily notifiable in the United States and Canada (OIE 2008b).
- Subclinically infected horses are common during outbreaks (Mumford et al. 1998a; Mumford et al. 1998b) but latent infection is not known to occur (McCluskey and Mumford 2000).
- Spread of VS during an outbreak is irregular. During outbreaks, a majority of positive identified premises are not contiguous with other positive premises (McCluskey et al. 1999).
- According to the Code, the incubation period for VS is considered to be 21 days (OIE 2009).
- Shedding of virus from an active lesion is thought to stop 6–7 days after lesions form (McCluskey and Mumford 2000).

- The United States Department of Agriculture's VS control strategy for an outbreak indicates vector control (from biting and non-biting insects) and a 21-day quarantine (after the last animal's lesions have healed and other susceptible animals are found free of lesions) for case-positive premises (Clifford 2007).

Based on this information, premises status was considered a risk management option for VS. Due to the uncertain epidemiology of VS, a requirement for horses to have been resident on premises for 30 days where no clinical, epidemiological or other evidence of VS has occurred during the previous 90 days in any species was considered appropriate.

Due to subclinical infections being common, premises status alone was not considered sufficient to reduce the likelihood of release.

Vaccination

- Vaccines are not commercially available in the United States (McCluskey and Mumford 2000). Killed vaccines are manufactured in Colombia and Venezuela for the Indiana and New Jersey serotypes of VS (OIE 2008a).

Based on this information, vaccination was not considered a risk management option for VS.

Pre-export quarantine

- According to the Code, the incubation period for VS is considered to be 21 days (OIE 2009). In the literature, the incubation period of VS is 1–3 days (Letchworth 1996).
- Subclinically infected horses are common during outbreaks (Mumford et al. 1998a; Mumford et al. 1998b) but latent infection is not known to occur (McCluskey and Mumford 2000).
- Shedding of virus from an active lesion is thought to stop 6–7 days after lesions form (McCluskey and Mumford 2000). Persistence or shedding of infective VS virus from recovered animals is not known to occur (McCluskey and Mumford 2000).
- The epidemiology of VS is not well understood. Management factors suggested to reduce the risk of horses becoming infected during outbreaks include removal of horses from pastures; providing access to shelter or barns, particularly evenings and overnight; moving horses away from running water; and reducing contact with flying insects (McCluskey and Mumford 2000).

Based on this information, pre-export quarantine (PEQ) was considered a risk management option for VS. Due to the uncertain epidemiology of VS, the PEQ facility should be located in a defined area where no clinical, epidemiological or other evidence of VS has occurred during the previous 90 days in any species. During the period of PEQ, horses should be isolated from animals not of equivalent health status to minimise the risk of introducing VS into the PEQ facility.

A PEQ period of at least 14 days provides the opportunity for isolation and observation and allows sufficient time to cover the infectious period. Due to the uncertain epidemiology of VS, PEQ alone was not considered sufficient to reduce the likelihood of release.

Diagnostic testing

- It is common to have variable antibody responses among affected animals (McCluskey and Mumford 2000).
- OIE prescribed tests for international trade are liquid-phase blocking enzyme-linked immunosorbent assay (LP-ELISA), competitive ELISA (C-ELISA), virus neutralisation (VN) test and complement fixation test (OIE 2008a).
- The ELISA and VN test are preferable for identifying and quantifying specific antibodies in serum (OIE 2008a).
- Antibodies can usually be detected 5–8 days post-infection (OIE 2008a).

Based on this information, diagnostic testing was considered a risk management option for VS. For diagnostic testing to be useful, horses need to be isolated before export from animals not of equivalent health status to prevent potential subsequent transmission of VS to horses being tested. To identify recently infected horses, blood samples for ELISA or VN testing, as described in the OIE Manual for VS (both New Jersey and Indiana strains), should be taken from all horses at least eight days after commencement of a pre-export isolation period to allow time for antibodies to develop. Due to the imperfect sensitivity of diagnostic tests, diagnostic testing alone was not considered sufficient to reduce the likelihood of release.

Preventive treatment(s)

- No specific treatment is indicated for VS (McCluskey and Mumford 2000).

Based on this information, preventative treatment was not considered a risk management option for VS.

Conclusion

The unrestricted risk associated with VS was estimated to be **LOW**.

Other than country freedom, no single risk management option reduced the unrestricted risk sufficiently to achieve Australia's ALOP. However, the combination of premises status, PEQ and pre-export diagnostic testing would reduce the likelihood of release for VS from '*low*' to '*very low*'.

This would reduce the likelihood of release and exposure to '***very low***' and the restricted risk to at least **VERY LOW**, thereby achieving Australia's ALOP. A summary of the effect of risk management measures for VS is set out in Table 6.6.

Quarantine measures for vesicular stomatitis

To achieve Australia's ALOP with respect to the risk of VS in imported horses, the following quarantine measures are recommended.

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

OR

For 30 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous 90 days and the disease is compulsorily notifiable.

AND

The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids and domestic livestock not of equivalent health status.

AND

The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species for 90 days before export.

AND

A blood sample has been taken from the horse not less than eight days after commencement of PEQ and tested using an enzyme-linked immunosorbent assay or virus neutralisation test as described in the OIE Manual for vesicular stomatitis (both New Jersey and Indiana strains) with negative results.

Likelihood / Risk factor	Unrestricted	Restricted
Release and exposure assessment		
Likelihood of *release*	*Low*	*Very low*
Likelihood of *exposure*	*Moderate*	*Moderate*
Likelihood of *release and exposure*	***Low***	***Very low***
Consequence assessment		
Likelihood of *establishment and/or spread*	*Moderate*	*Moderate*
Overall effect of *establishment and/or spread*	*Moderate*	*Moderate*
Likely consequences	***Moderate***	***Moderate***
Risk estimation		
The risk of *release, exposure, establishment and/or spread*	**LOW**	**VERY LOW**

Table 6.6 Summary of unrestricted risk assessment and restricted risk estimations when risk management measures have been applied for VS.

References

Clifford JR (2007) Veterinary Services memorandum no. 555.17: vesicular stomatitis control strategy. United States Department of Agriculture, Animal and Plant Health Inspection Agency, Veterinary Services. http://www.aphis.usda.gov/animal_health/nahln/surveillance_VSV.shtml (Accessed 1 July 2009).

Ellis P, Watkins KL (2004) International movement of athletic horses - quarantine and regulatory controls. In *Equine sports medicine and surgery* (eds. Hinchcliff KW, Geor RJ, Kaneps AJ) pp. 1227-1238. Elsevier, Amsterdam.

Letchworth GJ (1996) Vesicular stomatitis. In *Virus infections of equines* (ed. Studdert MJ) pp. 265-279. Elsevier Science Publishers, Amsterdam.

McCluskey BJ, Hurd HS, Mumford EL (1999) Review of the 1997 outbreak of vesicular stomatitis in the western United States. *Journal of the American Veterinary Medical Association* 215: 1259-1262.

McCluskey BJ, Mumford EL (2000) Vesicular stomatitis and other vesicular, erosive, and ulcerative diseases of horses. *Emerging Infectious Diseases* 16: 457-469.

Mumford EL, McCluskey BJ, Traub-Dargatz JL, Schmitt B, Salman MD (1998a) Vesicular stomatitis: epidemiology of the 1995 and 1997 outbreaks in Colorado. In *Proceedings of the 44th annual convention of the AAEP, December 6-9, 1998, Baltimore, Maryland,* pp. 23-25, American Association of Equine Practitioners, Lexington.

Mumford EL, McClusky BJ, Traub-Dargatz JL, Schmitt BJ, Salman MD (1998b) Serologic evaluation of vesicular stomatitis virus exposure in horses and cattle in 1996. *Journal of the American Veterinary Medical Association* 213: 1265-1269.

OIE (2008a) Vesicular stomatitis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.01.19_VESICULAR_STOMITIS.pdf (Accessed 1 September 2009).

OIE (2008b) WAHID interface. World Animal Health Information Database: version 1.2. http://www.oie.int/wahid-prod/public.php?page=home (Accessed 14 November 2008).

OIE (2009) Vesicular stomatitis. Terrestrial Animal Health Code 2009. http://www.oie.int/eng/normes/mcode/en_chapitre_1.8.15.htm (Accessed 1 September 2009).

Rodriguez LL (2002) Emergence and re-emergence of vesicular stomatitis in the United States. *Virus Research* 85: 211-219.

7 Transport from approved countries

Horses travelling from approved countries to Australia can potentially be exposed to disease agents of quarantine concern during transport from the pre-export quarantine (PEQ) facility and during transit or transhipment. Imported horses may present a quarantine risk to susceptible animals in Australia during transport from an Australian airport to the post-arrival quarantine (PAQ) facility.

Transport of horses from approved countries to Australia is from departure from the PEQ facility until arrival at a PAQ facility in Australia. The Veterinary Authority of the exporting country must take steps to ensure that appropriate biosecurity procedures are in place and observed during the transport of horses from the PEQ facility to the approved airport.

Transport from the PEQ facility to the approved port of export can be undertaken by road or sea. Where such transport crosses an international border, Veterinary Authorities of each country must provide certification that Australia's quarantine requirements for the transport of horses have been met. All airports in approved countries are approved for the transit and transhipment of horses travelling to Australia.

Transit is when horses remain on an aircraft during a scheduled landing at an approved airport en route to Australia. Transhipment is when horses are unloaded from one aircraft then loaded onto another or are unloaded and reloaded onto the same aircraft for any reason, at an approved airport. Horses must not be removed from their air stalls during transit or transhipment.

Disease agents of quarantine concern during transport of horses to Australia include arthropod vectors and screw-worm-flies. Exposure of horses to insects outside the aircraft during transhipment presents a potential risk. In addition, open aircraft doors during loading and unloading of cargo may increase the likelihood of exposure to such disease agents. Fomites also present a potential source of exposure to disease agents of quarantine concern. Risk management options are intended to reduce the likelihood of exposure of horses to disease agents of quarantine concern during transport to Australia.

Transport requirements also consider measures that must be taken in case of unexpected delays or unscheduled diversions of vehicles for transporting horses and aircrafts. If an aircraft makes an unscheduled landing in a country other than an approved country, the Australian Quarantine and Inspection Service (AQIS) must be notified immediately, and it will be determined on a risk-based case-by-case basis whether the horses can continue to travel to Australia.

Transport requirements include measures that must be taken after arrival in Australia.

7.1 General pre-export transport requirements

Exporters or their agents must have detailed Standard Operating Procedures (SOPs) consistent with a risk-based approach and approved by AQIS, specifying procedures for transporting the horses from PEQ until arrival in Australia. The SOPs must include the following:

- cleaning and disinfection of vehicles for transporting horses before being used by horses
- veterinary equipment and consumables carried on the flight
- grooms accompanying horses on the aircraft and their personal effects
- veterinarians accompanying horses on the aircraft and their personal effects
- cleaning and disinfection of horse air stalls and ramps before being used by horses
- supply and use of insect repellents and knockdown aerosol insecticide spray for use on the aircraft
- supply and use of insect netting for use in the aircraft
- contingency plans for the management of delayed takeoffs and unscheduled landings
- contingency plans for vehicle and equipment failures
- if transport from the PEQ facility to the approved airport crosses an international border, contingency plans to address the potential for exposure to disease agents of quarantine concern must include how the risk is addressed and minimised in each country. Contingency plans must include arrangements to notify the Veterinary Authorities of all countries through which a consignment travels en route to the airport of export.

All personnel involved in the transport of horses (e.g. exporters and their agents, official veterinarians, grooms, transport operators, and grooms and veterinarians accompanying horses on the aircraft) must be familiar with relevant SOPs.

All feed accompanying the horses to Australia must enter the PEQ facility at the beginning of the PEQ period. The use of hay or straw as bedding during transport to Australia is not permitted. Treated wood shavings, sterilised peat and soft board can be used.

All PEQ facilities must be located within 250 km of an approved airport to reduce the likelihood of exposure to disease agents of quarantine concern en route and minimise travel stress. The transport route from the PEQ facility to the approved airport must be approved by the Official Veterinarian. Where the distance and duration of travel to the approved airport exceeds the time specified by legislation in the exporting country for a non-stop journey, approval to stop and rest the horses must be obtained from AQIS as part of the PEQ facility approval. In such cases, the exporter must provide specific contingency plans for potential exposure to fomites, vectors, equids or other animals not of the equivalent health status or to personnel not involved in the transport of horses, as well as plans for the provision of feed and water.

The Official Veterinarian must be present during loading of horses when leaving the PEQ facility to ensure vehicles for transporting horses are adequately cleaned and disinfected, to supervise sealing of vehicles for transporting horses with tamper-evident seals before departure from the PEQ facility and to certify that the horses are fit to travel. A government officer authorised by the Veterinary Authority must be available at the airport to check the vehicle seals are intact on arrival and ensure ramps and air stalls are adequately cleaned and disinfected.

Horses must remain isolated from all animals not of equivalent health status and be protected from insect vectors during transport from the PEQ facility until arrival in Australia. All personnel likely to be in direct contact with the horses during transport to Australia (including transport from the PEQ facility to the airport, at the airport, and on the aircraft) must shower and wear new or clean protective clothing and footwear before coming into contact with the horses. They must not have any contact with horses not of equivalent health status during transport to Australia.

The design of air stalls, the recommended requirements for horses, the preparation for transport, disinsection of the interior of the aircraft, disinfection of removable equipment, penning and air stalls, including loading ramps, must be in accordance with the recommendations of the Code and International Air Transport Association (IATA) Live Animal Regulations unless otherwise agreed by AQIS.

Insect netting must be carried on the flight at all times for contingencies. There must be sufficient insect netting to cover all air stalls completely. Insect netting must be in good condition to minimise entry of insect vectors into the air stalls.

An Australian government veterinarian may be required to accompany the shipment to Australia at the importer's expense. AQIS must receive adequate notice of the intention to import so that arrangements can be made.

The consignment may be accompanied by other horses of equivalent health status or animals of other species only with the prior approval of AQIS.

7.2 Transit and transhipment procedures

Horses must transit or tranship only at an approved airport. Any transhipment requires the prior approval of AQIS. Stops en route to Australia will need approval and permits from relevant authorities in the countries of transit and transhipment. Transit and transhipment times must not exceed six hours. Horses are not to leave the airport and must not be removed from their air stalls during transit or transhipment.

Horses must remain on board the aircraft at approved transit airports. Unauthorised personnel must not have contact with the horses. Cargo doors can be opened at approved transit airports to allow for unloading or loading of freight. Immediately after the cargo hold doors are closed, an approved knockdown aerosol insecticide must be sprayed throughout the cargo hold, in the manner recommended by the manufacturer.

In cases where horses in air stalls are to be unloaded, before opening the cargo door, the air stalls must be completely covered in netting to minimise insect access to the horses. The netting must remain in place until the horses are reloaded on an aircraft. Immediately after the horses are reloaded on an aircraft and the cargo hold doors are closed, an approved knockdown aerosol insecticide spray must be sprayed throughout the cargo hold in the manner recommended by the manufacturer. The insect netting must not be removed until 30 minutes after spraying.

7.3 Procedures for delayed takeoffs and unscheduled landings

In the event that transit or transhipment exceeds six hours, AQIS must be notified immediately and the horses must not proceed to Australia without approval from AQIS. If the aircraft lands at any airport other than in an approved country, AQIS must be informed immediately. The decision as to whether the horses can continue to travel to Australia, and additional quarantine measures that may be required, will be made by AQIS on a risk-based case-by-case basis.

7.4 Procedures after arrival in Australia

Importers or their agents must have detailed SOPs specifying post-arrival procedures. These SOPs are to be developed in consultation with AQIS and must include roles and responsibilities for their staff, including grooms, cleaning and disinfection of air stalls, the area used to transfer horses to road transport at the airport, vehicles for transporting horses at the PAQ facility, and road transport arrangements including contingency plans for vehicle and equipment failures.

After horses arrive at an Australian airport they must be transferred from their air stalls onto vehicles for transporting horses, along with personnel and equipment, and proceed directly to the PAQ facility. AQIS door seals must be applied to vehicles for transporting horses to maintain biosecurity integrity during transport to the PAQ facility.

AQIS must check each horse's documentation on arrival at the PAQ facility.

All personnel travelling with the horses on the aircraft and road transport, or that have had contact with the horses, quarantine risk material or the air stalls, must undertake appropriate decontamination measures as specified by AQIS before leaving the airport or the PAQ facility if they are accompanying the horses to the PAQ facility.

Feed and water used during transport can travel with the horses to the PAQ facility for use only during PAQ.

All quarantine risk material (e.g. bedding, feed and water and waste material) remaining at the airport must be sealed in bags, ordered into quarantine and disposed of under AQIS supervision.

Air stalls must be secured at the airport in a manner that prevents the release of quarantine risk material and cleaned and disinfected under AQIS supervision.

Vehicles for transporting horses from the port of entry to the PAQ facility must be cleaned and disinfected to the satisfaction of the AQIS quarantine officer before loading the horses. AQIS must be advised of the transport route to the PAQ facility.

All equipment used during transport of horses, and all baggage and personal equipment accompanying personnel must be cleaned and disinfected under AQIS supervision before leaving the airport or the PAQ facility (if removed from the PAQ facility before the end of the PAQ period).

8 Quarantine measures for importation of horses

The quarantine measures described in this IRA report apply to the importation of horses, mules and donkeys from approved countries. Specific measures will be developed for each approved country and will reflect that country's animal health status.

As described in chapter 6, horses must be continuously resident in an approved country for not less than 60 days immediately before export to Australia. The 60 days residency requirement may be achieved in more than one approved country if specifically authorised by the Australian Quarantine and Inspection Service (AQIS). Conditions for each country of residence must be addressed.

The World Organisation for Animal Health (OIE) Terrestrial Animal Health Code (the Code) recommends that horses must be resident on premises, for certain diseases of horses, for periods ranging from less than 30 days to 90 days. In most cases, 60 days is recommended. The premises residency period provides additional assurance that diseases of quarantine concern may be detected if present on the premises.

Biosecurity Australia and the Expert Panel have reviewed these diseases and the relevant recommendations in the Code, and have concluded, for consistency and clarity of certification, that a 60 day requirement for premises residency is reasonable and appropriate in most cases.

In addition, the Code recommends a period for which a premises should remain free from certain diseases ranging from the same as the premises residency period to two or more years.

Biosecurity Australia and the Expert Panel have reviewed these diseases and the relevant recommendations in the Code, and have concluded, for consistency and clarity of certification, that a 90 day requirement for premises to be free from certain diseases is reasonable and appropriate in most cases.

For disease agents of quarantine concern that have no recommendations in the Code for the periods of premises residency and/or disease freedom, the periods are based on the epidemiology and information detailed in the relevant disease sections in chapters 5 and 6.

Testing requirements also vary for particular disease agents based on recommendations in the Code or where risk assessments were undertaken. Biosecurity Australia and the Expert Panel considered that the tests could in most instances be conducted at similar times in order to provide less trade restrictive options and facilitate certification without compromising the quarantine risk.

The quarantine measures for the permanent importation of horses are in section 8.1, and for the temporary importation of horses for racing or competition are in section 8.2. The operational and quarantine facilities requirements are the same for horses imported permanently or temporarily. Horses imported temporarily undergo pre-export quarantine (PEQ), post-arrival quarantine (PAQ) and quarantine surveillance during which they are denied the opportunity to mate. Therefore, risk management

measures have been amended for some diseases, including those transmitted venereally, for horses imported temporarily.

The permanent importation of horses seropositive for equine piroplasmosis is not permitted. For the temporary importation of horses seropositive for equine piroplasmosis, an import risk analysis has been conducted (Animal Quarantine Policy Memorandum 1999/81) and the quarantine measures from this report are recommended.

An example of the quarantine measures for a hypothetical approved country, Country X, is provided in section 8.3.

The residency periods and timing of tests recommended in the Code remain in the permanent (section 8.1) and temporary (section 8.2) quarantine requirements. The amended periods are included in an example for a hypothetical country (section 8.3) and will be included in the specific measures developed for each country.

8.1 Quarantine measures for the permanent importation of horses from approved countries

8.1.1 Documentation

Each horse, other than an unweaned foal under six months of age travelling with its dam, must travel with an original international veterinary certificate that conforms to Article 5.10.2. of the Code, signed by the Official Veterinarian* of the country of export.

These quarantine requirements apply to horses, donkeys and mules.

> * Official Veterinarian means a veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the Code.

The veterinary certificate must:

- be written in English and a language understood by the Official Veterinarian of the country of export
- meet the requirements of the **certification before export** section and state that all **pre-export quarantine requirements** have been met
- provide identification for each animal (passport details and/or microchip number/site or brand or silhouette) including description, species, sex and age
- include the name and address of the exporter and importer and identify the import permit against which it was issued.

The Official Veterinarian must:

- provide a separate veterinary certificate for each horse, including foals over six months of age
- attach certification applicable to unweaned foals under six months of age to the veterinary certificate of the foal's dam
- sign, date and stamp (with the stamp of the Veterinary Authority) each page of the veterinary certificate and attach all original documents, e.g. laboratory reports, that form part of the extended veterinary certification
- record his/her name, signature and contact details on the veterinary certificate.

Copies of supporting documents must be endorsed with the original signature, date and stamp of the Official Veterinarian on every page.

8.1.2 Pre-export quarantine requirements

Pre-export quarantine

For disease agents for which PEQ was considered a risk management measure, the length of the PEQ period is specified in section 8.1.3. The minimum PEQ period is 14 days.

Any variation from the **pre-export quarantine requirements** must be specifically authorised by AQIS.

Location

1. The PEQ facility must be located within 250 km of the port of export.
2. The PEQ facility must be conveniently located for supervision by the Official Veterinarian.

Facilities

1. The PEQ facility must meet the country and premises requirements specified in the **certification before export** section.
2. The entire PEQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to isolate horses in PEQ.
3. The PEQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
4. Stables in the PEQ facility must be constructed so that they can be cleaned and disinfectant applied and must be maintained in good order.
5. The PEQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
6. The PEQ facility must have facilities for veterinary examination and collection of samples.

Operation

1. The PEQ facility must have current approval from AQIS and the Veterinary Authority of the exporting country before commencement of PEQ.
2. AQIS may audit the approved PEQ facility.
3. All PEQ operations and procedures must be detailed in Standard Operating Procedures (SOPs), consistent with a risk-based approach and approved by AQIS.
4. The Official Veterinarian must inspect the PEQ facility before commencement of PEQ and must ensure that the facility has been cleaned and disinfectant applied to his/her satisfaction.
5. PEQ must be under the supervision of the Official Veterinarian.
6. All feed to be used during PEQ and transport to Australia must enter the PEQ facility before commencement of PEQ.
7. All bedding to be used during PEQ must enter the PEQ facility before commencement of PEQ.
8. The PEQ period commences from the time the last horse in the export consignment has entered the PEQ facility and all horses have been examined by the Official Veterinarian.
9. All equipment used in feeding, handling and treating horses in PEQ must be new, or cleaned and disinfected before entry, and must be used only in the facility during PEQ.
10. During PEQ, the facility must be occupied only by horses of the export consignment.
11. Horses must not have the opportunity to mate and must not be subjected to reproductive manipulation, other than required for certification, while in PEQ.
12. Only personnel specifically authorised by the Official Veterinarian are permitted entry to the PEQ facility. Details of all visitor entries must be recorded.
13. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results, treatments and reasons for removal from PEQ of any horse, must be reported to the Official Veterinarian within 24 hours, and to AQIS within 48 hours.
14. A detailed health record must be kept for each horse and be available to the Official Veterinarian and to AQIS on request.
15. Horses that leave the facility during PEQ for any reason cannot rejoin the consignment in PEQ.
16. Before the consignment of horses leaves the PEQ facility for export the Official Veterinarian must provide evidence to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.

8.1.3 Certification before export

The Official Veterinarian must certify:

1. During PEQ:
    a. the horse, including unweaned foals under six months of age, has been treated with a broad spectrum anthelmintic (date and treatment schedule stated on the veterinary certificate)
    b. the horse has not been vaccinated
    c. the horse has not been mated or subjected to reproductive manipulation, other than required for certification
    d. all horses in the PEQ facility remained free from evidence of infectious or contagious disease, and had no contact with equids not of equivalent health status
    e. all samples for testing have been taken by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian
    f. all testing has been conducted in a laboratory approved and monitored by the Veterinary Authority of the country of export. If there is no approved laboratory in the country of export, testing in another country must be conducted in a laboratory recognised by the Veterinary Authority of the country of export.
2. The horse has been examined by the Official Veterinarian within 24 hours before leaving the PEQ facility for the port of export and has been found to be:
    a. free from evidence of infectious or contagious disease
    b. visibly free of external parasites
    c. after due enquiry, in the case of a mare, either not pregnant or less than seven months pregnant
    d. healthy and fit to travel.
3. Vehicles for transporting horses from the PEQ facility to the port of export have been cleaned and disinfected to the satisfaction of the Official Veterinarian before entering the PEQ facility to load the horses.
4. The Official Veterinarian was present during loading of horses when leaving the PEQ facility to supervise sealing of vehicles for transporting horses, with tamper-evident seals.
5. During transport to the port of export, the horse had no contact with equids not of equivalent health status.
6. The compartment of the aircraft or vessel to be occupied by the horse and all removable equipment, penning and containers including loading ramps were satisfactorily cleaned and disinfected before loading.
7. All of the following risk management measures apply:

African horse sickness

a. For 40 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of African horse sickness has occurred during the previous two years and the disease is compulsorily notifiable.

AND

b. The horse has not been vaccinated against African horse sickness during 40 days before export.

Anthrax

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

Borna disease

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical evidence of Borna disease has occurred during the previous 90 days.

Contagious equine metritis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of contagious equine metritis has occurred during the previous two years.

OR

For all horses excluding geldings and unweaned foals under six months of age:

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of contagious equine metritis has occurred in equids during the previous 60 days.

AND

b. The horse has never been mated to or inseminated with semen from, a horse that was, at the time of mating or semen collection, known to be infected with contagious equine metritis.

NOTE: If a horse does not meet this requirement, or has been known to be infected with contagious equine metritis, it may be permitted entry subject to an approved method of treatment and testing considered appropriate by the Director of Quarantine (or delegate (AQIS)).

AND

c. Samples have been taken from the horse during the 30 days immediately before export and tested for *Taylorella equigenitalis* by culture* with negative results.

For colts and stallions, separate samples from each of the the urethra, the urethral fossa and sinus, and the penile sheath have been collected on three occasions, not less than seven days apart.

OR

For fillies and mares, one sample from the clitoral fossa, including the clitoral sinuses, has been collected on three occasions, not less than seven days apart, and for non-pregnant fillies and mares one sample from the endometrium or deep cervix has been collected on at least one occasion during oestrus.

* The samples were set up for culture within 48 hours of collection.

AND

d. The horse has not been treated with antibiotics for at least seven days before collection of the first samples for culture nor during the sample collection period.

AND

e. The horse has not been mated to or inseminated with semen from, a horse after collection of the first samples for culture.

AND

f. During PEQ the horse was not mated.

Dourine

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of dourine has occurred during the previous two years and the disease is compulsorily notifiable.

Eastern and Western equine encephalomyelitides

For 90 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Eastern or Western equine encephalomyelitis has occurred during the previous two years.

OR

For 90 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of Eastern or Western equine encephalomyelitis has occurred during the previous 90 days.

OR

The horse has been held in a PEQ facility for at least 21 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status **and**

during PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting insects before leaving the stables.

OR

During the 12 months before export, but not during PEQ, the horse has been vaccinated against Eastern and Western equine encephalomyelitis using an approved vaccine according to the manufacturer's recommendations.

Epizootic lymphangitis

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of epizootic lymphangitis has occurred during the previous 60 days.

Equid herpesvirus-1 (abortigenic and neurological strains)

For 21 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equid herpesvirus-1 (abortigenic and neurological strains) has occurred during the previous 21 days.

Equine infectious anaemia

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine infectious anaemia has occurred during the previous 90 days.

   AND

b. For all horses including unweaned foals under six months of age:

A blood sample has been taken from the horse during PEQ and tested using an agar gel immunodiffusion test or enzyme-linked immunosorbent assay as described in the OIE Manual for equine infectious anaemia with negative results.

Equine influenza

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine influenza has occurred during the previous 12 months, vaccination against equine influenza is not practised, and the disease is compulsorily notifiable.

OR

For all horses including unweaned foals under six months of age, except where otherwise specified:

a. For 21 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine influenza has occurred during the previous 30 days.

   AND

b. The horse (other than foals under six months of age) has been vaccinated against equine influenza 21–90 days before commencement of PEQ with either a primary course or a booster according to the manufacturer's recommendations using a vaccine that complies with the standards described in the OIE Manual.

NOTE: Vaccines used must contain the following or equivalent strains of equine influenza virus in accordance with the recommendations of the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition[18]:

an A/eq/South Africa/4/2003 (H3N8)-like virus (American lineage)[19].

AND

c. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids not of equivalent health status.

AND

d. For the duration of PEQ the horse has not been held, housed or exercised within 100 metres of other equids not of equivalent health status, unless specifically authorised by AQIS.

AND

e. Nasopharyngeal samples have been taken from the horse four to six days after commencement of PEQ and during the four days before leaving the PEQ facility and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

f. A reference serum sample has been taken from the horse during the four days after commencement of PEQ and stored in the exporting country in a laboratory approved and monitored by the Veterinary Authority until completion of PAQ.

AND

g. For the duration of PEQ the rectal temperature of the horse has been taken and recorded twice daily at least eight hours apart. If the temperature was 38.5 °C or higher on two consecutive recordings or other signs of infectious respiratory

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[18] Vaccines containing both American and European lineage (A/eq/Newmarket/2/93) are acceptable.

[19] A/eq/Ohio/2003, A/eq/Wisconsin/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are acceptable as A/eq/South Africa/4/2003-like viruses: Other strains (A/eq/Newmarket/1/93 or A/eq/Kentucky/94-like virus) are acceptable until such time as vaccine containing updated strains are available in the country of export.

disease were present, a nasopharyngeal sample has been taken and tested for influenza A virus and AQIS has been notified within 48 hours. If the temperature has not been taken for any reason on two consecutive occasions, AQIS has been notified within 48 hours and a clinical examination by a registered veterinarian performed. Temperature records must be kept until completion of PAQ.

Requirements for PEQ include:

1. The PEQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status unless specifically authorised by AQIS.
2. All personnel entering the PEQ facility during PEQ must shower and change clothing on entry. Alternatively, they may shower off-site and must have no contact with horses or horse facilities between showering and entering the PEQ facility. Outer clothing used in the PEQ facility should be freshly laundered or dedicated to the facility and stored on site or disposable. Footwear used in the PEQ facility should be cleaned and disinfected before entry or dedicated to the facility and stored on site, or disposable covering should be used over existing footwear.
3. All equipment used in feeding, handling and treating the horse in PEQ must be new or cleaned and disinfected with a product effective against equine influenza virus before use and must be used only in the PEQ facility for the duration of PEQ.
4. Horses in PEQ must not access any areas used by other horses unless specifically authorised by AQIS.
5. Vehicles for transporting horses from the PEQ facility to the place of export must be cleaned and disinfected with a product effective against equine influenza virus.

Equine piroplasmosis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous two years and the disease is compulsorily notifiable.

OR

For all horses including unweaned foals under six months of age:

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous 60 days.

   AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

   AND

c. On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

AND

d. If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

AND

e. During PEQ there has been no opportunity for iatrogenic transmission.

AND

f. Blood samples have been taken from the horse not less than seven days after commencement of PEQ and tested using an indirect fluorescent antibody test for *Babesia caballi* and *Theileria equi* as described in the OIE Manual for equine piroplasmosis with negative results in each case. If there is no approved laboratory in the country of export, testing in another country must be conducted in a laboratory recognised by the Veterinary Authority of the country of export.

AND

g. The horse has not been treated with imidocarb, or other anti-babesial agents active against *B. caballi* and *T. equi*, for at least 60 days before commencement of PEQ.

AND

h. The horse has not been tested with any test for equine piroplasmosis (*B. caballi* or *T. equi*) with a positive result for at least 60 days before export.

Equine viral arteritis

For colts or stallions:

a. For 28 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine viral arteritis has occurred during the previous 28 days.

AND

b. For 28 days before export the horse was not vaccinated against equine viral arteritis.

AND

c. A single blood sample has been taken from the horse not less than seven days after commencement of PEQ and tested using a virus neutralisation test as

described in the OIE Manual for equine viral arteritis with negative results (tested seronegative horse).

OR

Blood samples have been taken from the horse between six and nine months of age on two occasions at least 14 days apart and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre. The horse has been vaccinated against equine viral arteritis on the same day, after the second blood sample was taken, and has been revaccinated regularly according to the manufacturer's recommendations (known seropositive horse because of complying pre-pubertal vaccination).

OR

The horse was isolated for 28 days and a single blood sample taken not less than seven days after commencement of isolation and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results. The horse has been vaccinated against equine viral arteritis on the same day, after the blood sample was taken and remained isolated from other equids not of equivalent health status for 21 days immediately after vaccination and has been revaccinated regularly according to the manufacturer's recommendations (known seropositive horse because of complying vaccination).

OR

A single blood sample has been taken from the horse and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with positive results (non-complying vaccinated or non-vaccinated tested seropositive horse) **and**

the horse has not been treated with gonadotrophin-releasing hormone (GnRH) antagonists, or vaccinated against GnRH, for at least 12 months before test mating or collection of the first semen sample **and either**

i. the horse has subsequently been test-mated to two mares during the 12 months before export. Blood samples have been taken from the mares on two occasions, at the time of mating and again 28 days after test mating, and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results in each case **or**

ii. two semen samples have been taken from the horse during the 28 days before export and tested using a virus isolation test as described in the OIE Manual for equine viral arteritis with negative results.

<u>For fillies, mares and geldings:</u>

a. For 28 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine viral arteritis has occurred during the previous 28 days.

AND

b. For 28 days before PEQ the horse was not mated.

AND

c. For 28 days before export the horse was not vaccinated against equine viral arteritis.

AND

d. A single blood sample has been taken from the horse not less than seven days after commencement of PEQ and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results (tested seronegative horse).

OR

Blood samples have been taken from the horse on two occasions, at least 14 days apart, during the 28 days before export and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre (tested seropositive horse).

OR

Blood samples have been taken from the horse between six and nine months of age on two occasions at least 10–14 days apart and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre. The horse has been vaccinated against equine viral arteritis immediately after the second blood sample was taken and has been revaccinated regularly according to the manufacturer's recommendations (known seropositive horse because of complying pre-pubertal vaccination).

Glanders

For 180 days immediately before export, or since birth if under six months of age, the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of glanders has occurred during the previous three years and the disease is compulsorily notifiable.

Horse pox

For 90 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of horse pox has occurred during the previous 90 days.

Japanese encephalitis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Japanese encephalitis has occurred during the previous 12 months.

OR

The horse has been held in PEQ for at least 21 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status **and**

during PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting insects before leaving the stables.

OR

Within 12 months before export, but not during PEQ, the horse has been vaccinated against Japanese encephalitis using an approved vaccine according to the manufacturer's recommendations.

Lyme disease

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Lyme disease has occurred during the previous two years.

OR

For all horses including unweaned foals under six months of age:

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of Lyme disease has occurred in any species during the previous 90 days.

   AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

   AND

c. On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

   AND

d. If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

Rabies

For 180 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other

evidence of rabies has occurred during the previous two years and the disease is compulsorily notifiable.

OR

For 180 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of rabies has occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

Screw-worm-fly myiasis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of screw-worm-fly (*Cochliomyia hominivorax* or *Chrysomya bezziana*) myiasis has occurred during the previous 12 months.

OR

For all horses including unweaned foals under six months of age:

a. On arrival at the PEQ facility, the horse has been thoroughly examined, under the direct supervision of the Official Veterinarian, and no screw-worm-fly infestation has been found.

   AND

b. Within 24 hours of export the horse has been thoroughly examined, under the direct supervision of the Official Veterinarian, and no screw-worm-fly infestation has been found.

Surra

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months.

OR

For all horses including unweaned foals under six months of age:

a. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred in equids during the previous 12 months.

   AND

b. For 60 days immediately before export the horse has not resided on premises in the country of export where there has been clinical, epidemiological or other evidence of surra during the previous 12 months.

   AND

c. The horse has been held in PEQ for at least 21 days immediately before export.

AND

d. The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of surra has occurred in equids for 12 months before export.

AND

e. During PEQ the horse has been isolated and not held, housed or exercised within 200 metres of ruminants or camelids.

AND

f. During PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting flies before leaving the stables.

AND

g. Blood samples have been taken from the horse not less than ten days after commencement of PEQ and tested using an antibody-detection enzyme-linked immunosorbent assay and microhaematocrit centrifugation technique as described in the OIE Manual for surra (*Trypanosoma evansi*) with negative results in each case.

AND

h. The horse has been treated with an insect repellent for protection from biting flies before leaving the stable and being loaded into the vehicle for transporting horses from the PEQ facility to the port of export, and after loading the vehicle was disinsected.

Venezuelan equine encephalomyelitis

a. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Venezuelan equine encephalomyelitis has occurred during the previous two years and the disease is compulsorily notifiable.

AND

b. The horse has not been vaccinated against Venezuelan equine encephalomyelitis during the 60 days before export.

Vesicular stomatitis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

OR

a. For 30 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other

evidence of vesicular stomatitis has occurred in any species during the previous 90 days and the disease is compulsorily notifiable.

AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids and domestic livestock not of equivalent health status.

AND

c. The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species for 90 days before export.

AND

d. A blood sample has been taken from the horse not less than eight days after commencement of PEQ and tested using an enzyme-linked immunosorbent assay or virus neutralisation test as described in the OIE Manual for vesicular stomatitis (both New Jersey and Indiana strains) with negative results.

West Nile fever

For horses from countries where clinical West Nile fever occurs:

During the 12 months before export, but not during PEQ, the horse has been vaccinated against West Nile virus using an approved vaccine (not containing live West Nile virus) according to the manufacturer's recommendations.

8.1.4 Transport

1. Exporters or their agents must have detailed SOPs consistent with a risk-based approach and approved by AQIS, to cover procedures including contingency plans, for transporting the horses from PEQ until arrival in Australia.

2. The transport route from the PEQ facility to the approved airport must be approved by the Official Veterinarian.

3. The Official Veterinarian must be present during loading of the horses when leaving the PEQ facility to ensure vehicles for transporting horses are adequately cleaned and disinfected before loading, to supervise sealing of horse transport vehicles with tamper-evident seals and to certify that the horses are fit to travel. A government officer authorised by the Veterinary Authority must be available at the airport to check the vehicle seals are intact on arrival and ensure ramps and air stalls are adequately cleaned and disinfected.

4. All personnel likely to be in direct contact with the horses during transport to Australia (including transport from the PEQ facility to the airport, at the airport, and on the aircraft) must shower and wear new or clean protective clothing and footwear before coming into contact with the horses. They must not have any contact with horses not of equivalent health status during transport to Australia.

5. All feed to be used during transport to Australia must enter the PEQ facility before commencement of PEQ.

6. The use of hay or straw as bedding during transport is not permitted. Treated wood shavings, sterilised peat and soft board can be used.

7. Horses must remain isolated from all animals not of equivalent health status during transport from the PEQ facility until arrival in Australia.

8. Insect netting must be carried on the flight at all times for contingencies. There must be sufficient insect netting to cover all air stalls completely. Insect netting must be in good condition to minimise entry of insect vectors into the air stalls.

9. An Australian government veterinarian may be required to accompany the shipment to Australia at the importer's expense.

10. The consignment may be accompanied by other horses of equivalent health status or animals of other species only with the prior approval of AQIS.

11. The design of the air stalls, the recommended requirements for horses, the preparation for transport, and the disinfection of the interior of the aircraft, removable equipment, penning and containers must be in accordance with the recommendations of the Code and International Air Transport Association Live Animal Regulations unless otherwise agreed by AQIS.

Transit and transhipment

1. Horses must transit or tranship only at an approved airport. Any transhipment requires the prior approval of AQIS. Stops en route to Australia will need approval and permits from relevant authorities in the countries of transit and transhipment. Transit and transhipment times must not exceed six hours. Horses are not to leave the airport and must not be removed from their air stalls during transit or transhipment.

2. Horses must remain on board the aircraft at approved transit airports. Unauthorised personnel must not have contact with the horses. Cargo doors can be opened at approved transit airports to allow for unloading or loading of freight. Immediately after the cargo hold doors are closed, an approved knockdown aerosol insecticide must be sprayed throughout the cargo hold, in the manner recommended by the manufacturer.

3. In cases where horses in air stalls are to be unloaded, before opening the cargo door, the air stalls must be completely covered in netting to prevent insect access to the horses. The netting must remain in place until the horses are reloaded on an aircraft. Immediately after the horses are reloaded on an aircraft and the cargo hold doors are closed, an approved knockdown aerosol insecticide spray must be sprayed throughout the cargo hold in the manner recommended by the manufacturer. The insect netting must not be removed until 30 minutes after spraying.

Delayed takeoffs and unscheduled landings

1. Exporters or their agents must have contingency plans for the management of delayed takeoffs and unscheduled landings.

2. In the event that transit or transhipment exceeds six hours, AQIS must be notified immediately and the horse must not proceed to Australia without approval from

AQIS. If the aircraft lands at any airport other than in an approved country, AQIS must be informed immediately. The decision as to whether the horses can continue to travel to Australia, and additional quarantine measures that may be required, will be made by AQIS on a risk-based case-by-case basis.

Arrival in Australia

1. Importers or their agents must have detailed SOPs consistent with a risk-based approach and approved by AQIS, to cover post-arrival procedures. These SOPs are to be developed in consultation with AQIS and must include roles and responsibilities for their staff, including grooms, cleaning and disinfection of air stalls, the area used to transfer horses to road transport at the airport, vehicles for transporting horses at the PAQ facility, and road transport arrangements including contingency plans for vehicle and equipment failures.

2. After the horses arrive at an Australian airport they must be transferred from their air stalls onto vehicles for transporting horses, along with personnel and equipment, and proceed directly to the PAQ facility. AQIS door seals must be applied to vehicles for transporting horses to maintain biosecurity integrity during transport to the PAQ facility.

3. All personnel travelling with the horses on the aircraft and road transport, or that have had contact with the horses, quarantine risk material or air stalls, must undertake appropriate decontamination measures as specified by AQIS before leaving the airport or the PAQ facility if they are accompanying the horses to the PAQ facility.

4. Feed and water used during transport can travel with the horses to the PAQ facility for use only during PAQ.

5. All quarantine risk material (e.g. bedding, feed, water and waste material) remaining at the airport must be sealed in bags, ordered into quarantine and disposed of under AQIS supervision.

6. Air stalls must be secured at the airport in a manner that prevents release of quarantine risk material and cleaned and disinfected under AQIS supervision.

7. Vehicles for transporting horses from the port of entry to the PAQ facility must be cleaned and disinfected to the satisfaction of the AQIS quarantine officer before loading the horses. AQIS must be advised of the transport route to the PAQ facility.

8. All equipment used during transport of the horses, and all baggage and personal equipment accompanying personnel, must be cleaned and disinfected under AQIS supervision before leaving the airport or the PAQ facility (if removed from the PAQ facility before the end of the PAQ period).

8.1.5 Post-arrival quarantine requirements

Post-arrival quarantine

For disease agents for which a PAQ period was considered a risk management measure, the length of PAQ is specified. The minimum PAQ period is 14 days.

Any variation from the **post-arrival quarantine requirements** must be specifically authorised by AQIS.

The following post-arrival risk management measures apply as appropriate:

Contagious equine metritis

After completion of PAQ each imported pregnant mare must be held under quarantine surveillance until it has foaled and has been subsequently tested for *Taylorella equigenitalis* by culture with negative results. One sample must be collected from the clitoral fossa, including clitoral sinuses, and one sample from the endometrium or deep cervix during oestrus.

NOTE. Foals must remain under quarantine surveillance until the dam has returned negative results for *T. equigenitalis* from each sample.

Equine influenza

a. For horses originating from a single PEQ facility:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

nasopharyngeal samples must be taken from the horse four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities within the same region:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities NOT within the same region:

The horse must be held in PAQ for at least 21 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

b. A single consignment must not be split between PAQ facilities on arrival in Australia.

AND

c. For the duration of PAQ the horse must not be held, housed or exercised within 100 metres of other equids not of equivalent health status.

AND

d. A reference serum sample must be taken from the horse within 24 hours of arrival into the PAQ facility and stored at the National Animal Serum Bank at the Australian Animal Health Laboratory.

AND

e. For the duration of PAQ the rectal temperature of the horse must be taken and recorded twice daily at least eight hours apart. If the temperature is 38.5 °C or higher on two consecutive recordings or other signs of infectious respiratory disease are present, a nasopharyngeal sample must be taken and tested for influenza A virus and AQIS notified. If the temperature cannot been taken for any reason on two consecutive occasions, AQIS must be notified and a clinical examination by a registered veterinarian performed. Temperature records must be made available for inspection by AQIS.

Requirements for PAQ include:

1. The PAQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status.

2. All personnel entering the PAQ facility during PAQ must wear dedicated or disposable outer clothing and dedicated, cleaned and disinfected or disposable footwear. All personnel must shower and change outer clothing before leaving the PAQ facility. Outer clothing and footwear used within the PAQ facility must be cleaned to the satisfaction of AQIS before removal from the facility.

3. All equipment used in feeding, handling and treating the horse in PAQ must either be cleaned and disinfected with a product effective against equine influenza virus to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.

4. Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.

Equine piroplasmosis

a. Within 24 hours of arrival at the PAQ facility, the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

   AND

b. If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks and all horses in the facility must be tested for piroplasmosis at least 11 days after treatment for ticks.

Lyme disease

a. Within 24 hours of arrival at the PAQ facility the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

   AND

b. If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks.

Surra

a. The horse must be held in PAQ for at least 14 days.

   AND

b. Stables at the PAQ facility must have been sprayed with a residual insecticide (e.g. synthetic pyrethroid) during the 24 hours before the horse arrives at the facility. For the duration of PAQ the horse must be treated with insect repellent according to manufacturer's recommendations for protection from biting flies.

Location

1. The PAQ facility should be close to the port of arrival and be conveniently located for supervision by the AQIS veterinarian.

2. The facility must be located in an area that has been free from equine infectious anaemia during the previous 12 months.

Facilities

1. The PAQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to prevent horses in PAQ having contact with animals outside the facility.
2. The PAQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
3. Stables in the PAQ facility must be constructed so that they can be cleaned and disinfected.
4. The PAQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
5. The PAQ facility must have facilities for veterinary examination and collection of samples.

Operation

1. The PAQ facility must be approved by AQIS before entry of any horse into the facility.
2. AQIS may audit the approved PAQ facility.
3. All PAQ operations and procedures must have detailed SOPs, consistent with a risk-based approach and approved by AQIS.
4. The process from the time horses arrive at the airport to the completion of PAQ must be auditable.
5. PAQ must be under the supervision of the AQIS veterinarian.
6. The AQIS veterinarian must inspect the PAQ facility before entry of any horse and must certify that the facility has been cleaned and disinfectant applied to his/her satisfaction.
7. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake.
8. During PAQ, the only horses in the facility must be those of the import consignment.
9. Horses must not have the opportunity mate while in PAQ.
10. Appropriate biosecurity procedures must be implemented for vehicles for transporting horses, freight containers, equipment and associated personnel, including transport operators, before, during and after the transport of horses to the PAQ facility.
11. Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.
12. Each imported horse must be identified on arrival at the PAQ facility and the accompanying veterinary certificate and passport examined and checked by AQIS.

13. Only personnel specifically authorised by AQIS are permitted to enter the PAQ facility. Details of all visitor entries must be recorded.

14. All equipment used in feeding, handling and treating horses in PAQ must either be cleaned and disinfected to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.

15. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results and treatments must be reported to the AQIS veterinarian within 24 hours.

16. Any health problems affecting other animals on the facility undergoing PAQ must be reported to the AQIS veterinarian within 24 hours.

17. A detailed health record must be kept for each horse on the facility during the PAQ period and it must be available to the AQIS veterinarian.

18. The AQIS veterinarian must provide certification to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.

19. Horses must not leave the facility during PAQ.

8.2 Quarantine measures for the temporary importation of horses from approved countries

Importation under these conditions is restricted to horses that have been continuously resident and free of quarantine restriction in the exporting country for not less than 60 days immediately before export to Australia. The 60 days residency requirement may be achieved in more than one approved country if specifically authorised by the Australian Quarantine and Inspection Service (AQIS). The horses will be exported from Australia within 60 days of arrival unless specifically authorised by AQIS. After completion of post-arrival quarantine (PAQ), each horse imported under these conditions will be held under quarantine surveillance (see section 8.2.4) until exported. The imported horse must be denied the opportunity to mate either while in quarantine or under quarantine surveillance.

8.2.1 Documentation

Each horse must travel with:

- a current international passport that conforms to the Model Passport for International Movement of Competition Horses recommended in the World Organisation for Animal Health (OIE) Terrestrial Animal Health Code (the Code), Chapter 5.12, or a similar official document issued by an internationally recognised horse organisation
- an original international veterinary certificate that conforms to Article 5.10.2 of the Code, signed by the Official Veterinarian* of the country of export.

  * Official Veterinarian means a veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the Code.

The veterinary certificate must:

- be written in English and a language understood by the Official Veterinarian of the country of export
- meet the requirements of the **certification before export** section and state that all the **pre-export quarantine requirements** have been met
- provide identification for each animal (passport details and/or microchip number/site or brand or silhouette) including description, species, sex and age
- include the name and address of the exporter and importer and identify the import permit against which it was issued.

The Official Veterinarian must:

- provide a separate veterinary certificate for each horse

- sign, date and stamp (with the stamp of the Veterinary Authority) each page of the veterinary certificate and attach all original documents, e.g. laboratory reports, that form part of the extended veterinary certification
- record his/her name, signature and contact details on the veterinary certificate.

Copies of supporting documents must be endorsed with the original signature, date and stamp of the Official Veterinarian on every page.

8.2.2 Pre-export quarantine requirements

Pre-export quarantine

For disease agents for which pre-export quarantine (PEQ) was considered a risk management measure, the length of the PEQ period is specified in section 8.1.3. The minimum PEQ period is 14 days.

Any variation from the **pre-export quarantine requirements** must be specifically authorised by AQIS.

Location

1. The PEQ facility must be located within 250 km of the port of export.
2. The PEQ facility must be conveniently located for supervision by the Official Veterinarian.

Facilities

1. The PEQ facility must meet the country and premises requirements specified in the **certification before export** section.
2. The entire PEQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to isolate horses in PEQ.
3. The PEQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
4. Stables in the PEQ facility must be constructed so that they can be cleaned and disinfectant applied and must be maintained in good order.
5. The PEQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
6. The PEQ facility must have facilities for veterinary examination and collection of samples.

Operation

1. The PEQ facility must have current approval from AQIS and the Veterinary Authority of the exporting country before commencement of PEQ.
2. AQIS may audit the approved PEQ facility.

3. All PEQ operations and procedures must be detailed in Standard Operating Procedures (SOPs), consistent with a risk-based approach and approved by AQIS.
4. The Official Veterinarian must inspect the PEQ facility before commencement of PEQ and must ensure that the facility has been cleaned and disinfectant applied to his/her satisfaction.
5. PEQ must be under the supervision of the Official Veterinarian.
6. All feed to be used during PEQ and transport to Australia must enter the PEQ facility before commencement of PEQ.
7. All bedding to be used during PEQ must enter the PEQ facility before commencement of PEQ.
8. The PEQ period commences from the time the last horse in the export consignment has entered the PEQ facility and all horses have been examined by the Official Veterinarian.
9. All equipment used in feeding, handling and treating horses in PEQ must be new, or cleaned and disinfected before entry, and must be used only in the facility during PEQ.
10. During PEQ, the facility must be occupied only by horses of the export consignment.
11. Horses must not have the opportunity to mate and must not be subjected to reproductive manipulation while in PEQ.
12. Only personnel specifically authorised by the Official Veterinarian are permitted entry to the PEQ facility. Details of all visitor entries must be recorded.
13. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results, treatments and reasons for removal from PEQ of any horse, must be reported to the Official Veterinarian within 24 hours, and to AQIS within 48 hours.
14. A detailed health record must be kept for each horse and be available to the Official Veterinarian and to AQIS on request.
15. Horses that leave the facility during PEQ for any reason, other than for exercise as authorised by AQIS, cannot rejoin the consignment in PEQ.
16. Before the consignment of horses leaves the PEQ facility for export the Official Veterinarian must provide evidence to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.

8.2.3 Certification before export

The Official Veterinarian must certify:

1. During PEQ:
   a. the horse has been treated with a broad spectrum anthelmintic (date and treatment schedule stated on the veterinary certificate)
   b. the horse has not been vaccinated
   c. the horse has not been mated or subjected to reproductive manipulation
   d. all horses in the PEQ facility remained free from evidence of infectious or contagious disease, and had no contact with equids not of equivalent health status.
   e. all samples for testing have been taken by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian
   f. all testing has been conducted in a laboratory approved and monitored by the Veterinary Authority of the country of export. If there is no approved laboratory in the country of export, testing in another country must be conducted in a laboratory recognised by the Veterinary Authority of the country of export.
2. The horse has been examined by the Official Veterinarian within 24 hours before leaving the PEQ facility for the port of export and has been found to be:
   a. free from evidence of infectious or contagious disease
   b. visibly free of external parasites
   c. after due enquiry, in the case of a mare, was not pregnant
   d. healthy and fit to travel.
3. Vehicles for transporting horses from the PEQ facility to the port of export have been cleaned and disinfected to the satisfaction of the Official Veterinarian before entering the PEQ facility to load the horses.
4. The Official Veterinarian was present during loading of horses when leaving the PEQ facility to supervise sealing of vehicles for transporting horses, with tamper-evident seals.
5. During transport to the port of export, the horse had no contact with equids not of equivalent health status.
6. The compartment of the aircraft or vessel to be occupied by the horse and all removable equipment, penning and containers including loading ramps were satisfactorily cleaned and disinfected prior to loading.
7. All of the following risk management measures apply:

African horse sickness

a. For 40 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of African horse sickness has occurred during the previous two years and the disease is compulsorily notifiable.

   AND

b. The horse has not been vaccinated against African horse sickness during 40 days before export.

Anthrax

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

Borna disease

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical evidence of Borna disease has occurred during the previous 90 days.

Contagious equine metritis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of contagious equine metritis has occurred during the previous two years.

OR

For all horses excluding geldings:

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of contagious equine metritis has occurred in equids during the previous 60 days.

   AND

b. The horse has never been mated to, or inseminated with semen from, a horse that was, at the time of mating or semen collection, known to be infected with contagious equine metritis.

NOTE: If a horse does not meet this requirement, or has been known to be infected with contagious equine metritis, it may be permitted entry subject to an approved method of treatment and testing considered appropriate by the Director of Quarantine (or delegate (AQIS)).

Dourine

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of dourine has occurred during the previous two years and the disease is compulsorily notifiable.

Eastern and Western equine encephalomyelitides

For 90 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Eastern or Western equine encephalomyelitis has occurred during the previous two years **or**

OR

For 90 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of Eastern or Western equine encephalomyelitis has occurred during the previous 90 days.

OR

The horse has been held in a PEQ facility for at least 21 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status **and**

during PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting insects before leaving the stables.

OR

During the 12 months before export, but not during PEQ, the horse has been vaccinated against Eastern and Western equine encephalomyelitis using an approved vaccine according to the manufacturer's recommendations.

Epizootic lymphangitis

For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of epizootic lymphangitis has occurred during the previous 60 days.

Equid herpesvirus-1 (abortigenic and neurological strains)

For 21 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equid herpesvirus-1 (abortigenic and neurological strains) has occurred during the previous 21 days.

Equine infectious anaemia

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine infectious anaemia has occurred during the previous 90 days.

AND

b. A blood sample has been taken from the horse during PEQ and tested using an agar gel immunodiffusion test or enzyme-linked immunosorbent assay as described in the OIE Manual for equine infectious anaemia with negative results.

Equine influenza

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine influenza has occurred during the previous 12 months, vaccination against equine influenza is not practised, and the disease is compulsorily notifiable.

OR

a. For 21 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine influenza has occurred during the previous 30 days.

AND

b. The horse has been vaccinated against equine influenza 21–90 days before commencement of PEQ with either a primary course or a booster according to the manufacturer's recommendations using a vaccine that complies with the standards described in the OIE Manual.

NOTE: Vaccines used must contain the following or equivalent strains of equine influenza virus in accordance with the recommendations of the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition[20]:

an A/eq/South Africa/4/2003 (H3N8)-like virus (American lineage)[21].

AND

c. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids not of equivalent health status.

AND

d. For the duration of PEQ the horse has not been held, housed or exercised within 100 metres of other equids not of equivalent health status, unless specifically authorised by AQIS.

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[20] Vaccines containing both American and European lineage (A/eq/Newmarket/2/93) are acceptable.

[21] A/eq/Ohio/2003, A/eq/Wisconsin/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are acceptable as A/eq/South Africa/4/2003-like viruses: Other strains (A/eq/Newmarket/1/93 or A/eq/Kentucky/94-like virus) are acceptable until such time as vaccine containing updated strains are available in the country of export.

AND

e. Nasopharyngeal samples have been taken from the horse four to six days after commencement of PEQ and during the four days before leaving the PEQ facility and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

f. A reference serum sample has been taken from the horse during the four days after commencement of PEQ and stored in the exporting country in a laboratory approved and monitored by the Veterinary Authority until completion of PAQ.

AND

g. For the duration of PEQ the rectal temperature of the horse has been taken and recorded twice daily at least eight hours apart. If the temperature was 38.5 °C or higher on two consecutive recordings or other signs of infectious respiratory disease were present, a nasopharyngeal sample has been taken and tested for influenza A virus and AQIS has been notified within 48 hours. If the temperature has not been taken for any reason on two consecutive occasions, AQIS has been notified within 48 hours and a clinical examination by a registered veterinarian performed. Temperature records must be kept until completion of PAQ.

Requirements for PEQ include:

1. The PEQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status unless specifically authorised by AQIS.

2. All personnel entering the PEQ facility during PEQ must shower and change clothing on entry. Alternatively, they may shower off-site and must have no contact with horses or horse facilities between showering and entering the PEQ facility. Outer clothing used in the PEQ facility should be freshly laundered or dedicated to the facility and stored on site or disposable. Footwear used in the PEQ facility should be cleaned and disinfected before entry or dedicated to the facility and stored on site, or disposable covering should be used over existing footwear.

3. All equipment used in feeding, handling and treating the horse in PEQ must be new or cleaned and disinfected with a product effective against equine influenza virus before use and must be used only in the PEQ facility for the duration of PEQ.

4. Horses in PEQ must not access any areas used by other horses unless specifically authorised by AQIS.

5. Vehicles for transporting horses from the PEQ facility to the place of export must be cleaned and disinfected with a product effective against equine influenza virus.

Equine piroplasmosis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous two years and the disease is compulsorily notifiable.

OR

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine piroplasmosis has occurred during the previous 60 days.

AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

AND

c. On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

AND

d. If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

AND

e. During PEQ there has been no opportunity for iatrogenic transmission.

AND

f. Blood samples have been taken from the horse not less than seven days after commencement of PEQ and tested using an indirect fluorescent antibody test for *Babesia caballi* and *Theileria equi* as described in the OIE Manual for equine piroplasmosis with negative results in each case. If there is no approved laboratory in the country of export, testing in another country must be conducted in a laboratory recognised by the Veterinary Authority of the country of export.

NOTE: Horses that test positive for piroplasmosis may be permitted temporary importation under specific quarantine management measures (section 8.2.4).

AND

g. The horse has not been treated with imidocarb, or other anti-babesial agents active against *B. caballi* or *T. equi*, for at least 60 days before commencement of PEQ.

AND

h. The horse has not been tested with any test for equine piroplasmosis (*B. caballi* or *T. equi*) with a positive result for at least 60 days before export.

NOTE: Horses that test positive for piroplasmosis may be permitted temporary importation under specific quarantine management measures (section 8.2.4).

Equine viral arteritis

For colts or stallions:

For 28 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine viral arteritis has occurred during the previous 28 days.

OR

For fillies, mares and geldings:

a. For 28 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of equine viral arteritis has occurred during the previous 28 days.

AND

b. For 28 days before PEQ the horse was not mated.

Glanders

For 180 days immediately before export, or since birth if under six months of age, the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of glanders has occurred during the previous three years and the disease is compulsorily notifiable.

Horse pox

For 90 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of horse pox has occurred during the previous 90 days.

Japanese encephalitis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Japanese encephalitis has occurred during the previous 12 months.

OR

The horse has been held in PEQ for at least 21 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status **and**

during PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting insects before leaving the stables.

OR

Within 12 months before export, but not during PEQ, the horse has been vaccinated against Japanese encephalitis using an approved vaccine according to the manufacturer's recommendations.

Lyme disease

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Lyme disease has occurred during the previous two years.

OR

a. For 60 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of Lyme disease has occurred in any species during the previous 90 days.

   AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from animals not of equivalent health status.

   AND

c. On arrival at the PEQ facility the horse has been thoroughly examined under the direct supervision of an Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

   AND

d. If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

Rabies

For 180 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of rabies has occurred during the previous two years and the disease is compulsorily notifiable.

OR

For 180 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of rabies has

occurred in any species during the previous 12 months and the disease is compulsorily notifiable.

Screw-worm-fly myiasis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of screw-worm-fly (*Cochliomyia hominivorax* or *Chrysomya bezziana*) myiasis has occurred during the previous 12 months.

OR

a. On arrival at the PEQ facility, the horse has been thoroughly examined, under the direct supervision of the Official Veterinarian, and no screw-worm-fly infestation has been found.

AND

b. Within 24 hours of export the horse has been thoroughly examined, under the direct supervision of the Official Veterinarian, and no screw-worm-fly infestation has been found.

Surra

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred in any species during the previous 12 months.

OR

a. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of surra has occurred in equids during the previous 12 months.

AND

b. For 60 days immediately before export the horse has not resided on premises in the country of export where there has been clinical, epidemiological or other evidence of surra during the previous 12 months.

AND

c. The horse has been held in PEQ for at least 21 days immediately before export.

AND

d. The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of surra has occurred in equids for 12 months before export.

AND

e. During PEQ the horse has been isolated and not held, housed or exercised within 200 metres of ruminants or camelids.

AND

f. During PEQ the horse has been stabled in insect-screened stables. The horse has been treated with an insect repellent for protection from biting flies before leaving the stables.

AND

g. Blood samples have been taken from the horse not less than ten days after commencement of PEQ and tested using an antibody-detection enzyme-linked immunosorbent assay and microhaematocrit centrifugation technique as described in the OIE Manual for surra (*Trypanosoma evansi*) with negative results in each case.

AND

h. The horse has been treated with an insect repellent for protection from biting flies before leaving the stable and being loaded into the vehicle for transporting horses from the PEQ facility to the port of export, and after loading the vehicle was disinsected.

Venezuelan equine encephalomyelitis

a. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of Venezuelan equine encephalomyelitis has occurred during the previous two years and the disease is compulsorily notifiable.

AND

b. The horse has not been vaccinated against Venezuelan equine encephalomyelitis during the 60 days before export.

Vesicular stomatitis

For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in a country where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous two years and the disease is compulsorily notifiable.

OR

a. For 30 days immediately before export the horse has not resided on any premises in the country of export where clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species during the previous 90 days and the disease is compulsorily notifiable.

AND

b. The horse has been held in PEQ for at least 14 days immediately before export. During this time the horse has been isolated from equids and domestic livestock not of equivalent health status.

AND

c. The PEQ facility is located in a defined area where no clinical, epidemiological or other evidence of vesicular stomatitis has occurred in any species for 90 days before export.

AND

d. A blood sample has been taken from the horse not less than eight days after commencement of PEQ and tested using an enzyme-linked immunosorbent assay or virus neutralisation test as described in the OIE Manual for vesicular stomatitis (both New Jersey and Indiana strains) with negative results.

West Nile fever

For horses from countries where clinical West Nile fever occurs:

During the 12 months before export, but not during PEQ, the horse has been vaccinated against West Nile virus using an approved vaccine (not containing live West Nile virus) according to the manufacturer's recommendations.

8.2.4 Quarantine measures for the temporary importation of horses that are serologically positive for equine piroplasmosis

Horses which give positive results in tests for equine piroplasmosis may be permitted temporary import to compete in international competitions such as dressage, showjumping, eventing, races and exhibitions under the following conditions.

NOTE: AQIS will not permit the importation of horses serologically positive for equine piroplasmosis to compete in events where there is prolonged exposure to vegetation and opportunity for tick attachment such as endurance rides and driving events which involve a marathon phase.

General

These quarantine requirements are additional to the quarantine measures for the temporary importation of horses from approved countries.

Certification

The veterinary certificate must attest that each horse for export in the consignment:

1. Showed no clinical sign of equine piroplasmosis during PEQ.
2. Blood samples were taken from the horse not less than seven days after commencement of PEQ and tested using an indirect fluorescent antibody test for *Babesia caballi* and *Theileria equi* as described in the OIE Manual for equine piroplasmosis, with the following results:
    - *Theileria equi* POSITIVE/NEGATIVE
    - *Babesia caballi* POSITIVE/NEGATIVE

    (delete result which is not applicable)

3. On arrival at the PEQ facility the horse has been thoroughly examined at the PEQ facility, under the direct supervision of the Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks.

   AND

   If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks.

Post-arrival quarantine and surveillance requirements

1. All horses irrespective of serological status must be thoroughly examined for ticks within 24 hours of arrival at the PAQ facility by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be taken with close examination of ears, false nostrils, under-body areas (axilla, inguinal and under jawbone), perineum, mane and tail.

   AND

   Seropositive horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks.

   AND

   If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks and all horses in the facility (excluding horses previously identified as being seropositive) must be tested for piroplasmosis at least 11 days after treatment for ticks. Any ticks found should be identified.

2. Seropositive horses must be easily identified (for example by a unique colour coded disc on their head collar and bridle) and under quarantine surveillance while in Australia. Access to seropositive horses will be restricted.

3. PAQ and competition sites must be approved by AQIS to hold seropositive horses. This could involve a survey for ticks. PAQ must be conducted in areas free of *Rhipicephalus microplus*.

4. Seropositive horses must remain on approved PAQ facilities and competition sites. Movement between these sites must be controlled.

5. Grass in exercise areas of PAQ and competition sites must be kept very short by such measures as regular mowing.

6. Seropositive horses can compete and remain in areas *where Rhipicephalus microplus* is present for a maximum of seven days.

7. Seropositive horses must be maintained in a separate building from other horses on the PAQ and competition sites in a clearly demarcated area except at the actual time of training, competition, exhibition or racing.

8. Seropositive horses and all other horses on the same site including those in separate buildings as seropositive horses must be examined daily for ticks by AQIS or a under the supervision of AQIS until export of the seropositive horses.

9. Seropositive horses must be treated weekly with a parasiticide effective against ticks.

10. Measures, including the use of disposable equipment, must be taken to prevent iatrogenic spread of the disease to ensure there is no risk of cross-contamination to other animals.

11. Bedding for seropositive horses must be rubber, wood shavings or shredded paper. If straw is used for stable bedding for seronegative horses located at the same site as seropositive horses it must be sourced from *Rhipicephalus microplus* free areas.

12. Hay, chaff and any other feedstuffs not heat treated for seropositive horses and all horses on site with seropositive horses must be sourced from *Rhipicephalus microplus* free areas.

13. Seropositive horses must be exported within ten days of the completion of the competition, exhibition or racing event for which they were imported unless specifically authorised by AQIS.

8.2.5 Transport

1. Exporters or their agents must have detailed SOPs consistent with a risk-based approach and approved by AQIS, to cover procedures including contingency plans, for transporting the horses from PEQ until arrival in Australia.

2. The transport route from the PEQ facility to the approved airport must be approved by the Official Veterinarian.

3. The Official Veterinarian must be present during loading of the horse when leaving the PEQ facility to ensure vehicles for transporting horses are adequately cleaned and disinfected before loading, to supervise sealing of vehicles for transporting horses with tamper-evident seals and to certify that the horses are fit to travel. A government officer authorised by the Veterinary Authority must be available at the airport to check the vehicle seals are intact on arrival and ensure ramps and air stalls are adequately cleaned and disinfected.

4. All personnel likely to be in direct contact with the horses during transport to Australia (including transport from the PEQ facility to the airport, at the airport, and on the aircraft) must shower and wear new or clean protective clothing and footwear before coming into contact with the horses. They must not have any contact with horses not of equivalent health status during transport to Australia.

5. All feed to be used during transport to Australia must enter the PEQ facility before commencement of PEQ.

6. The use of hay or straw as bedding during transport is not permitted. Treated wood shavings, sterilised peat and soft board can be used.

7. Horses must remain isolated from all animals not of equivalent health status during transport from the PEQ facility until arrival in Australia.

8. Insect netting must be carried on the flight at all times for contingencies. There must be sufficient insect netting to cover all air stalls completely. Insect netting must be in good condition to minimise entry of insect vectors into the air stalls.

9. An Australian government veterinarian may be required to accompany the shipment to Australia at the importer's expense.

10. The consignment may be accompanied by other horses of equivalent health status or animals of other species only with the prior approval of AQIS.

11. The design of the air stalls, the recommended requirements for horses, the preparation for transport, and the disinfection of the interior of the aircraft, removable equipment, penning and containers must be in accordance with the recommendations of the Code and International Air Transport Association Live Animal Regulations unless otherwise agreed by AQIS.

Transit and transhipment

1. Horses must transit or tranship only at an approved airport. Any transhipment requires the prior approval of AQIS. Stops en route to Australia will need approval and permits from relevant authorities in the countries of transit and transhipment. Transit and transhipment times must not exceed six hours. Horses are not to leave the airport and must not be removed from their air stalls during transit or transhipment.

2. Horses must remain on board the aircraft at approved transit airports. Unauthorised personnel must not have contact with the horses. Cargo doors can be opened at approved transit airports to allow for unloading or loading of freight. Immediately after the cargo hold doors are closed, an approved knockdown aerosol insecticide must be sprayed throughout the cargo hold, in the manner recommended by the manufacturer.

3. In cases where horses in air stalls are to be unloaded, before opening the cargo door, the air stalls must be completely covered in netting to prevent insect access to the horses. The netting must remain in place until the horses are reloaded on an aircraft. Immediately after the horses are reloaded on an aircraft and the cargo hold doors are closed, an approved knockdown aerosol insecticide spray must be sprayed throughout the cargo hold in the manner recommended by the manufacturer. The insect netting must not be removed until 30 minutes after spraying.

Delayed takeoffs and unscheduled landings

1. Exporters or their agents must have contingency plans for the management of delayed takeoffs and unscheduled landings.

2. In the event that transit or transhipment exceeds six hours, AQIS must be notified immediately and the horse must not proceed to Australia without approval from AQIS. If the aircraft lands at any airport other than in an approved country, AQIS must be informed immediately. The decision as to whether the

horse can continue to travel to Australia, and additional quarantine measures that may be required, will be made by AQIS on a risk-based case-by-case basis.

Arrival in Australia

1. Importers or their agents must have detailed SOPs consistent with a risk-based approach and approved by AQIS, to cover post-arrival procedures. These SOPs are to be developed in consultation with AQIS and must include roles and responsibilities for their staff, including grooms; cleaning and disinfection of air stalls, the area used to transfer horses to road transport at the airport, vehicles for transporting horses at the PAQ facility, and road transport arrangements including contingency plans for vehicle and equipment failures.

2. After the horses arrive at an Australian airport they must be transferred from their air stalls onto vehicles for transporting horses, along with personnel and equipment, and proceed directly to the PAQ facility. AQIS door seals must be applied to vehicles for transporting horses to maintain biosecurity integrity during transport to the PAQ facility.

3. All personnel travelling with the horses on the aircraft and road transport, or that have had contact with the horses, quarantine risk material or air stalls, must undertake appropriate decontamination measures as specified by AQIS before leaving the airport or the PAQ facility if they are accompanying the horses to the PAQ facility.

4. Feed and water used during transport can travel with horses to the PAQ facility for use only during PAQ.

5. All quarantine risk material (e.g. bedding, feed, water and waste material) remaining at the airport must be sealed in bags, ordered into quarantine and disposed of under AQIS supervision.

6. Air stalls must be secured at the airport in a manner that prevents release of quarantine risk material and cleaned and disinfected under AQIS supervision.

7. Vehicles for transporting horses from the port of entry to the PAQ facility must be cleaned and disinfected to the satisfaction of the AQIS quarantine officer before loading the horses. AQIS must be advised of the transport route to the PAQ facility.

8. All equipment used during transport of the horses, and all baggage and personal equipment accompanying personnel, must be cleaned and disinfected under AQIS supervision before leaving the airport or the PAQ facility (if removed from the PAQ facility before the end of the PAQ period).

8.2.6 Post-arrival quarantine requirements

After completion of PAQ, each horse imported under these conditions will be held under quarantine surveillance until exported. Horses will be exported from Australia within 60 days of arrival, unless specifically authorised by AQIS. Imported horses must be denied the opportunity to mate either while in quarantine or under quarantine surveillance.

Post-arrival quarantine

For disease agents for which PAQ was considered a risk management measure, the length of the PAQ period is specified. The minimum PAQ period is 14 days.

Any variation from the **post-arrival quarantine requirements** must be specifically authorised by AQIS.

The following post-arrival risk management measures apply as appropriate:

Equine influenza

a. For horses originating from a single PEQ facility:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

nasopharyngeal samples must be taken from the horse four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities within the same region:

The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

OR

For horses originating from multiple PEQ facilities NOT within the same region:

The horse must be held in PAQ for at least 21 days. During this time the horse must be isolated from equids not of equivalent health status **and**

the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after

commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

AND

b. A single consignment must not be split between PAQ facilities on arrival in Australia.

AND

c. For the duration of PAQ the horse must not be held, housed or exercised within 100 metres of other equids not of equivalent health status.

AND

d. A reference serum sample must be taken from the horse within 24 hours of arrival into the PAQ facility and stored at the National Animal Serum Bank at the Australian Animal Health Laboratory.

AND

e. For the duration of PAQ the rectal temperature of the horse must be taken and recorded twice daily at least eight hours apart. If the temperature is 38.5 °C or higher on two consecutive recordings or other signs of infectious respiratory disease are present, a nasopharyngeal sample must be taken and tested for influenza A virus and AQIS notified. If the temperature cannot been taken for any reason on two consecutive occasions, AQIS must be notified and a clinical examination by a registered veterinarian performed. Temperature records must be made available for inspection by AQIS.

Requirements for PAQ include:

1. The PAQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status.

2. All personnel entering the PAQ facility during PAQ must wear dedicated or disposable outer clothing and dedicated, cleaned and disinfected or disposable footwear. All personnel must shower and change outer clothing before leaving the PAQ facility. Outer clothing and footwear used within the PAQ facility must be cleaned to the satisfaction of AQIS before removal from the facility.

3. All equipment used in feeding, handling and treating horses in PAQ must either be cleaned and disinfected with a product effective against equine influenza virus to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.

4. Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.

Equine piroplasmosis

a. Within 24 hours of arrival at the PAQ facility, the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

AND

b. If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks and all horses in the facility must be tested for piroplasmosis at least 11 days after treatment for ticks.

Lyme disease

a. Within 24 hours of arrival at the PAQ facility the horse must be thoroughly examined by a registered veterinarian under the direct supervision of the AQIS veterinarian, and no ticks found. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

AND

b. If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks.

Surra

a. The horse must be held in PAQ for at least 14 days.

AND

b. Stables at the PAQ facility must have been sprayed with a residual insecticide (e.g. synthetic pyrethroid) during the 24 hours before the horse arrives at the facility. For the duration of PAQ the horse must be treated with insect repellent according to the manufacturer's recommendations for protection from biting flies.

Location

1. The PAQ facility should be close to the port of arrival and be conveniently located for supervision by the AQIS veterinarian.

2. The facility must be located in an area that has been free from equine infectious anaemia during the previous 12 months.

Facilities

1. The PAQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to prevent horses in PAQ having contact with animals outside the facility.

2. The PAQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
3. Stables in the PAQ facility must be constructed so that they can be cleaned and disinfected.
4. The PAQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
5. The PAQ facility must have facilities for veterinary examination and collection of samples.

Operation

1. The PAQ facility must be approved by AQIS before entry of any horse into the facility.
2. AQIS may audit the approved PAQ facility.
3. All PAQ operations and procedures must have detailed SOPs, consistent with a risk-based approach and approved by AQIS.
4. The process from the time horses arrive at the airport to the completion of PAQ must be auditable.
5. PAQ must be under the supervision of the AQIS veterinarian.
6. The AQIS veterinarian must inspect the PAQ facility entry of any horse and must certify that the facility has been cleaned and disinfectant applied to his/her satisfaction.
7. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake.
8. During PAQ, the only horses in the facility must be those of the import consignment.
9. Horses must not have the opportunity mate while in PAQ.
10. Appropriate biosecurity procedures must be implemented for vehicles for transporting horses, freight containers, equipment and associated personnel, including transport operators, before, during and after the transport of horses to the PAQ facility.
11. Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.
12. Each imported horse must be identified on arrival at the PAQ facility and the accompanying veterinary certificate and passport examined and checked by AQIS.
13. Only personnel specifically authorised by AQIS are permitted to enter the PAQ facility. Details of all visitor entries must be recorded.

14. All equipment used in feeding, handling and treating horses in PAQ must either be cleaned and disinfected to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.
15. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results and treatments must be reported to the AQIS veterinarian within 24 hours.
16. Any health problems affecting other animals on the facility undergoing PAQ must be reported to the AQIS veterinarian within 24 hours.
17. A detailed health record must be kept for each horse on the facility during the PAQ period and it must be available to the AQIS veterinarian.
18. The AQIS veterinarian must provide certification to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.
19. Horses must not leave the facility during PAQ.

8.3 Quarantine measures for the permanent importation of horses from Country X

8.3.1 Documentation

Each horse, other than an unweaned foal under six months of age travelling with its dam, must travel with an original international veterinary certificate that conforms to Article 5.10.2. of the World Organisation for Animal Health (OIE) Terrestrial Animal Health Code (the Code), signed by the Official Veterinarian* of the country of export.

These quarantine requirements apply to horses, donkeys and mules.

> * Official Veterinarian means a veterinarian authorised by the Veterinary Authority of the country of export to perform certain official tasks associated with animal health and/or public health, and inspections of commodities and, when appropriate, to certify in conformity with the Certification Procedures of Chapter 5.2 of the Code.

The veterinary certificate must:

- be written in English and a language understood by the Official Veterinarian of the country of export
- meet the requirements of the **certification before export** section and state that all **pre-export quarantine requirements** have been met
- provide identification for each animal (passport details and/or microchip number/site or brand or silhouette) including description, species, sex and age
- include the name and address of the exporter and importer and identify the import permit against which it was issued.

The Official Veterinarian must:

- provide a separate veterinary certificate for each horse, including foals over six months of age
- attach certification applicable to unweaned foals under six months of age to the veterinary certificate of the foal's dam
- sign, date and stamp (with the stamp of the Veterinary Authority) each page of the veterinary certificate and attach all original documents, e.g. laboratory reports, that form part of the extended veterinary certification
- record his/her name, signature and contact details on the veterinary certificate.

  Copies of supporting documents must be endorsed with the original signature, date and stamp of the Official Veterinarian on every page.

8.3.2 Pre-export quarantine requirements

Pre-export quarantine requirements for the importation of horses from Country X

Any variation from **the pre-export quarantine requirements** must be specifically authorised by the Australian Quarantine and Inspection Service (AQIS).

Location

1. The pre-export quarantine (PEQ) facility must be located within 250 km of the port of export.
2. The PEQ facility must be conveniently located for supervision by the Official Veterinarian.

Facilities

1. The PEQ facility must meet the country and premises requirements specified in the **certification before export** section.
2. The entire PEQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to isolate horses in PEQ.
3. The PEQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
4. Stables in the PEQ facility must be constructed so that they can be cleaned and disinfectant applied and must be maintained in good order.
5. The PEQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status.
6. The PEQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
7. The PEQ facility must have facilities for veterinary examination and collection of samples.

Operation

1. The PEQ facility must have current approval from AQIS and the Veterinary Authority of the exporting country before commencement of PEQ.
2. AQIS may audit the approved PEQ facility.
3. All PEQ operations and procedures must be detailed in Standard Operating Procedures (SOPs), consistent with a risk-based approach and approved by AQIS.
4. The Official Veterinarian must inspect the PEQ facility before commencement of PEQ and must ensure that the facility has been cleaned and disinfectant applied to his/her satisfaction.

5. PEQ must be under the supervision of the Official Veterinarian.
6. All feed to be used during PEQ and transport to Australia must enter the PEQ facility before commencement of PEQ.
7. All bedding to be used during PEQ must enter the PEQ facility before commencement of PEQ.
8. The PEQ period commences from the time the last horse in the export consignment has entered the PEQ facility and all horses have been examined by the Official Veterinarian.
9. All equipment used in feeding, handling and treating horses in PEQ must be new, or cleaned and disinfected before entry, and must be used only in the facility during PEQ.
10. During PEQ, the facility must be occupied only by horses of the export consignment.
11. For the duration of PEQ the horse has not been held, housed or exercised within 100 metres of other equids not of equivalent health status.
12. Horses in PEQ must not access any areas used by other horses.
13. Horses must not have the opportunity to mate and must not be subjected to reproductive manipulation, other than required for certification, while in PEQ.
14. Only personnel specifically authorised by the Official Veterinarian are permitted entry to the PEQ facility. Details of all visitor entries must be recorded.
15. All personnel entering the PEQ facility during PEQ must shower and change clothing on entry. Alternatively, they may shower off-site and must have no contact with horses or horse facilities between showering and entering the PEQ facility. Outer clothing used in the PEQ facility should be freshly laundered or dedicated to the facility and stored on site or disposable. Footwear used in the PEQ facility should be cleaned and disinfected before entry or dedicated to the facility and stored on site, or disposable covering should be used over existing footwear.
16. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results, treatments and reasons for removal from PEQ of any horse, must be reported to the Official Veterinarian within 24 hours, and to AQIS within 48 hours.
17. A detailed health record must be kept for each horse and be available to the Official Veterinarian and to AQIS on request.
18. A reference serum sample must be taken from the horse, including unweaned foals under six months of age, during the four days after commencement of PEQ and stored in the exporting country in a laboratory approved and monitored by the Veterinary Authority until completion of PAQ.
19. For the duration of PEQ the rectal temperature of the horse, including unweaned foals under six months of age, must be taken and recorded twice daily at least eight hours apart. If the temperature is 38.5 °C or higher on two consecutive

recordings or other signs of respiratory disease were present, a nasopharyngeal sample must be taken and tested for influenza A virus and AQIS must be notified within 48 hours. If the temperature is not taken for any reason on two consecutive occasions, AQIS must be notified within 48 hours and a clinical examination by a registered veterinarian performed. Temperature records must be kept until completion of PAQ.

20. Horses that leave the facility during PEQ for any reason cannot rejoin the consignment in PEQ.

21. Before the consignment of horses leaves the PEQ facility for export the Official Veterinarian must provide evidence to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.

8.3.3 Certification before export

The Official Veterinarian must certify:

1. During PEQ:

   a. the horse has not been vaccinated

   b. the horse has not been mated or subjected to reproductive manipulation, other than required for certification

   c. all horses in the PEQ facility remained free from evidence of infectious or contagious disease, and had no contact with equids not of equivalent health status

   d. all samples for testing have been taken by the Official Veterinarian or a veterinarian authorised by the Official Veterinarian

   e. all testing has been conducted in a laboratory approved and monitored by the Veterinary Authority of the country of export. If there is no approved laboratory in the country of export, testing in another country must be conducted in a laboratory recognised by the Veterinary Authority of the country of export.

2. The horse has been examined by the Official Veterinarian within 24 hours before leaving the PEQ facility for the port of export and has been found to be:

   a. free from evidence of infectious or contagious disease

   b. visibly free of external parasites

   c. after due enquiry, in the case of a mare, either not pregnant or less than seven months pregnant

   d. healthy and fit to travel.

3. Vehicles for transporting horses from the PEQ facility to the port of export have been cleaned and disinfected to the satisfaction of the Official Veterinarian before entering the PEQ facility to load the horses.

4. The Official Veterinarian was present during loading of horses when leaving the PEQ facility to supervise sealing of vehicles for transporting horses, with tamper-evident seals.

5. During transport to the port of export, the horse had no contact with equids not of equivalent health status.

6. The compartment of the aircraft or vessel to be occupied by the horse and all removable equipment, penning and containers including loading ramps were satisfactorily cleaned and disinfected before loading.

7. After due enquiry, for 180 days immediately before export, or since birth if under six months of age, the horse has been continuously resident and free of quarantine restriction in Country X, or other countries, where no clinical, epidemiological or other evidence of glanders has occurred during the previous three years and the disease is compulsorily notifiable.

8. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in Country X where no clinical, epidemiological or other evidence of African horse sickness, dourine, equine piroplasmosis, rabies, Venezuelan equine encephalomyelitis or vesicular stomatitis has occurred during the previous two years and the diseases are compulsorily notifiable. The horse has not been vaccinated against African horse sickness or Venezuelan equine encephalomyelitis during the 60 days before export.

9. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in Country X where no clinical, epidemiological or other evidence of Eastern or Western equine encephalomyelitis has occurred during the previous two years.

10. For 60 days immediately before export the horse has been continuously resident and free of quarantine restriction in Country X where no clinical, epidemiological or other evidence of Japanese encephalitis, screw-worm-fly (*Cochliomyia hominivorax* or *Chrysomya bezziana*) myiasis or surra has occurred during the previous 12 months.

11. After due enquiry, for 60 days immediately before export the horse has not resided on any premises in Country X where clinical evidence of Borna disease has occurred during the previous 90 days.

12. After due enquiry, for 60 days immediately before export the horse has not resided on any premises in Country X where clinical, epidemiological or other evidence of contagious equine metritis, epizootic lymphangitis, equine infectious anaemia, horse pox or Lyme disease has occurred during the previous 90 days.

13. After due enquiry, for 30 days immediately before export the horse has not resided on any premises in Country X where clinical, epidemiological or other evidence of anthrax, equid herpesvirus-1 (abortigenic and neurological strains) equine influenza or equine viral arteritis has occurred during the previous 30 days.

14. The horse has been held in PEQ for at least 14 days immediately before export in a facility that met the requirements specified in the PEQ requirements. During this time the horse has been isolated from equids not of equivalent health status.

15. Contagious equine metritis

For all horses excluding geldings and unweaned foals under six months of age:

a. The horse has never been mated to, or inseminated with semen from, a horse that was, at the time of mating or semen collection, known to be infected with contagious equine metritis.

NOTE: If a horse does not meet this requirement, or has been known to have been infected with contagious equine metritis, it may be permitted entry subject to an approved method of treatment and testing considered appropriate by the Director of Quarantine (or delegate (AQIS)).

AND

b. Samples have been taken from the horse during the 30 days immediately before export and tested for *Taylorella equigenitalis* by culture* with negative results.

For colts and stallions, separate samples from each of the urethra, urethral fossa and sinus, and the penile sheath, have been collected on three occasions, not less than seven days apart

OR

For fillies and mares, one sample from the clitoral fossa, including the clitoral sinuses, has been collected on three occasions, not less than seven days apart, and for non-pregnant fillies and mares one sample from the endometrium or deep cervix has been collected on at least one occasion during oestrus.

* The samples were set up for culture within 48 hours of collection.

AND

c. The horse has not been treated with antibiotics for at least seven days before collection of the first samples for culture nor during the sample collection period.

AND

d. The horse has not been mated to, or inseminated with semen from, a horse after collection of the first samples for culture.

16. Equine infectious anaemia

For all horses including unweaned foals under six months of age:

A blood sample has been taken from the horse four to six days after commencement of PEQ and tested using an agar gel immunodiffusion test or enzyme-linked immunosorbent assay as described in the OIE Manual for equine infectious anaemia with negative results.

17. Equine influenza

For all horses including unweaned foals under six months of age, except where otherwise specified:

a. The horse (other than foals under six months of age) has been vaccinated against equine influenza 21–90 days before commencement of PEQ with either a primary course or a booster according to the manufacturer's recommendations using a vaccine that complies with the standards described in the OIE Manual.

NOTE: Vaccines used must contain the following or equivalent strains of equine influenza virus in accordance with the recommendations of the OIE Expert Surveillance Panel for Equine Influenza Vaccine Composition[22]:

an A/eq/South Africa/4/2003 (H3N8)-like virus (American lineage)[23].

AND

b. Nasopharyngeal samples have been taken from the horse four to six days after commencement of PEQ and during the four days before leaving the PEQ facility and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

18. Equine viral arteritis

<u>For colts or stallions:</u>

a. For 28 days before export the horse was not vaccinated against equine viral arteritis.

AND

b. A single blood sample has been taken from the horse four to six days days after commencement of PEQ and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results (tested seronegative horse).

OR

[22] Vaccines containing both American and European lineage (A/eq/Newmarket/2/93) are acceptable.

[23] A/eq/Ohio/2003, A/eq/Wisconsin/03, A/eq/Ibaraki/07 and A/eq/Sydney/07 are acceptable as A/eq/South Africa/4/2003-like viruses: Other strains (A/eq/Newmarket/1/93 or A/eq/Kentucky/94-like virus) are acceptable until such time as vaccine containing updated strains are available in the country of export.

Blood samples have been taken from the horse between six and nine months of age on two occasions at least 14 days apart and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre. The horse has been vaccinated against equine viral arteritis on the same day, after the second blood sample was taken, and has been revaccinated regularly according to the manufacturer's recommendations (known seropositive horse because of complying pre-pubertal vaccination).

OR

The horse was isolated for 28 days and a single blood sample taken four to six days after commencement of isolation and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results. The horse has been vaccinated against equine viral arteritis on the same day, after the blood sample was taken and remained isolated from other equids not of equivalent health status for 21 days immediately after vaccination and has been revaccinated regularly according to the manufacturer's recommendations (known seropositive horse because of complying vaccination).

OR

A single blood sample has been taken from the horse and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with positive results (non-complying vaccinated or non-vaccinated tested seropositive horse) **and**

the horse has not been treated with gonadotrophin-releasing hormone (GnRH) antagonists, or vaccinated against GnRH, for at least 12 months before test mating or collection of the first semen sample **and either**

i. the horse has been test-mated to two mares during the 12 months before export. Blood samples have been taken from the mares on two occasions, at the time of mating and again 28 days after test mating, and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with negative results in each case **or**

ii. two semen samples have been taken from the horse during the 28 days before export and tested using a virus isolation test as described in the OIE Manual for equine viral arteritis with negative results.

<u>For fillies, mares and geldings:</u>

a. For 28 days before PEQ the horse was not mated.

   AND

b. For 28 days before export the horse was not vaccinated against equine viral arteritis.

   AND

c. A single blood sample has been taken from the horse four to six days after commencement of PEQ and tested using a virus neutralisation test as

described in the OIE Manual for equine viral arteritis with negative results (tested seronegative horse).

OR

Blood samples have been taken from the horse on two occasions, at least 14 days apart, during the 28 days before export and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre (tested seropositive horse).

OR

Blood samples have been taken from the horse between six and nine months of age on two occasions at least 14 days apart and tested using a virus neutralisation test as described in the OIE Manual for equine viral arteritis with a stable or decreasing antibody titre. The horse has been vaccinated against equine viral arteritis on the same day after the second blood sample was taken and has been revaccinated regularly according to the manufacturer's recommendations (complying pre-pubertal vaccination).

19. For all horses including unweaned foals under six months of age:

    a. On arrival at the PEQ facility the horse has been thoroughly examined, under the direct supervision of an Official Veterinarian, and no ticks have been found. A systematic approach was undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

    AND

    b. The horse was then treated immediately, under the direct supervision of the Official Veterinarian, with a parasiticide effective against ticks (date and treatment schedule stated on the veterinary certificate).

    AND

    c. If any horse in the PEQ facility was found to have ticks, all horses in the facility were treated again seven days later with a parasiticide effective against ticks (date and treatment schedule stated on the veterinary certificate).

20. During PEQ the horse, including unweaned foals under six months of age, has been treated with a broad-spectrum anthelmintic (date and treatment schedule stated on the veterinary certificate).

8.3.4 Transport

1. Exporters or their agents must have detailed SOPs consistent with a risk-based approach and approved by AQIS, to cover procedures including contingency plans, for transporting the horse from PEQ until arrival in Australia.

2. The transport route from the PEQ facility to the approved airport must be approved by the Official Veterinarian.

3. The Official Veterinarian must be present during loading of horses when leaving the PEQ facility to ensure vehicles for transporting horses are adequately cleaned

and disinfected before loading, to supervise sealing of vehicles for transporting horses with tamper-evident seals and to certify that the horses are fit to travel. A government officer authorised by the Veterinary Authority must be available at the airport to check the vehicle seals are intact on arrival and ensure ramps and air stalls are adequately cleaned and disinfected.

4. All personnel likely to be in direct contact with the horses during transport to Australia (including transport from the PEQ facility to the airport, at the airport, and on the aircraft) must shower and wear new or clean protective clothing and footwear before coming into contact with the horses. They must not have any contact with horses not of equivalent health status during transport to Australia.

5. All feed to be used during transport to Australia must enter the PEQ facility before commencement of PEQ.

6. The use of hay or straw as bedding during transport is not permitted. Treated wood shavings, sterilised peat and soft board can be used.

7. Horses must remain isolated from all animals not of equivalent health status during transport from the PEQ facility until arrival in Australia.

8. Insect netting must be carried on the flight at all times for contingencies. There must be sufficient insect netting to cover all air stalls completely. Insect netting must be in good condition to minimise entry of insect vectors into the air stalls.

9. An Australian government veterinarian may be required to accompany the shipment to Australia at the importer's expense.

10. The consignment may be accompanied by other horses of the equivalent health status or animals of other species only with the prior approval of AQIS.

11. The design of the air stalls, the recommended requirements for horses, the preparation for transport, and the disinfection of the interior of the aircraft, removable equipment, penning and containers must be in accordance with the recommendations of the Code and International Air Transport Association Live Animal Regulations unless otherwise agreed by AQIS.

Transit and transhipment

1. Horses must transit or tranship only at an approved airport. Any transhipment requires the prior approval of AQIS. Stops en route to Australia will need approval and permits from relevant authorities in the countries of transit and transhipment. Transit and transhipment times must not exceed six hours. Horses are not to leave the airport and must not be removed from their air stalls during transit or transhipment.

2. Horses must remain on board the aircraft at approved transit airports. Unauthorised personnel must not have contact with the horses. Cargo doors can be opened at approved transit airports to allow for unloading or loading of freight. Immediately after the cargo hold doors are closed, an approved knockdown aerosol insecticide must be sprayed throughout the cargo hold, in the manner recommended by the manufacturer.

3. In cases where horses in air stalls are to be unloaded, before opening the cargo door, the air stalls must be completely covered in netting to prevent insect access to the horse. The netting must remain in place until the horses are reloaded on an aircraft. Immediately after the horse is reloaded on an aircraft and the cargo hold doors are closed, an approved knockdown aerosol insecticide spray must be sprayed throughout the cargo hold in the manner recommended by the manufacturer. The insect netting must not be removed until 30 minutes after spraying.

Delayed takeoffs and unscheduled landings

1. Exporters or their agents must have contingency plans for the management of delayed takeoffs and unscheduled landings.

2. In the event that transit or transhipment exceeds six hours, AQIS must be notified immediately and the horses must not proceed to Australia without approval from AQIS. If the aircraft lands at any airport other than in an approved country, AQIS must be informed immediately. The decision as to whether the horses can continue to travel to Australia, and additional quarantine measures that may be required, will be made by AQIS on a risk-based case-by-case basis.

Arrival in Australia

1. Importers or their agents must have detailed SOPs to cover post-arrival procedures. These SOPs are to be developed in consultation with AQIS and must include roles and responsibilities for their staff, including grooms; cleaning and disinfection of air stalls, the area used to transfer horses to road transport at the airport, vehicles for transporting horses at the PAQ facility, and road transport arrangements including contingency plans for vehicle and equipment failures.

2. After horses arrive at an Australian airport they must be transferred from their air stalls onto vehicles for transporting horses, along with personnel and equipment, and proceed directly to the PAQ facility. AQIS door seals must be applied to vehicles for transporting horses to maintain biosecurity integrity during transport to the PAQ facility.

3. All personnel travelling with the horses on the aircraft and road transport, or that have had contact with the horses, quarantine risk material or air stalls, must undertake appropriate decontamination measures as specified by AQIS before leaving the airport or the PAQ facility if they are accompanying the horses to the PAQ facility.

4. Feed and water used during transport can travel with the horses to the PAQ facility for use only during PAQ.

5. All quarantine risk material (e.g. bedding, feed, water and waste material) remaining at the airport must be sealed in bags, ordered into quarantine and disposed of under AQIS supervision.

6. Air stalls must be secured at the airport in a manner that prevents release of quarantine risk material and cleaned and disinfected under AQIS supervision.

7. Vehicles for transporting horses from the port of entry to the PAQ facility must be cleaned and disinfected to the satisfaction of the AQIS quarantine officer

before loading the horses. AQIS must be advised of the transport route to the PAQ facility.

8. All equipment used during transport of the horses, and all baggage and personal equipment accompanying personnel, must be cleaned and disinfected under AQIS supervision before leaving the airport or the PAQ facility (if removed from the PAQ facility before the end of the PAQ period).

8.3.5 Post-arrival quarantine requirements

Post-arrival quarantine requirements for the importation of horses from Country X

Any variation from the **post-arrival quarantine requirements** must be specifically authorised by AQIS.

1. For horses originating from a single PEQ facility:

   The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

   nasopharyngeal samples must be taken from the horse four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

   OR

   For horses originating from multiple PEQ facilities within the same region:

   The horse must be held in PAQ for at least 14 days. During this time the horse must be isolated from equids not of equivalent health status **and**

   the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

   nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

   OR

   For horses originating from multiple PEQ facilities NOT within the same region:

   The horse must be held in PAQ for at least 21 days. During this time the horse must be isolated from equids not of equivalent health status **and**

   the period of intake of consignments into the PAQ facility should be kept to a minimum. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake **and**

nasopharyngeal samples must be taken from the horse within 24 hours of arrival into the PAQ facility and four to six days after commencement of PAQ and within four days of release from PAQ and tested using a polymerase chain reaction for influenza A virus with negative results in each case.

2. A single consignment must not be split between PAQ facilities on arrival in Australia.

3. A reference serum sample must be taken from the horse within 24 hours of arrival into the PAQ facility and stored at the National Animal Serum Bank at the Australian Animal Health Laboratory.

4. For the duration of PAQ the rectal temperature of the horse must be taken and recorded twice daily at least eight hours apart. If the temperature is 38.5 °C or higher on two consecutive recordings or other signs of respiratory disease are present, a nasopharyngeal sample must be taken and tested for influenza A virus and AQIS notified. If the temperature cannot been taken for any reason on two consecutive occasions, AQIS must be notified and a clinical examination by a registered veterinarian performed. Temperature records must be made available for inspection by AQIS.

5. Within 24 hours of arrival at the PAQ facility the horse must be thoroughly examined for ticks by a registered veterinarian under the direct supervision of the AQIS veterinarian. A systematic approach must be undertaken with close examination of ears, false nostrils, under-body areas (axilla, inguinal region and under the jawbone), perineum, mane and tail.

   AND

   If any horse in the PAQ facility is found to have ticks, all horses in the facility must be treated immediately, under the direct supervision of the AQIS veterinarian, with a parasiticide effective against ticks.

6. After completion of PAQ each imported pregnant mare must be held under quarantine surveillance until it has foaled and has been subsequently tested for *Taylorella equigenitalis* by culture with negative results. One sample must be collected from the clitoral fossa, including clitoral sinuses, and one sample from the endometrium or deep cervix during oestrus.

   NOTE: Foals must remain under quarantine surveillance until the dam has returned negative results for *T. equigenitalis* from each sample.

Location

1. The PAQ facility should be close to the port of arrival and be conveniently located for supervision by the AQIS veterinarian.

2. The facility must be located in an area that has been free from equine infectious anaemia during the previous 12 months.

Facilities

1. The PAQ facility must be surrounded by two secure stock-proof fences at least five metres apart, or a physical barrier providing equivalent security to prevent horses in PAQ having contact with animals outside the facility.

2. The PAQ facility including stables, yards, fences, feeding and watering arrangements must address animal welfare considerations.
3. Stables in the PAQ facility must be constructed so that they can be cleaned and disinfected.
4. The PAQ facility must provide a separation of at least 100 metres from other equids not of equivalent health status.
5. The PAQ facility must have a separate area for the cleaning and disinfection of vehicles for transporting horses, and facilities for the safe unloading and loading of horses.
6. The PAQ facility must have facilities for veterinary examination and the collection of samples.

Operation

1. The PAQ facility must be approved by AQIS before entry of any horse into the facility.
2. AQIS may audit the approved PAQ facility.
3. All PAQ operations and procedures must have detailed SOPs, consistent with a risk-based approach and approved by AQIS.
4. The process from the time horses arrive at the airport to the completion of PAQ must be auditable.
5. PAQ must be under the supervision of the AQIS veterinarian.
6. The AQIS veterinarian must inspect the PAQ facility before entry of any horse and must certify that the facility has been cleaned to his/her satisfaction.
7. The PAQ period will commence from the time of entry into the facility of the last horse of the PAQ intake.
8. During PAQ, the only horses in the facility must be those of the import consignment.
9. For the duration of PAQ the horse must not be held, housed or exercised within 100 metres of other equids not of equivalent health status.
10. Horses must not have the opportunity to mate while in PAQ.
11. Appropriate biosecurity procedures must be implemented for vehicles for transporting horses, freight containers, equipment and associated personnel, including transport operators, before, during and after the transport of horses to the PAQ facility.
12. Vehicles for transporting horses are not permitted to leave the PAQ facility until thoroughly cleaned and disinfected to the satisfaction of the AQIS quarantine officer.

13. Each imported horse must be identified on arrival at the PAQ facility and the accompanying veterinary certificate and passport examined and checked by AQIS.

14. Only personnel specifically authorised by AQIS are permitted to enter the PAQ facility. Details of all visitor entries must be recorded.

15. All personnel entering the PAQ facility during PAQ must wear dedicated or disposable outer clothing and dedicated, cleaned and disinfected or disposable footwear. All personnel must shower and change outer clothing before leaving the PAQ facility. Outer clothing and footwear used within the PAQ facility must be cleaned to the satisfaction of AQIS before removal from the facility.

16. All equipment used in feeding, handling and treating horses in PAQ must either be cleaned and disinfected to the satisfaction of AQIS before removal from the PAQ facility, or remain on-site for the duration of PAQ and then be released with AQIS approval at the completion of PAQ.

17. Other than inspections, visits and treatments required for certification, all veterinary visits, health problems, tests, test results and treatments must be reported to the AQIS veterinarian within 24 hours.

18. Any health problems affecting other animals on the facility undergoing PAQ must be reported to the AQIS veterinarian within 24 hours.

19. A detailed health record must be kept for each horse on the facility during the PAQ period and it must be available to the AQIS veterinarian.

20. The AQIS veterinarian must provide certification to AQIS, in the form of a checklist, that veterinary certificates and health records have been inspected and comply with the quarantine requirements.

21. Horses must not leave the facility during PAQ.

Besnoitiosis

Besnoitiosis in horses is caused by *Besnoitia bennetti* — a tissue cyst-forming coccidial parasite in the phylum Apicomplexa, family Sarcocystidae, subfamily Toxoplasmatinae. A disease of horses and donkeys, it was first described in Sudan in 1933 (Bennett, cited in Bigalke and Prozesky 2004). It has since been reported in other African countries (Bigalke and Prozesky 2004) and Central and North America (Terrell and Stookey 1973; Davis et al. 2003; Elsheikha et al. 2005; Elsheikha 2007).

Several *Besnoitia* spp. have been described according to the host range. The definitive host for many is thought to be the felid family and the intermediate hosts vary with each species. Based on observations of other *Besnoitia* spp. it is assumed that intermediate hosts become infected by the ingestion of mature isosporan-type oocysts shed in the faeces of felids. The sporozoites enter the circulation and multiply in endothelial cells, before forming cysts (Bigalke and Prozesky 2004). The definitive host is infected by ingesting tissues containing infective cysts and therefore transmission is not possible from a live horse. It has been indicated that blood-sucking insects, such as tabanids, play a minor role in the transmission of bovine besnoitiosis (*B. besnoiti*) from chronically infected cattle (Bigalke and Prozesky 2004).

Equine besnoitiosis is rare and sporadic and the epidemiology is unclear (Bigalke and Prozesky 2004). Horses and donkeys are intermediate hosts for *B. bennetti* and there is little information on the definitive hosts and modes of transmission.

In cattle, the incubation period is about four days and is followed by anorexia, photophobia, hyperaemia, anasarca, increased respiratory rate and pyrexia of around seven days duration. Within three to four weeks of the initial temperature rise, affected animals develop localised or widespread scleroderma, alopecia, dermatitis, lymphadenopathy, a mucopurulent nasal discharge and emaciation. Six to seven weeks after the initial pyrexia, cysts become visible in the skin and nasal mucosa, and although most animals survive, convalescence is slow and scleroderma and alopecia may be permanent. Only the sclerodermic stage has been observed in horses and recovery is usually more rapid than in cattle (Bigalke and Prozesky 2004).

Clinical diagnosis is confirmed by demonstration of large numbers of typical cysts in biopsies of the skin or conjunctiva (Sellon 2007). There are no reports on treatment of affected horses, however administration of trimethoprim and sulphamethoxazole for seven months was effective for treatment of a donkey (Dubey et al. 2005).

Besnoitiosis is not an OIE-listed disease (OIE 2009).

Besnoitiosis is present in some approved countries and cannot be transmitted by live horses.

Besnoitiosis was not considered further in the IRA.

References

Bigalke RD, Prozesky L (2004) Besnoitiosis. In *Infectious diseases of livestock* (eds. Coetzer JAW, Tustin RC) pp. 351-359. Oxford University Press, Oxford.

Davis WP, Peters DF, Dunstan RW (2003) Besnoitiosis in a miniature donkey. *Veterinary Dermatology* 8: 139-143.

Dubey JP, Sreekumar C, Donovan T, Rozmanec M, Rosenthal BM, Vianna MCB, Davis WP, Belden JS (2005) Redescription of *Besnoitia bennetti* (Protozoa: Apicomplexa) from the donkey (*Equus asinus*). *International Journal for Parasitology* 35: 659-672.

Elsheikha HM (2007) *Besnoitia bennetti* infection in miniature donkeys: an emerging protozoan of increasing concern. *Veterinary Parasitology* 145: 390-391.

Elsheikha HM, Mackenzie CD, Rosenthal BM, Marteniuk JV, Steficek B, Windsor S, Saeed AM, Mansfield LS (2005) An outbreak of besnoitiosis in miniature donkeys. *The Journal of Parasitology* 91: 877-881.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Sellon DC (2007) Miscellaneous parasitic diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 473-480. Saunders Elsevier, St. Louis.

Terrell TG, Stookey JL (1973) *Besnoitia bennetti* in two Mexican burros. *Veterinary Pathology* 10: 177-184.

Equine coronavirus

Coronaviruses infect a range of avian and mammalian species, including humans. They have a worldwide distribution (Büchen-Osmond 2006). There are three major antigenic groups of coronaviruses. Group 2 includes porcine haemagluttinating encephalomyelitis virus (HEV), bovine coronavirus (BCoV), human coronavirus OC43 (HCoV-OC43) and severe acute respiratory syndrome coronavirus (SARS-CoV). There is evidence of both a shared host range and the potential for recombination between viruses in Group 2. Although not listed by the International Committee on the Taxonomy of Viruses (Spaan et al. 2005), genome sequencing has identified a coronavirus in horses, equine coronavirus (ECoV), that is genetically distinct from BCoV and HEV (Zhang et al. 2007). ECoV has close antigenic relationships to other mammalian Group 2 coronaviruses (Davis et al. 2000; Guy et al. 2000; Zhang et al. 2007).

Coronaviruses cause respiratory, gastrointestinal, neurological and general infections in cattle, sheep, deer and horses, and have been associated with outbreaks of profuse, watery diarrhoea in calves (Durham et al. 1979) and foals (Bass and Sharpee 1975). Enteric coronavirus infections are generally self-limiting, but secondary complications can occur (Davis et al. 2000). Coronavirus infections are most frequently spread by respiratory, faecal–oral and mechanical transmission (Horzinek 1996). Vector transmission is not known to occur (Spaan et al. 2005).

BCoV antigens, coronavirus, coronavirus-like particles and ECoV have been isolated from the faeces of foals with fatal enterocolitis (Davis et al. 2000), neonatal diarrhoea (Bass and Sharpee 1975; Guy et al. 2000) primary severe combined immunodeficiency disease (Mair et al. 1990), and from an adult horse with Potomac fever (Huang et al. 1983).

In Australia, coronavirus is included as a differential diagnosis for diarrhoea and enteritis in foals (Hungerford 1990).

ECoV is not an OIE-listed disease (OIE 2009).

Equine coronavirus is present in approved countries and in Australia. The disease is not subject to official control in Australia.

ECoV was not considered further in the IRA.

References

Bass EP, Sharpee RL (1975) Coronavirus and gastroenteritis in foals. *The Lancet* 2: 822.

Büchen-Osmond C (2006) Coronaviridae. ICTVdb: The Universal Virus Database, version 4. http://www.ncbi.nlm.nih.gov/ICTVdb/ICTVdB/index.htm (Accessed 27 October 2008).

Davis E, Rush BR, Cox J, DeBey B, Kapil S (2000) Neonatal enterocolitis associated with coronavirus infection in a foal: a case report. *Journal of Veterinary Diagnostic Investigation* 12: 153-156.

Durham PJK, Stevenson BJ, Farquharson BC (1979) Rotavirus and coronavirus associated diarrhoea in domestic animals. *New Zealand Veterinary Journal* 27: 30-32.

Guy JS, Breslin JJ, Breuhaus B, Vivrette S, Smith LG (2000) Characterization of a coronavirus isolated from a diarrheic foal. *Journal of Clinical Microbiology* 38: 4523-4526.

Horzinek MC (1996) Coronaviridae. In *Virus infections of equines* (ed. Studdert MJ) pp. 201-202. Elsevier Science Publishers, Amsterdam.

Huang JC, Wright SL, Shipley WD (1983) Isolation of coronavirus-like agent from horses suffering from acute equine diarrhoea syndrome. *The Veterinary Record* 113: 262-263.

Hungerford TG (1990) Diseases of horses. In *Diseases of livestock* pp. 1023-1029. McGraw-Hill, Sydney.

Mair TS, Taylor FGR, Harbour DA, Pearson GR (1990) Concurrent cryptosporidium and coronavirus infections in an Arabian foal with combined immunodeficiency syndrome. *The Veterinary Record* 126: 127-130.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Spaan WJM, Brian D, Cavanagh D, de Groot RJ, Enjuanes L, Gorbalenya AE, Holmes KV, Masters P, Rottier P, Taguchi F, Talbot P (2005) Coronaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 947-964. Elsevier, San Diego.

Zhang J, Guy JS, Snijder EJ, Denniston DA, Timoney PJ, Balasuriya UBR (2007) Genomic characterization of equine coronavirus. *Virology* 369: 92-104.

Equine parainfluenza

Equine parainfluenza virus appears to have been reported only once when it was isolated from 48 young thoroughbred horses in Toronto, Canada, exhibiting signs of acute upper respiratory disease (Ditchfield et al. 1963). There have been no subsequent reports of the disease.

Equine clinical textbooks and journals ascribe doubtful significance to the disease due to the lack of reported cases (Coggins and Kemen 1975; Radostits et al. 1994; Jones et al. 1997; Pringle 2005). The International Committee on Taxonomy of Viruses does not list equine parainfluenza (Fauquet et al. 2005). Bovine parainfluenza virus 3 causes similar upper respiratory signs in calves, but it is unclear whether the viruses are related.

Canada is an approved country. There has been only one reported occurrence of equine parainfluenza in Canada — this occurred 45 years ago and caused mild and transitory clinical signs, and was restricted to Toronto.

Equine parainfluenza is not an OIE-listed disease (OIE 2009).

Equine parainfluenza is no longer present in any approved country.

Equine parainfluenza was not considered further in the IRA.

References

Coggins L, Kemen MJ (1975) Viral respiratory infections of horses: some specific viruses affecting the horse. *Journal of the American Veterinary Medical Association* 166: 80-83.

Ditchfield J, Zbitnew A, Macpherson LW (1963) Association of *Myxovirus para-influenzae* (RE 55) with upper respiratory tract infection of horses. *The Canadian Veterinary Journal* 4: 175-180.

Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA (2005) Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses. (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) Elsevier, San Diego.

Jones TC, Hunt RD, King NW (1997) Diseases caused by viruses. In *Veterinary pathology* (ed. Cann C) pp. 307-370. Lippincott Williams & Wilkins, Baltimore.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Pringle J (2005) Diseases of the upper respiratory tract. In *Large animal internal medicine* pp. 115-135. Blackwell Publishing, Ames.

Radostits OM, Blood DC, Gay CC (1994) Diseases caused by viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, sheep, pigs, goats and horses* (eds. Radostits OM, Blood DC, Gay CC) pp. 1034-1143. Bailliere, Tindall & Cassell, London.

Equine rhinitis A virus

Previously known as equine rhinovirus type 1, equine rhinitis A virus (ERAV), is a member of the aphthovirus genus in the family Picornaviridae together with foot-and-mouth disease virus (Stanway et al. 2005). ERAV has a worldwide distribution and is endemic in Australia. The virus is pathogenic for horses, guinea pigs, rabbits and monkeys but does not seem to spread horizontally in these species (Plummer 1963). Humans can also be affected, although the risk of humans acquiring ERAV infections is low (Kriegshäuser et al. 2005) ERAV has been identified as causing abortions in dromedaries in the United Arab Emirates (Wernery et al. 2008).

In horses, ERAV causes mild to severe upper respiratory tract infections and viraemia. Morbidity in affected stables can approach 100%, especially if there is a high proportion of young horses (Studdert 1996). Clinical signs of infection with ERAV in horses range from severe pharyngitis — with pyrexia, lymphadenitis and serous or mucopurulent nasal discharge — to transient pyrexia with mild or no other signs (Studdert 1996).

The virus is shed from the nasal cavity and in urine, and spreads readily by aerosol inhalation. Urine is important in transmission of the virus and horses can carry and shed the virus in urine for up to 147 days post-infection (McCollum and Timoney 1992). ERAV is resistant to environmental extremes and can readily contaminate the environment (Plummer 1962; McCollum and Timoney 1992).

In a survey in Australia of 291 thoroughbred horses aged from birth to 22 years, the prevalence of serum neutralising antibody to ERAV was 37% (Black et al. 2007).

ERAV is not an OIE-listed disease (OIE 2009).

ERAV is present in approved countries and in Australia. The disease is not subject to official control in Australia.

ERAV was not considered further in the IRA.

References

Black WD, Wilcox RS, Stevenson RA, Hartley CA, Ficorilli NP, Gilkerson JR, Studdert MJ (2007) Prevalence of serum neutralising antibody to equine rhinitis A virus (ERAV), equine rhinitis B virus 1 (ERBV1) and ERBV2. *Veterinary Microbiology* 119: 65-71.

Kriegshäuser G, Deutz A, Kuechler E, Skern T, Lussy H, Nowotny N (2005) Prevalence of neutralizing antibodies to *Equine rhinitis A* and *B* virus in horses and man. *Veterinary Microbiology* 106: 293-296.

McCollum WH, Timoney PJ (1992) Studies on the seroprevalence and frequency of equine rhinovirus-I and -II infection in normal horse urine. In *Equine infectious diseases VI: proceedings of the sixth international conference, July 7-11, 1991, Cambridge, UK,* (eds. Plowright W, Rossdale PD, Wade JF) pp. 83-88, R & W Publications (Newmarket) Limited, Newmarket.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Plummer G (1962) An equine respiratory virus with enterovirus properties. *Nature* 195: 519-520.

Stanway G, Brown F, Christian P, Hovi T, Hyypiä T, King AMQ, Knowles NJ, Lemon SM, Minor PD, Pallansch MA, Palmenberg AC, Skern T (2005) Picornaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on Taxonomy of Viruses* (eds. Fauquet M, Mayo A, Maniloff J, Desselberger U, Ball LA) pp. 757-778. Elsevier, San Diego.

Studdert MJ (1996) Equine rhinovirus infections. In *Virus infections of equines* (ed. Studdert MJ) pp. 213-217. Elsevier Science Publishers, Amsterdam.

Wernery U, Knowles NJ, Hamblin C, Wernery R, Joseph S, Kinne J, Nagy P (2008) Abortions in dromedaries *(Camelus dromedarius)* caused by equine rhinitis A virus. *Journal of General Virology* 89: 660-666.

Equine rhinitis B virus

Equine rhinitis B virus is a member of the erbovirus genus in the family Picornaviridae. It comprises two serotypes — equine rhinitis B virus 1 (ERBV1), formerly equine rhinovirus 2, and equine rhinitis B virus 2 (ERBV2), formerly equine rhinovirus 3. A third serotype, equine rhinitis B virus 3 (ERBV3) has been proposed (Stanway et al. 2005; Black and Studdert 2006; Dynon et al. 2007).

Equine rhinitis occurs in horses in Europe, Japan, North America and Australia (McCollum and Timoney 1992; Studdert 1996; Carman et al. 1997; Powell 1998a; Dynon et al. 2007). Clinical signs range from severe pharyngitis — with pyrexia, a serous or mucopurulent nasal discharge, and swelling and abscessation of lymph nodes — to transient pyrexia with no other signs (Powell 1998b). The virus replicates in the upper respiratory tract and there is no viraemia. Serum neutralisation antibodies are not completely protective. Reinfection and recrudescent infections are significant features of the epidemiology of equine rhinitis, particularly in horses under 12 months of age (Black et al. 2007b).

Seroprevalence studies in racing stables and on studs and properties in Victoria and the Hunter Valley, New South Wales, have demonstrated that ERBV1 and ERBV2 are widespread among the horses sampled (Dynon et al. 2007). ERBV1, ERBV2 and the proposed ERBV3 have been isolated in Australia — using reverse transcriptase-polymerase chain reaction and cell culture — from horses showing similar clinical signs of infection (Black et al. 2007a; Black et al. 2007b).

Equine rhinitis B virus is not an OIE-listed disease (OIE 2009).

ERBV is present in approved countries and in Australia. The disease is not subject to official control in Australia.

ERBV was not considered further in the IRA.

References

Black WD, Hartley CA, Ficorilli NP, Studdert MJ (2007a) Reverse transcriptase-polymerase chain reaction for the detection of equine rhinitis B viruses and cell culture isolation of the virus. *Archives of Virology* 152: 137-149.

Black WD, Studdert MJ (2006) Formerly unclassified, acid-stable equine picornaviruses are a third equine rhinitis B virus serotype in the genus *Erbovirus*. *Journal of General Virology* 87: 3023-3027.

Black WD, Wilcox RS, Stevenson RA, Hartley CA, Ficorilli NP, Gilkerson JR, Studdert MJ (2007b) Prevalence of serum neutralising antibody to equine rhinitis A virus (ERAV), equine rhinitis B virus 1 (ERBV1) and ERBV2. *Veterinary Microbiology* 119: 65-71.

Carman S, Rosendal S, Huber L, Gyles C, McKee S, Willoughby RA, Dubovi E, Thorsen J, Lein D (1997) Infectious agents in acute respiratory disease in horses in Ontario. *Journal of Veterinary Diagnostic Investigation* 9: 17-23.

Dynon K, Black WD, Ficorilli N, Hartley CA, Studdert MJ (2007) Detection of viruses in nasal swab samples from horses with acute, febrile, respiratory disease

using virus isolation, polymerase chain reaction and serology. *Australian Veterinary Journal* 85: 46-50.

McCollum WH, Timoney PJ (1992) Studies on the seroprevalence and frequency of equine rhinovirus-I and -II infection in normal horse urine. In *Equine infectious diseases VI: proceedings of the sixth international conference, July 7-11, 1991, Cambridge, UK,* (eds. Plowright W, Rossdale PD, Wade JF) pp. 83-88, R & W Publications (Newmarket) Limited, Newmarket.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Powell DG (1998a) Diagnosis of equine viral disease. In *Equine infectious diseases: the A T Reid memorial refresher course for veterinarians, 23-27 February, 1998, University of Sydney,* pp. 87-93, The University of Sydney, The Post-Graduate Foundation in Veterinary Science, Sydney.

Powell DG (1998b) Respiratory disease caused by strangles, adeno and rhinovirus. In *Equine infectious diseases: proceedings 298, February 23-27, 1998, Sydney, Australia,* pp. 21-26, Post Graduate Foundation in Veterinary Science, University of Sydney, Sydney.

Stanway G, Brown F, Christian P, Hovi T, Hyypiä T, King AMQ, Knowles NJ, Lemon SM, Minor PD, Pallansch MA, Palmenberg AC, Skern T (2005) Picornaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on Taxonomy of Viruses* (eds. Fauquet M, Mayo A, Maniloff J, Desselberger U, Ball LA) pp. 757-778. Elsevier, San Diego.

Studdert MJ (1996) Equine rhinovirus infections. In *Virus infections of equines* (ed. Studdert MJ) pp. 213-217. Elsevier Science Publishers, Amsterdam.

Equine torovirus

Equine torovirus (Berne virus strain P138/72) was isolated from a horse in Berne, Switzerland in 1972, during a routine diagnostic evaluation for gastrointestinal disease (Weiss et al. 1983). It is classified in the Torovirus genus, within the family Coronaviridae and is antigenically related to bovine torovirus and to torovirus-like particles found in human faecal specimens (Spaan et al. 2005). Reports of seropositive horses suggest a worldwide distribution and there is a higher prevalence in adult than in younger horses (Weiss et al. 1984; Weiss and Horzinek 1996).

Serological evidence indicates that equine torovirus also infects cattle, sheep, goats, pigs, rabbits and some species of mice(Weiss et al. 1984).

No clinical disease has been associated with infection with equine torovirus (Rao and Chandra 2002). Despite a high percentage of seropositive adult horses in Switzerland, there have been no reports of clinical disease. Experimental inoculation of Berne virus into two foals induced neutralising antibody, but did not cause any clinical signs of disease (Weiss et al. 1984).

Equine torovirus is not an OIE-listed disease (OIE 2009).

Equine torovirus is present in approved countries but there are no reports of clinical disease associated with infection. It was considered unlikely to cause disease or have any measureable consequences if it were introduced into Australia.

Equine torovirus was not considered further in the IRA.

References

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Rao VDP, Chandra R (2002) Equine torovirus infection: an emerging problem. *Centaur* 18: 61-65.

Spaan WJM, Brian D, Cavanagh D, de Groot RJ, Enjuanes L, Gorbalenya AE, Holmes KV, Masters P, Rottier P, Taguchi F, Talbot P (2005) Torovirus. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Taxonomy of Viruses* (eds. Fauquet M, Mayo A, Maniloff J, Desselberger U, Ball LA) pp. 956-964. Elsevier, San Diego.

Weiss M, Horzinek MC (1996) Torovirus infections. In *Virus infections of equines* (ed. Studdert MJ) pp. 205-209. Elsevier Science Publishers, Amsterdam.

Weiss M, Steck F, Horzinek MC (1983) Purification and partial isolation of a new enveloped RNA virus (Berne virus). *Journal of General Virology* 64: 1849-1858.

Weiss M, Steck F, Kaderli R, Horzinek MC (1984) Antibodies to Berne virus in horses and other animals. *Veterinary Microbiology* 9: 523-531.

Getah virus

Getah virus is part of the Semliki Forest antigenic complex of alphaviruses in the family Togaviridae (Weaver et al. 2005).The virus has been reported in southeast Asia including Hong Kong and Japan. It causes clinical disease in horses and pigs and in experimentally infected mice (Radostits et al. 2007). Studies in the 1960s reported that Getah virus antibodies were identified in Australian animals (Doherty et al. 1966) and the virus was detected in mosquitoes (Doherty et al. 1963). However, current serological techniques that differentiate between antibodies to Getah virus and antibodies to the closely-related Ross River virus have not confirmed the presence of Getah virus in Australia (Geering et al. 1995; Radostits et al. 2007; Hinchcliff 2007). Furthermore, no clinical cases have been reported in Australia (Radostits et al. 2007) and Australia is considered free from the virus (Animal Health Australia 2005).

The life cycle of Getah virus has not been elucidated and it has not been confirmed that horses can infect mosquitoes (Hinchcliff 2007). Getah virus is transmitted via *Aedes* spp. and *Culex* spp. mosquitoes and is maintained in a cycle between mosquitoes and vertebrate hosts in regions where there is year-round mosquito activity (Fukunaga et al. 2000). The mechanism of maintenance in other areas is not known and there are no reports of trans-stadial or transovarial transmission in mosquitoes. *Culex gelidus*, one species of mosquito from which Getah virus has been isolated in Asia, has been identified in the Northern Territory (Whelan et al. 2000). The definitive amplifying host (or hosts) is/are unknown, although a number of vertebrates including horses, pigs and cattle can be infected by the virus (Geering et al. 1995; Radostits et al. 2007; Hinchcliff 2007).

Getah virus has not been recovered from faeces or urine of infected horses and there are no reports of vertical transmission in horses (CFSPH 2006; Radostits et al. 2007). Although direct transmission is feasible, the concentration of virus in secretions of naturally infected horses is unlikely to be an infectious dose. Results from experimental infections of horses suggest that transmission through direct contact via nasal and oral secretions is unlikely in natural outbreaks (Kamada et al. 1991).

Getah virus infection in horses is usually subclinical and self-limiting (Radostits et al. 2007). Signs of clinical disease last for 7–10 days and include pyrexia, hind limb oedema, stiffness, submandibular lymphadenopathy, skin eruptions and urticaria (CFSPH 2006). Mortality from Getah virus infections is rare (Radostits et al. 2007).

An inactivated vaccine, combined with a Japanese encephalitis vaccine, is registered for use in horses in Japan and is considered effective (Sugiura and Shimada 1999).

Getah virus disease is not an OIE-listed disease (OIE 2009).

Getah virus is present in some approved countries but there is no evidence of natural transmission from horses to potential mosquito vectors nor directly to horses or other vertebrates.

Getah virus was not considered further in the IRA.

References

Animal Health Australia (2005) Getah virus. Animal Health Australia. http://www.animalhealthaustralia.com.au/programs/adsp/nahis/diseases/getah.cfm (Accessed 26 February 2009).

CFSPH (2006) Getah virus infection. The Center for Food Security and Public Health, Iowa State University. http://www.cfsph.iastate.edu/diseaseinfo/factsheets.htm (Accessed 6 January 2008).

Doherty RL, Carley JG, Mackerras MJ, Marks EN (1963) Studies of arthropod-borne virus infections in Queensland. III. isolation and characterization of virus strains from wild-caught mosquitoes in North Queensland. *The Australian Journal of Experimental Biology and Medical Science* 41: 17-39.

Doherty RL, Gorman BM, Whitehead RH, Carley JG (1966) Studies of arthropod-borne virus infections in Queensland. V. survey of antibodies to group A arboviruses in man and other animals. *Australian Journal of Experimental Biology and Medical Science* 44: 365-377.

Fukunaga Y, Kumanomido T, Kamada M (2000) Getah virus as an equine pathogen. *Veterinary Clinics of North America: Equine Practice* 16: 605-617.

Geering WA, Forman AJ, Nunn MJ (1995) Getah virus disease. In *Exotic diseases of animals: a field guide for Australian veterinarians* pp. 132-134. Australian Government Publishing Service, Canberra.

Hinchcliff KW (2007) Miscellaneous viral diseases. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 233-235. Saunders Elsevier, St. Louis.

Kamada M, Kumanomido T, Wada R, Fukunaga Y, Imagawa H, Sugiura T (1991) Intranasal infection of Getah virus in experimental horses. *The Journal of Veterinary Medical Science* 53: 855-858.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Radostits OM, Gay CC, Hinchcliff KW, Constable PD (2007) Diseases associated with viruses and chlamydia II. In *Veterinary medicine: a textbook of the diseases of cattle, horses, sheep, pigs and goats* (eds. Radostits OM, Gay CC, Hinchcliff KW, Constable PD) pp. 1307-1437. Saunders Elsevier, Edinburgh.

Sugiura T, Shimada K (1999) Seroepizootiological survey of Japanese encephalitis virus and Getah virus in regional horse race tracks from 1991 to 1997 in Japan. *The Journal of Veterinary Medical Science* 61: 877-881.

Weaver SC, Frey TK, Huang HV, Kinney RM, Rice CM, Roehrig JT, Shope RE, Strauss EG (2005) Togaviridae. In *Virus taxonomy: classification and nomenclature of viruses: eighth report of the International Committee on the Taxonomy of Viruses* (eds. Fauquet CM, Mayo MA, Maniloff J, Desselberger U, Ball LA) pp. 999-1008. Elsevier, San Diego.

Whelan P, Hayes G, Carter J, Wilson A, Haigh B (2000) Detection of the exotic mosquito *Culex gelidus* in the Northern Territory. *Communicable Diseases Intelligence* 24: 74-75.

Nagana

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

The three trypanosome species causing nagana — *Trypanosoma brucei brucei*, *T. congolense* and *T. vivax* — are usually transmitted by *Glossina* spp. (tsetse fly). Despite possible mechanical transmission, the epidemiology of nagana depends primarily on the species of the various tsetse fly vectors, their distribution, abundance, and host-feeding preferences. Trypanosomes can survive for long periods in the mammalian host, maximising opportunity for transmission by vectors. It is not likely that either *T. brucei* or *T. congolense* could establish outside the range of their usual vectors.

T. vivax was introduced into South America with the importation of cattle from Africa and the disease has not been reported in horses outside of Africa (Osorio et al. 2008). *T. vivax* has become extensively established in regions beyond the range of the tsetse fly, in south and central America and parts of the Caribbean. Mechanical transmission by biting flies is of particular interest in Australia because of the presence of suitable vectors of the genera *Tabanus* and *Stomoxys* in some regions.

Australian marsupials and monotremes harbour endemic trypanosomes (O'Donoghue and Adlard 2000), without apparent ill effect (Stevens et al. 1998). Two species of wallaby are highly susceptible to experimental infection with *T. evansi*, a species closely related to *T. brucei brucei*, resulting in acute disease and high mortality (Reid et al. 2001). These and other macropods have the potential to spread *T. evansi* if introduced into Australia.

Experimental infection of horses with *T. vivax* causes acute disease with parasitaemic peaks corresponding with fluctuations in temperature, oedema and anaemia typical of trypanosomal disease (Stephen 1986).

Tsetse-transmitted trypanosomosis is an OIE-listed disease of cattle (OIE 2009). There are no recommendations in the Code for the importation of animals for tsetse-transmitted trypanosomosis. There is a chapter in the OIE Manual on the disease relating to the disease in cattle (OIE 2008).

Nagana is not present in any approved country.

Nagana was not considered further in the IRA.

References

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

O'Donoghue PJ, Adlard RD (2000) Catalogue of protozoan parasites recorded in Australia. *Memoirs of the Queensland Museum* 45: 1-163.

OIE (2008) Trypanosomosis (tsetse-transmitted). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals 2009. http://www.oie.int/eng/normes/mmanual/2008/pdf/2.04.18_TRYPANOSOMOSIS.pdf (Accessed 1 September 2009).

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

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

Reid SA, Husein A, Partoutomo S, Copeman DB (2001) The susceptibility of two species of wallaby to infection with *Trypanosoma evansi*. *Australian Veterinary Journal* 79: 285-288.

Stephen LE (1986) *Trypanosoma (Duttonella) Vivax* Ziemann, 1905. In *Trypanosomiasis: a veterinary perspective* pp. 19-68. Pergamon Press, Oxford.

Stevens J, Noyes H, Gibson W (1998) The evolution of trypanosomes infecting humans and primates. *Memoirs of Instituto Oswaldo Cruz (Rio de Janeiro)* 93: 669-676.

Stomach fluke

Stomach fluke, *Gastrodiscus aegyptiacus*, is a trematode belonging to the family Gastrodiscidae. Horses, donkeys, mules and pigs are the definitive hosts. Two species of snails, *Bulinus forskalii* and *B. sengalensis*, are their intermediate hosts. The parasites and their intermediate hosts occur in Africa, the Middle East and Asia (Kassai 1999). It has not been reported in Australia.

G. aegyptiacus is not an OIE-listed disease (OIE 2009).

The lifecycle of *G. aegyptiacus* is similar to other trematodes, with cercariae developing in snails. Immature and adult *G. aegyptiacus* are found in the large and small intestines of horses and are commonly of little clinical significance. Rarely, immature trematodes have been reported to cause a severe and hyperacute, possibly fatal, colitis in horses.

G. aegyptiacus is not reported in any approved country. Intermediate hosts are not present in Australia.

G. aegyptiacus was not considered further in the IRA.

References

Kassai T (1999) Phylum: Platyhelminthes flatworms. In *Veterinary helminthology* pp. 3-50. Butterworth-Heinemann, Oxford.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Warble-fly myiasis

Warble-fly myiasis — also known as hypodermosis or warbles — is caused by invasion of the host by larvae of *Hypoderma* spp. Adult *Hypoderma* spp. are known as 'heel', 'warble', 'bomb' or 'gad' flies. Usual hosts are cattle and Old World deer. However, *Hypoderma* spp. have been known to parasitise horses, humans, bison, goats and sheep. Warbles affects cattle in Asia, Europe and North America, between the latitudes 25° and 60°N (Arundel and Sutherland 1988). Larvae have been diagnosed and treated in cattle several times in Australia during post-arrival quarantine and post-quarantine surveillance but the fly has never established in Australia (Geering et al. 1995).

Infestation of cattle occurs during spring and summer when *Hypoderma* spp. flies deposit eggs on legs of animals. Hatching within four to seven days, the first instar larvae penetrate the skin and migrate over four weeks along fascial planes to the oesophagus (*H. lineatum*) or spine (*H. bovis*). They remain in these sites for several months over winter, maturing to second instar larvae, before moving to the sub-dermal tissues of the back where they produce the characteristic soft, painful swellings of 'warbles' (Soulsby 1982). Larvae mature in the warbles to the final fifth instar stage, after which they emerge, fall to the ground and pupate. Adult flies emerge 3–5 weeks later (Scharff 1973; Berkenkamp and Drummond 1990).

In horses, larvae seldom develop beyond the second instar (Scharff 1973; Soulsby 1982) and are unable to complete their life cycle (Radostits et al. 1994; Barbet 2007).

Rose (cited in Olander 1967) first described warble-fly myiasis in horses in England in 1842 and it has subsequently been reported in most parts of the world where *Hypoderma* spp. are prevalent, including Denmark, France, Germany, Italy, Sweden (Olander 1967) and the United States (Scharff 1973). There are several reports of spinal and intracranial myiasis in horses due to *Hypoderma* spp. (Olander 1967; Hadlow et al. 1977). Warbles along the back of horses have been described in the United States, where the incidence varied from 27% to 60% in some areas (Baker and Monlux 1939; Berkenkamp and Drummond 1990).

Diagnosis of warbles is based on identifying larvae in lesions along the back. In summer, eggs can be found on hairs especially on legs.

Warble-fly myiasis is not an OIE-listed disease (OIE 2009).

Warble-fly myiasis has not been reported in horses in approved countries for more than 25 years and the horse is a dead-end host.

Warble-fly myiasis was not considered further in the IRA.

References

Arundel JH, Sutherland AK (1988) *Ectoparasitic diseases of sheep, cattle, goats and horses*. Australian Government Publishing Service, Canberra.

Baker DW, Monlux WS (1939) Hypoderma myiasis in the horse: summary of a series of cases studied during spring and summer. *The Journal of Parasitology* 25: 16. (Abstract only)

Barbet J (2007) Ectoparasites of horses. In *Equine infectious diseases* (eds. Sellon DC, Long MT) pp. 500-509. Saunders Elsevier, St. Louis.

Berkenkamp SD, Drummond RO (1990) Hypodermosis: part I. *Compendium on Continuing Education for the Practicing Veterinarian* 12: 740-746, 754.

Geering WA, Forman AJ, Nunn MJ (1995) *Exotic diseases of animals: a field guide for Australian veterinarians*. Australian Government Publishing Service, Canberra.

Hadlow WJ, Ward JK, Krinsky WL (1977) Intracranial myiasis by *Hypoderma bovis* (Linnaeus) in a horse. *The Cornell Veterinarian* 67: 272-281.

OIE (2009) OIE listed diseases. World Organisation for Animal Health (OIE). http://www.oie.int/eng/maladies/en_classification2009.htm?e1d7 (Accessed 12 March 2009).

Olander HJ (1967) The migration of *Hypoderma lineatum* in the brain of a horse: a case report and review. *Veterinary Pathology* 4: 477-483.

Radostits OM, Blood DC, Gay CC (1994) Diseases caused by arthropod parasites. In *Veterinary medicine: a textbook of the diseases of cattle, sheep, pigs, goats and horses* (eds. Radostits OM, Blood DC, Gay CC) pp. 1280-1309. Bailliere, Tindall & Cassell, London.

Scharff DK (1973) Control of cattle grubs in horses. *Veterinary Medicine: Small Animal Clinician* 86: 791-792.

Soulsby EJL (1982) Arthropods. In *Helminths, arthropods and protozoa of domesticated animals* pp. 357-504. Baillière Tindall, London.