Import risk analysis:
horses and horse semen

20 January 2000

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Biosecurity Authority
Ministry of Agriculture and Forestry
Wellington
NEW ZEALAND

20 January 2000

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Import risk analysis: horses and horse semen *

20 January 2000

Approved for general release

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1 Health status of various countries for the OIE List A and B diseases of concern

Acronyms

ag-ELISA	Quarantine and Inspection Service
AWAC	Animal Welfare Advisory Committee, New Zealand
BD	Borna disease
BHK	baby hamster kidney (cell line)
CAHL	Central Animal Health Laboratory, MAF (now National Centre for Disease Investigation, NCDI)
CEM	contagious equine metritis
c-ELISA	competitive enzyme-linked immunosorbent assay
CFT	complement fixation (test)
CSF	cerebrospinal fluid
DAB	Director of Animal Biosecurity (formerly Chief Veterinary Officer), New Zealand Ministry of Agriculture and Forestry
EE	equine encephalosis
EEE	eastern equine encephalomyelitis
EI	equine influenza
EIA	equine infectious anaemia
ELISA	enzyme-linked immunosorbent assay
EPM	equine protozoal myeloencephalitis
EU	European Union
EVA	equine viral arteritis
FEI	Federation Equestre Internationale
GV	Getah virus
HBLB	Horserace Betting Levy Board, UK
HeV	Hendra virus
HI	haemagglutination inhibition (test)
IATA	International Air Transport Association
i-ELISA	indirect enzyme-linked immunosorbent assay
JE	Japanese encephalitis
lp-ELISA	liquid phase blocking enzyme-linked immunosorbent assay
MAF	New Zealand Ministry of Agriculture and Forestry
MSI	mouse subinoculation (test)
NCDI	National Centre for Disease Investigation, MAF (formerly Central Animal Health Laboratory, CAHL)
np-ELISA	nucleoprotein enzyme-linked immunosorbent assay
OIE	Office International des Epizooties (International Organisation for Animal Health)
PAQ	post-arrival quarantine
PCR	polymerase chain reaction
ppd	purified protein derivative
PRN	plaque reduction neutralisation (test)
RT-PCR	reverse transcriptase polymerase chain reaction
RRV	Ross River virus
sa-ELISA	synthetic antigen enzyme-linked immunosorbent assay
SPS Agreement	Agreement on the Application of Sanitary and Phytosanitary Measures
SRD	single radial immunodiffusion
SRH	single radial haemolysis
UK	United Kingdom
USA	United States of America
USDA	United States Department of Agriculture
VEE	Venezuelan equine encephalomyelitis
VN	virus neutralisation (test)
VS	vesicular stomatitis

WEE	western equine encephalomyelitis
WTO	World Trade Organisation

1 EXECUTIVE SUMMARY

The Ministry of Agriculture and Forestry (MAF) has completed a risk analysis examining the disease risks associated with importation into New Zealand of live horses and horse semen. The risk analysis was released for public consultation in May 1998. MAF received and considered public submissions prior to finalising the risk analysis, and this process is recorded in a separate document. The risk analysis provides the risk management recommendations for a review of import health standards for importation of live horses and horse semen.

The risk analysis considers the disease risks associated with imports of live animals and semen of the species horses, *Equus caballus*, and donkeys, *Equus asinus*. The risk analysis may also be relevant during development of import health standards for zoo equidae, such as zebra, although some further investigation of relevant clinical, epidemiological and diagnostic features of diseases in such species may be required.

The disease risks are considered pursuant to the Biosecurity Act 1993, which requires import health standards safeguarding New Zealand's biosecurity to be developed through processes involving consultation with stakeholders. The principal biosecurity concern during the development of import health standards is preventing the introduction of unwanted organisms.

Obligations with respect to measures in import health standards arise from New Zealand's membership of the World Trade Organisation (WTO) and the *Agreement on the application of sanitary and phyto-sanitary measures* (the SPS Agreement). One such obligation is harmonisation with the Office International des Epizooties (OIE) *International Animal Health Code* (the *Code*) recommendations for sanitary measures to prevent the spread of OIE list A and list B diseases during trade in horses and horse semen. Scientifically-based import risk analysis must justify sanitary measures that vary from the OIE *Code* recommendations.

A process of hazard identification has developed a list of the diseases of concern. The list comprises the infectious diseases affecting horses that constitute a risk during international trade in horses and equine products. Diseases endemic in New Zealand that are not subject to official control are not considered further, fulfilling the SPS Agreement obligation regarding consistency with national treatment.

The risk assessment presents a monograph for each disease of concern. Each monograph reviews the epidemiology of the disease, including distribution, clinical signs, transmission, diagnosis and treatment. This information is then used to provide a risk estimate by considering the risk of release (the disease agent being present at the time horses or semen are imported), exposure (the likelihood of spread in New Zealand if imported) and for adverse consequences to follow either of these events. Finally, risk management measures (safeguards) are recommended; these have been summarised in the following section.
The measures are those considered to be least trade restrictive while providing an appropriate level of protection from risk, commensurate with the likelihood and consequences of disease introduction and establishment.

Where available, empirical data relevant to the magnitude of a disease risk or the effect of a risk reduction measure are presented. However, the disease risk estimates have not been quantified. This format provides a transparent basis for measures to be included into import health standards for horses and semen, and allows for future updates as new information becomes available.

A specific measure recommended for certain diseases is post-arrival quarantine (PAQ). PAQ has not previously been a feature of New Zealand's import health policy for horses. The risk associated with equine influenza is of particular relevance to the discussion of post-arrival quarantine. Only Australia and New Zealand have major equine populations that are free of equine influenza. Any occurrence here would be likely to manifest as explosive outbreaks with severe financial consequences for the equine industries. Under these circumstances, a cautious approach to risk management is warranted. Post-arrival confinement of horses from countries where the disease occurs presents the best chance of containing an outbreak following importation of an infected horse.

The analysis has considered importation of horses to compete in equestrian events and invitation races (competition horses). Such horses must arrive in a state of competition fitness, and this is generally incompatible with requirements for long periods of isolation before and after importation. Appropriate exercise facilities are rarely available in isolation facilities, and the analysis recommends that such horses should be allowed supervised access to exercise facilities outside of isolation facilities so long as isolation from other horses is maintained. Particular post-arrival measures for competition horses, such as restricting sexual contact, will effectively manage the risks for diseases transmitted venereally, such as contagious equine metritis, equine viral arteritis and dourine. Similarly, diseases transmitted by arthropods exotic to New Zealand, such as piroplasmosis, may be managed by ensuring that iatrogenic transmission does not occur, and that the horses are eventually re-exported.

When horse semen is imported, the health status of donor stallions at the time of semen collection must be accurately determined to ensure the safety of imported product. This requires specific measures directed at the donor stallion, but the health status, location, technical facilities and operational requirements of the semen collection centre may also influence the disease risk. These aspects have been covered within a standard for semen collection centres collecting horse semen for importation into New Zealand.

2 SUMMARY OF RECOMMENDATIONS

2.1 LIVE HORSES

African horse sickness

Either:

1. The horses were resident since birth, or at least the previous 2 months, in a country that is free of AHS according to the criteria within Article 2.1.11.2 of the OIE Code.

Or:

1. AHS occurs in the exporting country; *and*

2. Either: i) The horses were resident prior to export in an approved AHS free zone;
Or: ii) The date of export and the 28 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export;
Or: iii) The horses were kept in a pre-export isolation facility for the 28 days (35 days in the case of donkeys) prior to export, and protected from vectors during this period and during movement to the port of export. (Protection from vectors shall comprise confinement within arthropod-proof stables at all times, with the exception of officially supervised exercise sessions between the period 2 hours after sunrise and 2 hours prior to sunset. Prior to these sessions the horses shall receive prophylactic treatment with an approved insect repellent.); *and*

3. The horses were subjected to an indirect ELISA or CFT for AHS during the 28 days prior to export on two occasions not less than 21 days apart, and demonstrated a negative, stable or declining antibody titre; *and*

4. The horses were showing no clinical signs of AHS on the day of export; *and*

5. Upon arrival in New Zealand the horses were subjected to a minimum 14 day period of post-arrival quarantine prior to biosecurity clearance.

Vesicular stomatitis

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free of VS according to the criteria in Article 2.1.2.2. of the OIE Code.

Or:

1. When importing from countries where VS occurs but approved surveillance systems are in place to provide rapid detection and on-going monitoring:
Either: i) the horses were resident since birth, or at least the 21 days prior to export, in a

part of the territory of the country where VS has not occurred during the previous 2 years;
Or: ii) The date of export and the 21 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident; *and*

2. During the 21 days prior to export the horses were subjected to a LP-ELISA, c-ELISA or VN test for VS. In the case of any positive result, all in-contact horses should be re-tested not less than 14 days subsequently. The results of testing should indicate all horses have negative, stable or declining titres; *and*

3. The horses were showing no clinical signs of VS on the day of export.

Or:

1. When importing from countries where VS occurs, the horses were kept in a pre-export isolation facility for the 21 days prior to export, and protected from vectors during this period and during movement to the port of export; *and*

2. The horses were subjected to a LP-ELISA, c-ELISA or VN test for VS during the 21 days prior to export. In the case of any positive result, all horses should be re-tested not less than 14 days subsequently. The results of testing should indicate all horses have negative, stable or declining titres; *and*

3. The horses were showing no clinical signs of VS during the 21 day period prior to export and on the day of export; *and*

4. Upon arrival in New Zealand the horses were subjected to a minimum 21 day period of post-arrival quarantine in an insect-proof facility prior to biosecurity clearance.

Venezuelan equine encephalomyelitis

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free from VEE according to the criteria within Article 3.4.12.2 of the OIE Code.

Or:

1. When importing from countries where VEE occurs, the horses were kept in a pre-export isolation facility during the minimum 21 days prior to export, and protected from vectors during this period and during movement to the port of export; *and*

2. Either i) The horses were fully vaccinated against VEE (two doses given 2-4 weeks apart as a primary regime, followed by annual revaccination, using an inactivated vaccine for VEE either alone or in combination with EEE and WEE) not less than 60 days and not more than 1 year prior to export;
Or ii) The horses were subjected to the CF, HI, PRN or IgM capture ELISA for VEE, not less than 7 days after entering pre-export isolation. If any positive results were recorded, all

horses were subjected to a repeat test not less than 14 days subsequently. The results must indicate all horses had negative, stable or declining antibody titres; *and*

3. The horses were showing no clinical signs of VEE during pre-export isolation and on the day of export; *and*

4. Upon arrival in New Zealand the horses were subjected to a minimum 7 day period of post-arrival quarantine in an insect-proof facility.

Eastern and Western equine encephalomyelitis

Either:

1. In the case of all horses, they were kept during the 21 days prior to export on premises where cases of equine encephalomyelitis have not occurred during that period; *and*

2. The horses were showing no clinical signs of equine encephalomyelitis on the day of export.

Or:

1. When importing from countries within the Americas, the horses were vaccinated against EEE and WEE (two doses given 2-4 weeks apart as a primary regime, followed by annual revaccination, using an inactivated vaccine for EEE and WEE either alone or in combination with VEE) not less than 60 days and not more than 1 year prior to the scheduled date of export; *and*

2. The horses were kept during the 3 months prior to export on premises where cases of equine encephalomyelitis have not occurred during that period; *and*

3. The horses were showing no clinical signs of equine encephalomyelitis on the day of export.

Equine infectious anaemia

1. EIA is a notifiable disease in the exporting country; *and*

2. The horses were kept for the 3 months prior to export on premises where EIA has not occurred during that period; *and*

3. The horses were subjected to the AGID or c-ELISA test for EIA during the 21 days prior to export, with negative results (unless being re-imported into New Zealand from Australia after a visit of less than 21 days); *and*

4. The horses were showing no clinical signs of EIA on the day of export; *and*

5. When the prevalence of EIA in the exporting country is assessed as medium to high, upon

arrival in New Zealand the horses were subjected to a minimum 7 day period of post-arrival quarantine, during which time they were tested for EIA using the AGID or c-ELISA with negative results, prior to biosecurity clearance.

Equine influenza

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free from EI.

Or:

1. When importing from countries where EI occurs, the horses were kept for the 3 months prior to export on premises where EI has not occurred during that period; *and*

2. During the 4 months prior to export the horses (except for foals less than 2 months old and accompanied by their vaccinated dam) were vaccinated against EI using an approved inactivated vaccine either twice not less than 21 days apart, or once as a booster to a previous primary course of vaccination;
(**N.B.** Approved vaccines must contain a Prague/56-like virus as the equine-1 (H7N7) component; either Suffolk/89 or a Newmarket/2/93-like virus as the European equine-2 (H3N8) component; and a Kentucky/94-like virus as the American equine-2 (H3N8) component.) *and*

3. The horses were subjected to a SRH test during the 30 days prior to entering pre-export isolation (or upon entry into isolation) and demonstrated a protective level of antibodies against EI (>150mm^2 or relative antibody concentration of > 44); *and*

4. The horses were kept for a minimum 21 day period prior to export in a pre-export isolation facility; *and*

5. Upon arrival in New Zealand the horses were subjected to a minimum 14 day period of post-arrival quarantine. No equidae should be allowed within 100 metres of the facility, and any quarantined horses exhibiting respiratory symptoms should be confined indoors until subjected to an antigen ELISA for EI with a negative result.

Equine viral abortion

1. The horses were kept for the 3 months prior to export on premises where equine viral abortion (EHV-1, including neurological disease) has not occurred during that period; *and*

2. The horses were showing no clinical sign of equine viral abortion (EHV-1, including neurological disease) on the day of export.

Equine viral arteritis

Either:

1. When female, castrated male and competition horses are imported, the horses were kept for the 3 months prior to export in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The horses were subjected to a VN test for EVA during the 28 days prior to export which demonstrated a negative titre;
Or: ii) The horses were subjected to two VN tests for EVA during the 28 days prior to export, on blood samples taken at least 14 days apart which demonstrated a negative, stable or declining titre;
Or: iii) The horses were vaccinated against EVA not more than one year nor less than 21 days prior to importation in accordance with the vaccine manufacturer's recommendations. (N.B. horses from Australia exempt this requirement).

Or:

1. When entire male horses are imported, the horses were kept for the 3 months prior to export in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The horses were subjected to a VN test for EVA during the 28 days prior to export which demonstrated a negative titre;
Or: ii) The horses were vaccinated against EVA under official veterinary control and have been re-vaccinated at regular intervals (at least annually);
(**N.B.** Approved programmes for initial vaccination are as follows:
a. vaccination on the day a blood sample was taken which was subjected to the VN test with a negative result;
b. vaccination during a period of isolation of not more than 15 days, commencing on the day a blood sample was taken which was subjected to the VN test with a negative result;
c. vaccination when the animal was at an age of 180 to 270 days during a period of isolation, during which two blood samples taken at least 10 days apart were subjected to the VN test and demonstrated a negative, stable or declining antibody titre.)
Or: iii) The horses are seropositive to EVA, and were found not to be a semen carrier during the one year prior to export;
(**N.B.** Approved methods for determining semen carriers are as follows:
a. test mating to two mares which were subjected to SN tests with negative results on two blood samples, one collected at the time of test mating and the other 28 days after mating;
b. virus isolation on cell culture carried out on the sperm rich fraction of two separate semen samples with negative results.)

Horse pox

1. The horses were kept for 3 months prior to export in premises where cases of horse pox have not occurred during that period; *and*

2. The horses were showing no clinical sign of horse pox on the day of export.

Japanese encephalitis

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free of JE.

Or:

1. The horses have been resident since birth, or at least the previous 21 days, in a part of Australia where no cases of JE have occurred.

Or:

1. During importation of horses from countries where JE occurs, the horses were vaccinated against JE with an inactivated vaccine according to the manufacturer's recommendations during the 12 months, but not less than 60 days, prior to export; *and*

2. Either: i) The horses were kept for a minimum 21 days prior to export in a pre-export isolation facility and protected from insect vectors during that period and during transport to the port of export;
Or: ii) The date of export and the 21 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses were showing no clinical signs of JE on the day of export.

Rabies

1. The horses were kept since birth or for the 6 months prior to export in an establishment where no case of rabies was reported for at least the 12 months prior to export; *and*

2. The horses were showing no clinical sign of rabies on the day of export.

Borna disease

1. The horses were resident during the 3 months prior to export in an area where no case of BD has occurred during the previous 12 months; *and*

2. The horses were showing no clinical sign of BD on the day of export.

Equine encephalosis

Either:

1. The horses were resident since birth, or at least the previous 28 days, in a country that is free of EE.

Or:

1. EE occurs in the exporting country; *and*

2. Either: i) The horses were kept in a pre-export isolation facility for the 28 days prior to export, and protected from arthropod vectors during this period and during movement to the port of export;
Or: ii) The date of export and the 28 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses were subjected to the indirect ELISA or CFT for EE during 28 days prior to export on two occasions at least 21 days apart, and demonstrated a negative, stable or declining titre; *and*

4. The horses were showing no clinical sign of EE on the day of export.

Hendra and Nipah viruses

Either:

1. The horses were resident since birth, or at least the previous 3 months, in a country that is free of HeV and Nipah virus.

Or:

1. The horses were imported from Australia, where infection of horses with HeV is a notifiable disease; *and*

2. During the 3 months prior to export the horses were kept on premises where infection of horses with HeV has not occurred during that period; *and*

3. The horses were showing no clinical signs of infection with HeV on the day of export.

Or:

1. The horses were imported from Malaysia, where infection of horses with Nipah virus is a notifiable disease; *and*

2. During the 3 months prior to export the horses were kept on premises where infection of horses with Nipah virus has not occurred during that period; *and*

3. During the 30 days prior to export the horses were tested for Nipah virus using either the IgG or IgM capture ELISA or SNT, with negative results; *and*

4. The horses were showing no clinical signs of infection with Nipah virus on the day of export.

Getah virus

Either:

1. The horses were resident since birth, or at least the previous 21 days, in a country in which clinical cases of Getah virus have not occurred during the 12 months prior to export.

Or:

1. The horses were kept for the 21 days prior to export on premises where GV disease has not occurred during that period;

2. Either: i) The horses were kept for a minimum 14 days prior to export in an insect-proof pre-export isolation facility, and were protected from insect vectors during transport from the pre-export isolation facility to the port of export;
Or: ii) The date of export and the 14 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses showed no clinical signs of GV on the day of export.

Anthrax

1. The horses were kept during the 20 days prior to export on premises were anthrax has not occurred during that period; *and*

2. The horses were showing no clinical sign of anthrax on the day of export.

Leptospirosis

1. The horses were kept for the 3 months prior to export on premises where clinical cases of leptospirosis in livestock have not occurred during that period; *and*

2. With the exception of horses imported from Australia, during the 30 day period prior to export:
Either: i) The horses were subjected to the MAT employing antigens from serogroups representative of serovars known to infect horses in the exporting country and *Leptospira* serovars *canicola, grippotyphosa* and *icterohaemorrhagiae*, with negative results (<50% agglutination at the 1:200 titre);
Or: ii) The horses were injected with dihydrostreptomycin or streptomycin (at a dose rate of 25 mg/ kg of live body weight) on two occasions with an interval of not less than 14 days;
Or: iii) The horses were injected with long-acting oxytetracycline (at a dose rate of 20 mg/ kg of live body weight) on two occasions with an interval of not less than 14 days.

Contagious equine metritis (CEM)

Either:

1. The horses have been resident since birth in a country that is free of CEM.

Or:

1. The horse is a gelding.

Or:

1. The horses were kept during the 3 months prior to export on premises where CEM has not occurred during that period; *and*

2. During the 60 day period prior to the export the horses have been tested for CEM by swabbing and culture on three occasions with a negative result for *Taylorella equigenitalis* in each case. The swabs may be taken on days 1, 4 and 7 over a 7 day period, or at 5-7 day intervals. (Horses less than 731 days of age are exempt testing if their dam is available for testing and is negative.) (In countries with approved Codes of Practice for CEM, any testing undertaken in the breeding season prior to export may be used to fulfil export requirements of testing on three occasions. The horse must be certified as not having had sexual contact with horses not of equivalent health status since the time of the first swab considered to be for export purposes.) ; *and*

3. The sites for swabbing are:
i) In stallions, from the prepuce, the urethral sinus, and the fossa glandis (including its diverticulum);
ii) In mares, from the mucosal surfaces of the urethra and the mucosal surfaces of the clitoral sinuses and clitoral fossa, and if the mare is greater than 731 days old, from the mucosal surfaces of the cervix, and the endometrium (on at least one occasion); *and*

4. Since the date of first swabbing for CEM testing, the animal has not been naturally mated except to horses of equivalent health status; *and*

5. In the case of pregnant mares:
Either: i) The stallion and mare were tested for CEM by swabbing on three occasions during the 60 day period prior to mating according to the protocols noted at points 2 and 3 above, and had no sexual contact with any other horses from the time of first swabbing until the time of last service (in the case of stallions, collection of semen for artificial insemination is permitted);
Or: ii) The pregnant mare has been swabbed and cultured prior to export, but the cervical and endometrial swab were not performed. After arrival in New Zealand, the pregnant mare must be held in a registered quarantine facility in New Zealand until the cervical and endometrial swab can be completed subsequent to foaling.

Glanders

Either:

1. The horses were resident since birth, or at least the previous six months, in a country that is free of glanders according to the criteria within Article 3.4.8.2 of the OIE Code.

Or:

1. The horses were kept for the 6 months prior to export on premises where glanders has not occurred during that period; *and*

2. The horses were subjected to the intradermopalpebral mallein test, CFT or dot ELISA for glanders not less than 7 days after entering pre-export isolation, with a negative result; *and*

3. The horses were showing no clinical signs of glanders on the day of export.

Melioidosis

1. The horses were kept during the 3 month period prior to export on premises where melioidosis has not occurred during that period; *and*

2. The horses were showing no clinical signs of melioidosis on the day of export.

Equine salmonellosis

1. The horses were kept during the 3 months prior to export on premises where equine salmonellosis (*S. abortus equi*) has not occurred during that period; *and*

2. The horses were showing no clinical signs of equine salmonellosis on the day of export.

Equine ehrlichiosis

1. The horses were kept during the 3 months prior to export on premises where equine ehrlichiosis (*E. risticii* and *E. equi*) has not occurred during that period; *and*

2. The horses were showing no clinical signs of equine ehrlichiosis on the day of export.

Epizootic lymphangitis

1. The horses were kept for the 3 months prior to export on premises where epizootic lymphangitis has not occurred during that period; *and*

2. The horses were showing no clinical signs of epizootic lymphangitis on the day of export.

Equine piroplasmosis

Either:

1. Prior to export the horses were resident in a country where equine piroplasmosis does not occur, and which does not permit the importation of seropositive horses.

Or:

1. The horses were kept for the 3 months prior to export on premises where equine piroplasmosis has not occurred during that period; *and*

2. The horses have undergone a minimum 10 day period of pre-export isolation. Ticks have been excluded from the isolation facility through prophylactic treatment of all horses upon entry, and absence of ticks has been monitored through regular inspections of isolated horses; *and*

3. Not less than 10 days after entering pre-export isolation the horses have been tested for equine piroplasmosis with a negative result for both *B. equi* and *B. caballi* using the CFT (positive is less than 25% lysis at dilution of 1:5), IFAT or an approved ELISA (with the exception of competition horses temporarily imported under special conditions); *and*

4. The horses were showing no clinical signs of equine piroplasmosis on the day of export.

Dourine

Either:

1. The horses were kept since birth, or for the 6 months prior to export, in a country that has been free from dourine for the past 6 months according to the criteria within Article 3.4.2.2. of the OIE Code.

Or:

1. The horses were kept for the 6 months prior to export on premises where dourine has not occurred during that period; *and*

2. The horses have not been naturally mated with horses not of equivalent health status during the period from 30 days prior to pre-export testing until the time of export; *and*

3. The horses were subjected to the CFT or c-ELISA for dourine with negative results prior to export; *and*

4. The horses were showing no clinical sign of dourine on the day of export.

Surra

Either:

1. The horses were resident since birth, or at least the previous 2 months, in a country that is free of surra.

Or:

1. The horses were kept during the 3 months prior to export on premises where surra has not occurred during that period; *and*

2. The horses have been subjected to a minimum 30 day period of pre-export isolation, and protected from insect vectors during this time and during transport to the port of departure; *and*

3. Not less than 48 hours after entering pre-export isolation the horses have been bled, and a 0.5ml sample of blood inoculated intraperitoneally into two mice per tested blood sample. The mice have been bled three times a week for 28 days and wet blood films examined for the presence of trypanosomes, with negative results; *and*

4. The horses were showing no clinical signs of surra on the day of export; *and*

5. The horses have been subjected to 30 days of post-arrival quarantine in an insect proof facility, during which time they have been bled on two occasions not less than 14 days apart and wet blood films and thick and thin blood smears made and examined for trypanosomes, with negative results.

Screwworm

Either:

1. The horses were resident for the 21 days prior to export in a country or region that has not reported cases of screwworm during the previous year.

Or:

1. The horses were examined, found to be free of screwworm infested wounds, and treated with a prophylactic insecticide during the 48 hours prior to entering pre-export isolation; *and*

2. The horses were subjected to a minimum 7 day period of pre-export isolation, during which time any horses with wounds were monitored for signs of screwworm infestation; *and*

3. The horses were examined, found to be free of screwworm infested wounds, and treated with a prophylactic insecticide during the 48 hours prior to export.

Warble fly

Either:

1. The horses were resident since birth, or at least the previous 3 months, in a country or region which has not reported cases of warble fly during the previous year.

Or:

1. The horses were treated with an ectoparasiticide capable of killing warble fly larvae during the 48 hours prior to export; *and*

2. The horses were showing no clinical signs of infestation on the day of export.

2.2 HORSE SEMEN

African horse sickness

Either:

1. The donor stallions have been resident since birth, or at least the two months prior to collection, in a country that is free of AHS according to the criteria within Article 2.1.11.2 of the OIE Code.

Or:

1. AHS occurs in the exporting country; *and*

2. Either: i) The donor stallions were resident prior to collection in an approved AHS free zone;
Or: ii) The date of collection and the 21 days immediately prior were during approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident; *and*

3. The donor stallions were subjected to indirect ELISA or CFT for AHS during the 28 days after semen collection, on two occasions not less than 21 days apart, which demonstrate a negative, stable or declining titre; *and*

4. The donor stallions were showing no clinical signs of AHS on the day of collection.

Vesicular stomatitis

Either:

1. The donor stallion has been resident since birth, or at least the previous 21 days, in a country that is free of VS according to the criteria within Article 2.1.2.2 of the OIE Code.

Or:

1. The donor stallions were kept on premises during the 21 days prior to semen collection where VS has not occurred during that period; *and*

2. The donor stallions were showing no clinical signs of VS on the day of semen collection.

Venezuelan equine encephalomyelitis

Either:

1. The donor stallions have been resident since birth, or at least the previous 21 days, in a country that is free from VEE according to the criteria within Article 3.4.12.2 of the OIE Code.

Or:

1. When importing from countries where VEE occurs, the donor stallions were subjected to the CF, HI, PRN or IgM capture ELISA for VEE during the 28 days after semen collection, on two occasions not less than 21 days apart. The results must indicate the donor stallion had a negative, stable or declining antibody titre; *and*

2. The donor stallions were showing no clinical signs of VEE on the day of collection and for the subsequent 21 days.

Equine infectious anaemia

1. EIA is a notifiable disease in the exporting country; *and*

2. The donor stallions were kept on premises during the 3 months prior to collection where EIA has not occurred during that period; *and*

3. The donor stallions were subjected to either the AGID or c-ELISA test for EIA not less than 21 days after entry onto the semen collection centre with a negative result; *and*

4. The donor stallions were showing no clinical signs of EIA on the day of collection.

Equine viral arteritis

1. The donor stallions were kept for the 3 months prior to collection in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The donor stallions were subjected to a VN test for EVA not less than 21 days after entering the semen collection centre, with negative results;
Or: ii) The donor stallions were vaccinated against EVA under official veterinary control and have been re-vaccinated at regular intervals (at least annually);
(**N.B.** Approved programmes for initial vaccination are as follows:
a. vaccination on the day a blood sample was taken which was subjected to the VN test with a negative result;
b. vaccination during a period of isolation of not more than 15 days, commencing on the day a blood sample was taken which was subjected to the VN test with a negative result;
c. vaccination when the animal was at an age of 180 to 270 days during a period of isolation, during which two blood samples taken at least 10 days apart were subjected to the VN test and demonstrated a negative, stable or declining antibody titre.)
Or: iii) The donor stallions are seropositive to EVA, and were found not to be a semen carrier during the one year prior to collection;
(**N.B.** Approved methods for determining semen carriers are as follows:
a. test mating to two mares which were subjected to SN tests with negative results on two blood samples, one collected at the time of test mating and the other 28 days after mating;
b. virus isolation on cell culture carried out on the sperm rich fraction of two separate semen samples with negative results.)

Leptospirosis

1. An effective combination of antibiotics, in particular against leptospires and mycoplasmas, must be added to the semen after final dilution. This combination must produce an effect at least equivalent to the following dilutions:
not less than: 500 IU per ml streptomycin,
500 IU per ml penicillin,
150 mg per ml lincomycin,
300 mg per ml spectinomycin.
Immediately after the addition of the antibiotics the diluted semen must be kept at a temperature of at least 15 °C for a period of not less than 45 minutes.

Contagious equine metritis

Either:

1. The donor stallions have been resident since birth in a country that is free of CEM.

Or:

1. The donor stallions were kept for the 3 months prior to collection on premises where CEM has not occurred during that period; *and*

2. During the breeding season in which the semen for export is collected the donor stallion has been tested for CEM by swabbing and culture on two occasions with a negative result for *Taylorella equigenitalis* in each case. The swabs must be taken at a 5-7 day interval; *and*

3. The sites for swabbing are from the prepuce, the urethral sinus, and the fossa glandis (including its diverticulum); *and*

4. If testing occurs prior to collection of semen, since the time the first swab was taken until the time of collection the donor stallion has been permitted to naturally serve only mares of equivalent health status.

Glanders

Either:

1. The donor stallions were resident since birth, or at least the previous six months, in a country that is free of glanders according to the criteria within Article 3.4.8.2 of the OIE Code.

Or:

1. The donor stallions were kept for the 6 months prior to collection on premises where glanders has not occurred during that period; *and*
2. The donor stallions were subjected to the intradermopalpebral mallein test, CFT or dot

ELISA not less than 21 days after entering the semen collection centre, with negative results; *and*

3. The donor stallions were showing no clinical signs of glanders on the day of collection.

Equine salmonellosis (*S. abortus equi*)

1. The donor stallions were kept for the 3 months prior to collection on premises where equine salmonellosis (*S. abortus equi*) has not occurred during that period; *and*

2. The donor stallions were showing no clinical signs of equine salmonellosis on the day of collection.

Dourine

Either:

1. The donor stallions were kept since birth, or for the 6 months prior to collection, in a country that has been free from dourine for the past 6 months according to the criteria within Article 3.4.2.2 of the OIE Code.

Or:

1. The donor stallions were kept for the 6 months prior to collection on premises where dourine has not occurred during that period; *and*

2. The donor stallions were subjected to the CFT or c-ELISA for dourine with negative results:
Either: i) prior to collection, and from the period 30 days prior to testing until collection has not naturally mated any mares not of the same health status;
Or: ii) not less than 30 days following collection of semen for export; *and*

3. The donor stallions were showing no clinical sign of dourine on the day of collection; *and*

4. Semen for export has been examined microscopically prior to freezing and no trypanosomes were detected.

3 INTRODUCTION

3.1 Risk analysis objectives

The objectives of this risk analysis are to:

- identify the infectious disease hazards posed by importation into New Zealand of live horses and horse semen;

- assess the likelihood of these diseases being introduced and establishing here as a result of imports;

- assess the potential for adverse consequences to result from disease introduction and/or establishment; and

- recommend appropriate measures to manage the risk.

The development of this risk analysis has included the release of a draft version for a period of public consultation. (1) Submissions were received from stakeholders of the livestock industries of New Zealand and from trading partners and have been reviewed prior to finalizing on the risk analysis.

The risk management measures recommended in this risk analysis will be included into MAF import health standards for the importation into New Zealand of live horses and horse semen. The import health standards will be subjected to consultation prior to implementation.

3.2 International trade in horses and horse semen

Dr Peter Timoney, speaking at the Eighth International Conference on Equine Infectious Diseases, Dubai, 1998, noted that there will always be an element of conflict between international trade and disease control.(2) Timoney noted that absolute freedom from the risk of disease introduction is unattainable, except through stopping all trade in risk materials.

Horses are moved internationally for competition, breeding, slaughter and as companion animals. There has been an increase in international movement associated with the use of air transport, the increased economic significance of the equine industries, and increased interest in leisure industries. With this increased international movement has come a corresponding increase in the risk of introducing infectious disease.

Timoney cited African horse sickness, contagious equine metritis, equine infectious anaemia, equine viral arteritis, equine piroplasmosis, equine influenza and Venezuelan equine encephalomyelitis as examples of equine diseases that have extended their respective distributions in the last 20 to 30 years as a direct result of international horse movements. Various disease factors may also influence spread, such as the emergence of new pathogens, the mutation of existing pathogens, climate changes and the corresponding effect on disease vector distribution, the establishment of infection in

new vectors, and contamination of vaccines or other biologicals. Timoney emphasised that facilitating the safe international movement (i.e. movement without disease transfer) required regulatory policies to be based on scientific facts, national import control policies to be harmonised, and rapid, sensitive, specific and standardised diagnostic tests to be available to detect the disease agent carrier state in horses.

3.3 Imports into New Zealand

Current MAF import health standards allow horses to be imported from Australia, Canada, Ireland, the United Kingdom and the USA. Data for annual imports of horses in the last 6 years appear as Table 1. Import health standards for horse semen currently allow imports from Australia, Canada, France, Germany, Ireland, United Kingdom and USA.

The current import health standards were developed in response to the requirements of industry stakeholders as conveyed to MAF. Many were developed in the 1980s and early 1990s. Since then, there have been significant advances in understanding the epidemiological features of some diseases, and new diseases with international significance for trade have emerged.

Table 1. Horses imported into New Zealand in the period 1993 to 1999 (Data: Statistics New Zealand.) *

	AUS	UK	USA	IRELAND	CANADA
1993	**494** [§]	**3**	**29**	**1**	**-**
1994	**544**	**10**	**9**	**-**	**7**
1995	**709**	**29**	**27**	**-**	**11**
1996	**472**	**24**	**53**	**-**	**-**
1997	**408**	**55**	**23**	**-**	**3**
1998	**410**	**17**	**39**	**1**	**-**
1999	**462**	**21**	**15**	**-**	**3**

***** Since 1996, the data do not include New Zealand-origin horses returning after short duration visits to other countries, such as for competition purposes. In 1998, Statistics New Zealand recorded 263 imported horses with country of origin noted as New Zealand; in 1999 there were 320 such horses. MAF suggests that the vast majority of these will have been racehorses temporarily visiting Australia.

[§] Year from 1 March 1993

3.4 Context of the risk analysis

The Biosecurity Act 1993, at section 22, requires import health standards to be developed using risk analysis processes involving consultation with stakeholders in New Zealand. The risk analysis must consider the likelihood of imported goods introducing organisms, and the possible effects of introduced organisms on people, the environment and the economy of New Zealand.

MAF has developed an unwanted organisms policy and lists of unwanted organisms in consultation with stakeholders.[3] There are numerous unwanted organisms that are pathogens of horses.

New Zealand is a member of the World Trade Organisation (WTO) and as such has obligations under the *Agreement on the Application of Sanitary and Phyto-sanitary Measures* (the SPS Agreement). These obligations include ensuring that zoosanitary measures are aligned with the recommendations of the Office International des Epizooties (OIE) unless a scientifically-based risk analysis determines that an appropriate level of protection can only be achieved by other measures. The OIE's recommended measures for considering and managing disease risks during trade in animals and animal products appear in the *International Animal Health Code* (the OIE Code). [4]

3.5 Risk analysis methodology

The methodology used for this risk analysis is consistent with the process recommended by the OIE. [4]

The commodities considered in this risk analysis are defined in Section 4.

The diseases of concern to New Zealand during importation of live horses and horse semen are identified in Section 5. A list of those infectious diseases affecting horses that are considered to constitute a potential threat during international trade has been developed from a variety of sources. A filtering process then defines the diseases of concern to New Zealand.

Imports of live horses may introduce metazoan endoparasites and ectoparasites. However, the risk of such an event may be effectively managed through prophylactic treatment protocols applied to all live horses prior to export. These measures are discussed within the hazard identification.

The diseases of concern are considered individually in Section 6. Relevant information is used to assess the risk. Each disease is discussed under the following headings:

1 Aetiology

Describes the causative organism.

2 Susceptibility

Notes the animal species that are susceptible to disease and/or infection. Susceptibility of various age groups and sex groups of the equine population may also be noted.

3 Distribution

Lists the countries where the disease is known to occur and the health status of horses in New Zealand. Data on prevalence or incidence of disease are also presented when available.

4 Clinical signs

Notes the clinical signs typically associated with infection and disease in horses.

5 Transmission

Discusses the mechanisms by which the causative organism is transmitted, including any involvement of vectors.

6 Diagnosis

Notes the methods that may be employed to diagnose the disease and/or infection, with particular emphasis paid to ante-mortem methods prescribed by the OIE.

7 Immunity

Notes whether immunity is achieved following natural infection or vaccination.

8 Treatment

Notes any prophylactic and/or therapeutic treatments for the disease.

9 Risk assessment

Considers the risk of disease introduction and establishment under the following sub-headings:

9.1 Release assessment

Discusses the factors influencing the likelihood that importation of horses or semen will present a pathway for the disease agent to enter New Zealand.

9.2 Exposure assessment

Discusses the likelihood that organisms that might be introduced in horses or semen would spread and establish here.

9.3 Consequence assessment

Discusses the impact of disease introduction and/or establishment in New Zealand.

9.4 Risk estimate

Integrates the conclusions of the previous three sections to deduce whether risk management measures are warranted.

10 Risk management

Considers measures that could be applied to manage the risk of disease introduction under the following sub-headings:

10.1 Risk management objective

States the broad objective of risk management, and discusses the measures that are most likely to achieve it.

10.2 Risk management measures

Notes whether a standard is prescribed in the OIE Code,[4] whether this standard satisfactorily achieves the risk management objectives as determined by the analysis, and whether other factors, in particular country factors that have not been considered within the generic format of the risk analysis, should modify the recommendations.

Finally, recommendations are made for measures to manage the risks of individual diseases when horses and semen are imported.

Throughout, post-arrival quarantine (PAQ) is discussed as a risk management option for live horses if the risk estimate warrants such a measure. The MAF standards for PAQ are discussed and the individual disease recommendations summarised in Section 7.

Horses imported on a temporary basis to participate in competitions (competition horses) may present a lower risk of introducing certain diseases than horses imported permanently. In particular, the risks of diseases spread by sexual contact may differ or may be able to be managed differently. The Biosecurity Act 1993 describes particular legal considerations for managing import animals that have not met all the requirements for biosecurity clearance. The disease measures that could be modified for competition horses, and the legal considerations of thereby not issuing such horses biosecurity clearance, are discussed in Section 8.

Veterinary escort of imported horses has been required for all imports from countries other than

Australia. The function and on-going need for veterinary escort, particularly in light of new recommendations for post-arrival quarantine, is discussed in Section 9.
Collection, processing and storage of semen for import must be conducted in a manner that ensures the health status of imported semen reflects the certified health status of the donor stallion at the time of collection. Specific measures for horse semen are discussed in Section 10.

3.6 Evaluation of Veterinary Services

The OIE Code [4] notes that every Member Country shall recognise the right of another Member Country to undertake, or request it to undertake, an evaluation of its Veterinary Services where reasons exist concerning trade in animals and animal genetic material between the two countries.

Assessment of the government veterinary authorities of potential exporting countries is not included in this risk analysis. Importation from non-traditional trading partners will probably require an assessment of the government veterinary authority prior to development of import health standards. In this regard, MAF may utilise self-assessments that provide the information noted in the OIE Code, or assessments completed by New Zealand's quadrilateral partners (Australia, Canada and the USA) or the European Union.

References:

1 MAF Regulatory Authority. Risk analysis for live equines and equine semen (Draft for consultation). Biosecurity 3: 6. 1 May 1998.

2 Timoney P J. Equids and equine germplasm: international trade versus disease control. Equine Infectious Diseases VIII. Proceedings of the Eighth International Conference on Equine Infectious Diseases, Dubai, 23-26 March 1998. R&W Publications (Newmarket) Ltd. 328-331. 1999.

3 MAF Regulatory Authority. MAF policy statement on unwanted organisms for the purposes of the Biosecurity Act 1993. Unwanted Organism. Biosecurity 6: 5-8. 15 September 1998.

4 OIE. International Animal Health Code: mammals, birds and bees. Office International des Epizooties, Paris. 1999.

4 THE COMMODITIES

4.1 Equine species

The species considered are primarily horses (*Equus caballus*) and donkeys (*Equus asinus*).

MAF expects interest for imports of other species in the genus *Equus*, such as zoo equids. Several factors may influence whether the risk assessment and risk management recommendations for horses can be extrapolated to other species. Where significant species differences in clinical or epidemiological features of disease have been found they are noted in this analysis. The exposure risk for animals entering zoos may be lower than for horses receiving biosecurity clearance, depending on the means of transmission. There may be differences in the ability to apply and interpret diagnostic procedures in species other than horses. For these reasons, further investigation may be required prior to formulating import health standards for other species.

4.2 Live horses

In the current context, "live horses" means any live domesticated animal which is a member of the species *Equus caballus* or *Equus asinus*, or the cross of these two species.

This group may also be called *horses for breeding purposes* or *horses for permanent importation*. Using the terminology of the Biosecurity Act 1993, the group can be further described as *horses eligible for biosecurity clearance*. To be eligible for such clearance the Biosecurity Act 1993 requires MAF to be satisfied that the animal does not harbour potentially harmful organisms. Once a biosecurity clearance has been issued, imported horses cannot be subject to further risk management measures (unless there are grounds to suspect infection with an exotic organism) and are considered resident in New Zealand.

Importation of horses for slaughter is considered unlikely in the New Zealand context, and has not been considered.

4.3 Horse semen

In the context of this analysis, "horse semen" means male germplasm of domesticated members of the species horses, *Equus caballus*, or donkeys, *Equus asinus*.

4.4 Competition horses

Competition horses covers those imported on a temporary basis for the purpose of competing or participating in an organised event. Importation of horses for racing or equestrian events is probably the most likely reason for competition horses to be imported, although the commodity definition would also include horses imported by circus or theatre companies.

Specific disease risks associated with competition horses may be managed through post-arrival restrictions on contact with domestically-resident horses. These post-arrival restrictions may allow a

reduction in pre-export risk management. Under such circumstances, competition horses would not be eligible for biosecurity clearance under the Biosecurity Act 1993. The Act describes specific legal considerations for managing uncleared goods (i.e. any imported good that has not received biosecurity clearance): uncleared goods must remain within MAF-regisitered transitional facilities. Because of these special considerations, the disease risks associated with competition horses are considered separately in Section 8.

4.5 Pregnant mares

Wherever disease risks associated with pregnant mares are assessed to differ from other horses, or require different risk management, these differences are noted.

Pregnant mares are specifically mentioned here because animal welfare implications of long distance transport suggest specific guidelines are appropriate. The International Air Transport Association (IATA) Live Animal Regulations [(1)] recommend that pregnant mares not be transported more than 300 days following the last service (gestation in the mare is approximately 330 days). The MAF *Code of Recommendations and Minimum Standards for the Welfare of Animals Transported Within New Zealand* [(2)] notes that careful consideration of consequences should be made prior to transporting animals in the last third of pregnancy.

With the exception of imports from the east coast of Australia and certain Pacific Island countries, imports of live horses will involve greater than 8 hours air travel. This will create a potential stressful environment. In such cases, eligibility for import should follow the more conservative MAF welfare recommendation and be confined to mares not in the last third of pregnancy. Importation of pregnant mares from eastern Australia should follow the IATA guidelines and not be made more than 300 days after the last service.

References

1 International Air Transport Association. Live Animal Regulations. IATA, Montreal-Geneva. 24th Edition, 1997.

2 MAF Animal Welfare Advisory Committee. Code of Recommendations and Minimum Standards for the Welfare of Animals Transported Within New Zealand. Code of Animal Welfare No. 15. November 1994, amended May 1996.

5 HAZARD IDENTIFICATION

Hazard identification is the process of identifying the pathogenic agents that could potentially be introduced in the commodity considered for importation. The process has been summarised in Table 2 (page 34).

5.1 Potential diseases of concern

The first step in the hazard identification for imports of live horses and horse semen is to develop a list of important pathogens associated with horses.

The left hand column of Table 2 lists pathogens associated with horses, compiled from the following sources:

- OIE List A and B; [1]
- FAO List C diseases to which horses are susceptible; [2]
- current MAF import health standards for live horses and horse semen; and
- veterinary texts. [3, 4, 5, 6]

5.2 Equine diseases exotic to New Zealand

The health status of animals in New Zealand and the methods of control, where appropriate, are noted in columns three and four of Table 2. The code used in these columns is that used by the OIE.

Diseases which have never been reported in New Zealand (as indicated by 0000), which have been eradicated (as indicated by the year of last occurrence), or which were not reported in the current year but for which date of last occurrence is unknown (as indicated by -) are considered exotic, and therefore of concern in this analysis.

5.3 Equine diseases endemic to New Zealand

If an identified pathogen is already present in a country, import measures should not be more trade restrictive than those applied for the domestic movement of horses or semen. The exception to this situation would be if the pathogen was very rare, confined to certain areas, or be a less pathogenic strain than present elsewhere.

5.3.1 Equine viral abortion (EHV-1)

Equine viral abortion occurs sporadically in New Zealand. A recent serological survey concluded that probably all New Zealand thoroughbred studs have mares latently infected with EHV-1.[7] However, a strain of EHV-1 causing abortions in other countries, designated EHV-1B, has not been recorded here. Further, neurological disease caused by EHV-1 occurs very rarely here. Abortive and neuropathogenic strains of EHV-1 should be considered.

5.3.2 Equine herpesvirus 2 (EHV-2)

Surveys suggest EHV-2 infection is common and the virus might play a role in respiratory disease here. [8]

5.3.3 Equine coital exanthema (EHV-3)

Equine coital exanthema occurs New Zealand-wide, although uncommonly. [9]

5.3.4 Equine viral rhinopneumonitis (EHV-4)

EHV-4 occurs widely in horses throughout the country. [8]

5.3.5 Equine herpesvirus 5 (EHV-5)

EHV-5 has been isolated from foals and yearlings in New Zealand. [8, 10]

5.3.6 Equine viral arteritis virus (EVA)

Clinical disease associated with EVA has never been recorded here, although the causative virus is known to be present. EVA is notifiable and an industry control programme restricts movement of carrier stallions, their semen and mares bred to them. [11] EVA should be considered.

5.3.7 Equine adenovirus (EAdV)

Two types of EAdV are recognised, designated EAdV1 and EAdV2. EAdV1 is recognised as a cause of respiratory disease. EAdV2 has been isolated from foals with diarrhoea.

Serology indicates that EAdV is common in New Zealand although not strongly associated with respiratory disease. [8] The survey used a type specific haemagglutination inhibition test employing EAdV1 antigen (pers. comm. Joanne Meers, Massey University, 4 June 1999).

Serological surveillance in Australia has demonstrated a high rate of exposure to both EAdV1 (86% positive, n=339) and EAdV2 (77% positive, n=339). [12] Considering no safeguards against EAdV have ever been included in import health standards for horses imported from Australia, both types are likely to be present here.

5.3.8 Equine coronavirus

Particles with typical coronavirus morphology have been identified by electron microscopy in faeces of foals with profuse watery diarrhoea and fever in this country. [13, 14]

5.3.9 Equine torovirus

There is no information on equine torovirus presence in this country. In Switzerland, it was isolated from a rectal swab of a horse with diarrhoea; whether it was the cause of disease could not be proven. The high percentage of seropositive animals in horses populations of different countries suggests a worldwide distribution and a high prevalence of infection in adult horses. [15]

No safeguards against equine torovirus have ever been included in import health standards for horses imported into New Zealand, suggesting it is likely to be present here.

5.3.10 Equine rotavirus

Rotavirus infections are a common cause of diarrhoea in foals here. [9]

5.3.11 Equine rhinovirus

A survey of horses in this country found evidence of both equine rhinovirus type 1 and type 2 infections. [16]

5.3.12 Strangles (*Streptococcus equi*)

Strangles occurs throughout New Zealand and is controlled by vaccination. [9]

5.3.13 Ulcerative lymphangitis (*Corynebacterium pseudotuberculosis*)

Although ulcerative lymphangitis has not been recorded in horses here, caseous lymphadenitis (*Corynebacterium ovis*, *C. pseudotuberculosis*) is relatively common in sheep. *C. equi* infections also occur here, though rarely. [9]

5.3.14 Leptospirosis

The serovars isolated from animals in this country are serovar *hardjobovis*, serovar *pomona* (including subtype *kennewicki*), serovar *copenhageni*, serovar *ballum*, serovar *tarassovi* and serovar *balcanica*. [17] Leptospirosis is a cause of equine abortions here. [18, 19, 20] Serology suggests that serovar *bratislava* is present. [18, 21]

The serovars *canicola, grippotyphosa* and *icterohaemorrhagiae* have not been recorded in New Zealand. With the current exception of horses, all MAF import health standards for livestock and semen contain safeguards against exotic serovars of *Leptospira*. The risks associated with imports of horses and semen should be considered.

5.3.15 Dermatophilosis (*Dermatophilus congolensis*)

Dermatophilosis is common in livestock in New Zealand. [9]

5.4 Endoparasites and ectoparasites

Imports of live animals present a particular risk of introducing endoparasites and ectoparasites, and this risk is typically managed through treatment prior to export. Veterinary examination prior to export may also provide some assurance of freedom from ectoparasites.
The efficacy of a parasiticide treatment may vary according to local conditions, including the availability of drugs and treatment methods, the presence of drug resistance and climatic conditions. It is difficult to prescribe a treatment regime that will be 100% effective against all endoparasites and ectoparasites in all circumstances. To a certain degree, freedom from parasites must rely upon the experience of the veterinarians supervising pre-export preparation; they will have a knowledge of what is required to be able to confidently make a declaration of freedom from parasites, in so far as is possible.

However, resistance of equine nematodes to both benzimidazole and pyrantel anthelmintics has been recorded in a number of countries. In New Zealand resistance to benzimadazole drenches has been recorded and is probably widespread. However, resistance to the macrocyclic lactone group of anthelmintics has not been reported anywhere. Further introductions of anthelmintic resistant endoparasites should be avoided, although the possibility of introduction cannot be totally eliminated. The most practical way to avoid introductions of resistant endoparasites is to specify treatment with a macrocyclic lactone (pers. comm. Phil McKenna, AgriQuality NZ Ltd, 9 March 1999).

Treatments effective against parasites should be required twice during the pre-export period, upon entry into isolation and prior to export, as follows:

1 During pre-export isolation the horses have been treated on two occasions, within 48 hours of entering the facility and export, in the following manner:

for ectoparasites, using the following compounds with efficacy against flies, ticks, lice and mites, according to the manufacturer's recommendations:

Ectoparasiticide and dose rate:.....

for endoparasites, using a macrocyclic lactone compound according to the manufacturer's recommendations:

Endoparasiticide and dose rate:.....

2 The animals were examined within 48 hours of export and were found to be free of evidence of infectious or contagious disease including ectoparasites.

When horses are required to undergo post-arrival quarantine they should receive a single repeat treatment.

In the case of imports from Australia, introduction of ticks must be considered when importing horses from areas where species commonly infecting livestock, in particular *Boophilus microplus*,

are endemic. Such areas are well described in Australia. A single pre-export ectoparasite treatment should be applied to all horses from Australia within 48 hours of export. When horses are imported from the tick areas of Australia, in particular Queensland, they will be held for a minimum of 3 days in AQIS approved stables and be treated for ectoparasites at the time of entry. AQIS will approve stables under the following guidelines (pers. comm. David Wilson, AQIS, with Barry O'Neil, 12 November 1997):

- Horses for export must be fully stabled at all times. Horses outside the stable complex (unstabled) are not eligible for export.
- No livestock may be held within 100 metres of the stable complex. Fencing must be stock proof and permanent.
- Horses in the stable complex are to be tick free, manageable and frequently groomed.
- Horses are treated with an approved acaricide prior to entry into the stables (note that this treatment is additional to the treatment at 48 hours prior to export).
- Records must be maintained of all horses entering and leaving the stables.

5.5 Arthropod disease vectors

The risk associated with diseases transmitted by arthropod vectors depends partly on the presence, distribution and potential vector-competence of arthropods in New Zealand.

5.5.1 Mosquitoes

Prior to 1999 there were fifteen species of mosquitoes recognised as being established in New Zealand: twelve endemic and three introduced. The endemic mosquitoes are *Aedes chathamicus, Ae. antipodeus, Coquillettida tenuipalpis, Cq. iracunda, Culex asteliae, Cx. rotoruae, Cx. pervigilans, Culiseta novaezealandiae, Cs. tonnoiri, Maorigoeldia aegyropus* and *Opifex fuscus*. Each has particular habitat requirements that affect their distribution here, but little is known about their vector-competence (pers. comm. Trevor Crosby, Landcare Research New Zealand Ltd, with Howard Pharo, 16 October 1997).

The introduced species are *Ae. notoscriptus, Ae. australis* and *Cx. quinquefaciatus*. *Ae. notoscriptus* occurs in the North Island and in the Nelson area. *Ae. australis* occurs in the southern South Island and Stewart Island. *Cx. quinquefaciatus* occurs in the Auckland area (pers. comm. Trevor Crosby, with Howard Pharo, 16 October 1997).

In January 1999 two further introduced species, *Ae. camptorhynchus* and *Cx. australicus*, were detected in salt-marshes in the Napier area. [(22)]

Ae. notoscriptus, Ae. australis, Ae. camptorhynchus and *Culex quinquefaciatus* are involved in arbovirus transmission within their respective endemic ranges, although their potential significance as vectors of disease under New Zealand conditions is uncertain.

The possibility of other exotic species of mosquito establishing here is also of concern. *Ae. albopictus*, the Asian tiger mosquito, has been detected in used tyre imports. *Ae. aegypti* and

Culex annulirostris have also been intercepted, but are not known to have established here. Both these species have proven arbovirus vector-competence in their respective endemic ranges. [23]

5.5.2 Ticks

Although nine species of ticks are known to have established here, only *Haemaphysalis longicornis*, the cattle tick, commonly affects livestock. Several *Ixodes* spp. ticks and *Ornithodoros capensis* occur on birds. [24]

H. longicornis is probably the vector of *Theileria orientalis* in cattle in New Zealand. Four of the tick species present, including *H. longicornis,* are capable of virus transmission. Since ticks are not very host specific it is possible, although unlikely, that any of the ticks established here could be acquired by humans or domestic animals. [24]

5.5.3 Biting flies

Culicoides spp. midges do not occur in New Zealand. Neither do livestock-biting species of Tabanid flies occur here.

Stomoxys calcitrans, the horse stable fly, does occur in association with horses and cattle in this country. The favoured breeding sites are composting organic material. [25] *S. calcitrans* is capable of transmitting equine infectious anaemia virus and surra (*Trypanosoma evansi*).

Several species of blackfly (or sandfly) are also present but have not been evaluated as potential vectors of disease.

Table 2. Hazard identification: import health risk analysis for horses and horse semen.

Potential diseases of concern	OIE or FAO lists	NZ status	NZ control	Exotic strains	Unwanted	Of concern?
Viruses						
African horse sickness	A110	0000	Qf*		Y	Y
vesicular stomatitis	A020	0000	Qf*		Y	Y
Venezuelan equine encephalomyelitis	B216	0000	Qf*		Y	Y
eastern and western equine encephalomyelitis	B204	0000	Qf*		Y	Y
equine infectious anaemia	B205	0000	Qf*		Y	Y
equine influenza	B206	0000	Qf*		Y	Y
equine viral abortion (EHV-1)	B208	+	V	Y?		Y
equine viral rhinopneumonitis (EHV-4)	B208	+	V	N		N
equine viral arteritis	B211	+?	Qf Qi V*	Y	Y	Y
horse pox	B210	0000	Qf*		Y	Y
Japanese encephalitis	B212	0000	Qf		Y	Y
rabies	B058	0000	Qf*		Y	Y
Borna disease		-	Qf		Y	Y
equine encephalosis		-	Qf		Y	Y
louping ill		-	Qf		Y	Y
Hendra virus		-	Qf		Y	Y
Nipah virus		-	Qf			Y
Getah virus group		-				Y
equine herpesvirus 2 (EHV-2)		+		N		N
equine coital exanthema (EHV-3)	C751	+		N		N
equine herpesvirus 5 (EHV-5)		+		N		N
equine adenovirus		+		N?		N
equine coronavirus		+		N?	Y	N
equine torovirus		...				N
equine rotavirus		+		N?		N
equine rhinovirus		+		N?		N

Disease name/agent	OIE or FAO lists	NZ status	NZ control	Exotic strains	Unwanted	Of concern
Bacteria						
anthrax	B051	1954	Qf*		Y	Y
contagious equine metritis	B201	0000	Qf*		Y	Y
glanders	B209	0000	Qf*		Y	Y
leptospirosis	B056	++	Qf V*	Y	Y	Y
bovine brucellosis (*B. abortus*)	B102	1989	Qf*		Y	Y
melioidosis	C613	-	*		Y	Y
salmonellosis (*Salmonella abortus equi*)	C754	-			Y	Y
lyme disease		-	Qf		Y	Y
Q fever	B057	0000	Qf*		Y	Y
equine ehrlichiosis		-	Qf		Y	Y
strangles (*Streptococcus equi*)	C753	+	V	N		N
ulcerative lymphangitis (*Corynebacterium pseudotuberculosis*)	C752	+?		N?		N
Fungi						
epizootic lymphangitis	B201	0000	Qf*		Y	Y
Dermatophilus congolensis		+		N		N
Protozoa						
equine piroplasmosis	B207	0000	Qf*		Y	Y
dourine	B202	0000	Qf*		Y	Y
surra	B215	0000	Qf*		Y	Y
equine protozoal myeloencephalitis (*Sarcocystis neurona*)		-				Y
Metazoa						
screwworm	B060	0000	Qf*		Y	Y
warble fly myiasis	C654	0000	Qf		Y	Y

Legend for disease occurrence reports

0000	Disease never reported
-	Disease not reported (date of last outbreak not known)
(month/year)	date of last reported occurrence of the disease in previous years
?	Disease suspected but presence not confirmed
+	Reported present or known to be present
+?	Serological evidence and/or isolation of the causal agent, but no clinical signs of disease
( )	Disease limited to specific zones
...	No information available

Legend for disease control reports

*	Notifiable disease
Cn	Control of arthropods
Cr	Control of wildlife reservoirs
M	Monitoring
Qf	Precautions at the border
Qi	Movement control inside the country
S	Stamping out
Sp	Stamping out (Modified ~)
Su	Surveillance
Te	Screening
V	Vaccination
Vp	Vaccination prohibited
Z	Zoning

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5.6 DISEASES OF CONCERN

The risks associated with the following diseases should be assessed in relation to the importation of live horses and horse semen into New Zealand:

Viral diseases:

African horse sickness
vesicular stomatitis
Venezuelan equine encephalomyelitis
eastern and western equine encephalomyelitis
equine infectious anaemia
equine influenza
equine viral abortion (EHV-1)
equine viral arteritis
horse pox
Japanese encephalitis
rabies
Borna disease
equine encephalosis
louping ill
Hendra and Nipah viruses
Getah and Ross River viruses

Bacterial diseases:

anthrax
contagious equine metritis
glanders
leptospirosis
bovine brucellosis (*B. abortus*)
melioidosis
salmonellosis (*Salmonella abortus equi*)
lyme disease
Q fever
equine ehrlichiosis

Fungal diseases:

epizootic lymphangitis

Protozoan diseases:

equine piroplasmosis
dourine
surra
equine protozoal myeloencephalitis (*Sarcocystis neurona*)

Metazoan diseases:

screwworm
warble fly myiasis

6 RISK ASSESSMENT AND RISK MANAGEMENT

6.1 VIRAL DISEASES

1 African Horse Sickness

1.1 Aetiology

African horse sickness (AHS) is a non-contagious vector-borne OIE List A disease of Equidae, caused by an orbivirus in the family Reoviridae.[1, 2] Nine serotypes are known. Serotypes 1-8 are highly pathogenic, whereas serotype 9 is less so. [2]

1.2 Susceptibility

AHS affects all Equidae, with horses being most susceptible (mortality 70-95%) and mules less so (mortality 50-70%). Most infections in donkeys and zebras are subclinical. Zebras are most probably the reservoir host in endemic regions. [2, 3]

Dogs may contract a fatal form of AHS after ingestion of infected horse meat. However, as *Culicoides* spp. do not usually feed on dogs, they probably play no role in the spread of AHS virus. [2]

Antibodies against AHS have not been found in ruminants other than camels. Pigs, cats and some other species are refractory to infection. Humans do not appear susceptible to field strains of the virus, although certain vaccine strains can cause encephalitis and retinitis following accidental transnasal infections. [2]

1.3 Distribution

AHS is endemic in tropical East and West Africa, from where it regularly spreads to southern and occasionally to northern Africa. Outbreaks have occurred outside Africa as a result of international movement of Equidae in the Middle East (1959-1963), in Spain (1966 and 1987-1990), and in Portugal (1989). [1, 2]

AHS has never occurred in New Zealand or North America.

In South Africa AHS occurs every summer in the northern provinces, with the first cases occurring in February. The most abundant serotypes in South Africa in recent years have been serotypes 1, 4 and 7. The disease spreads southwards over the next few months, with the epidemic abruptly halting in late April or early May after the first frosts. Early and heavy rains followed by warm dry spells favour the occurrence of epidemics. [2]

1.4 Clinical signs

The incubation period is 5-7 days in natural infections, but has been recorded as variably as 2-21

days in artificially induced infections. [4]

AHS is characterised by clinical signs and lesions associated with respiratory and circulatory impairment. The acute form is the most common during epidemics. Death is typically within 4-5 days of onset of clinical signs. [1, 2, 4]

The subacute form occurs most commonly in horses in endemic areas. Fever develops slowly and persists longer, and there is oedema of the head. Mortality is not as high as in the acute form. [1, 2, 4]

A form with no clinical signs also occurs in endemic areas, most commonly in donkeys and zebras. [2]

1.5 Transmission

AHS is a non-contagious disease. [1] The virus probably maintains an endemic cycle between *Culicoides* biting midges and a mammalian reservoir host, such as zebras. The most important natural vector is *C. imicola*. Seasonal occurrence is influenced by climatic conditions favouring breeding of *Culicoides* spp. [5, 6]

Other insect species have been investigated in relation to AHS transmission. *Aedes aegypti, Culex pipiens, Anopholes stephensi* and biting flies have experimentally transmitted AHS virus, but none of these have been shown to play a role under natural conditions. The dog tick *Rhipicephalus sanguineus* has also transmitted the virus experimentally. Natural mechanical transmission by arthropods is likely to be rare because the viral titre in horses is relatively low and the virus is relatively susceptible to inactivation outside its hosts. [1, 2, 4]

Following infection of the horse by the *Culicoides* vector, virus replication occurs in regional lymph nodes, followed by a primary viraemia with subsequent infection in lungs and various lymphoid tissues. Virus multiplication at these sites leads to a secondary viraemia. [1]

The virus is present in all body fluids and tissues from the onset of fever until recovery. Viraemia in horses is of variable duration, typically 4-8 days, but it does not exceed 21 days. Viraemia in donkeys may last up to 28 days. [1] During an epidemic infected horses provide the main source of virus. Horses that recover from the disease do not remain carriers of the virus [2], which partially explains the failure of the disease to become established outside tropical Africa. The distribution of *C. imicola* is also a major factor delimiting the spread of AHS.

There are no reports of transmission of AHS via artificial insemination, although virus would be expected to be present in semen of viraemic horses.

1.6 Diagnosis

Definitive diagnosis is by virus isolation from blood collected during the viraemic period or post-mortem samples. Antigen ELISA [7] and PCR assays [7, 8] can be used for virus identification from cell culture isolates or field samples.

Horses that survive natural infection develop antibodies 8-12 days following infection. Antibody detection by indirect ELISA and CFT are the tests prescribed by the OIE for international trade. The VN test employing type-specific antisera may be used to serotype the infecting virus. [7]

1.7 Immunity

There is no carrier state in horses. Survivors develop strong immunity to the particular serotype with which they were infected. While this may confer some cross-protection to infection with other serotypes, a strong challenge may overcome the acquired immunity. [2]

Vaccines are available for all 9 serotypes. [7]

1.8 Treatment

There is no effective treatment.

1.9 Risk assessment

1.9.1 Release assessment

Horses imported from the AHS endemic areas could potentially be infected with AHS virus. In the northern and southern zones of the endemic area infections occur seasonally, and the likelihood of a viraemic horse being imported could effectively be negligible for particular areas at certain times of the year.

No reliable estimate of the prevalence of infection within the endemic areas is possible. Estimates based on clinical cases do not account for the high rate of subclinical infections, particularly in donkeys and zebras. Seroprevalence does not provide a good indication of prevalence relevant to the risk of an infected horse being imported, because of the high rates of vaccination in the endemic areas and the poor correlation between viraemia and serology.

The viraemia is relatively shortlived and will have passed in the majority of horses by the time antibody titres develops. Thus, while a rising antibody titre indicates recent infection, horses with stable or declining titres will probably not be viraemic. The exception is if re-infection occurs, as prior infection does not always confer immunity to heterogeneous serotypes.

Because virus may be present in all body fluids of viraemic horses there is a theoretical risk associated with semen.

1.9.2 Exposure assessment

AHS is non-contagious and natural transmission requires an intermediate host that does not occur in New Zealand. Some uncertainties remain with regard to virus transmission, particularly the reservoir host and the full range of arthropod species capable of acting as vectors and/or mechanical

transmitters. However, New Zealand's temperate climate and arthropod fauna would not favour the establishment of endemic cycles, and the likelihood of AHS establishing here is considered to be remote. Indeed, it is unlikely that any transmission from an imported index case would occur.

Despite the theoretical risk of virus presence in semen, artificial insemination has not been suggested to be an important exposure pathway.

1.9.3 Consequence assessment

Importation of infected horses or semen would be unlikely to lead to further locally acquired cases of AHS. However, a single imported case could have adverse consequences associated with the exotic disease investigation and response, including potential impacts on exports from New Zealand of horses and semen.

New Zealand conducts no routine serological surveillance for AHS, so the presence of seropositive horses would be unlikely to have any significant impact for animal health surveillance.

1.9.4 Risk estimate

Horses incubating AHS or in the viraemic phase may be imported from endemic areas. Animal health surveillance is able to demonstrate seasonal variations in risk for some areas. AHS is very unlikely to establish in New Zealand, but a clinical case in an imported horse could have adverse consequences. In particular the indirect consequences associated with exports of horses and semen from New Zealand could be significant. Measures to ensure infected horses are not imported are warranted.

Semen collected from viraemic stallions may harbour virus, although the likelihood of transmission by artificial insemination is probably very low. Measures to ensure semen is not collected from infected stallions are warranted.

1.10 Risk management

1.10.1 Risk management objective

The primary objective is to avoid importing horses incubating AHS or in the viraemic phase of infection, or semen collected from such horses. All horses should be protected from infection during the pre-export period, and recently infected horses should be ineligible for export.

In particular areas, notably those in the southern part of the endemic range, protection from infection may be achieved by importing during a defined seasonal period during which virus transmission by *Culicoides* vectors has been demonstrated not to occur.

At other times of year, a pre-export isolation period of minimum 28 days duration for horses or 35 days for donkeys in facilities which preclude contact with *C. imicola* should ensure animals are not viraemic at the time of export. Supervised exercise outside the arthropod proof facility following

prophylactic insecticide treatment could be safely allowed outside the *Culicoides* biting period, which occurs from dusk till dawn.

Two serological tests applied twice 21 days apart during the isolation period will detect rising antibody titres, indicative of recent infection. All horses should demonstrate negative, stable or declining antibody titres during the pre-export period. The indirect ELISA or CFT should be considered acceptable serological tests.

A period of post-arrival quarantine of at least 14 days duration is appropriate to account for the potential adverse consequences if an imported case was released. Transmission from an imported horse after arrival in New Zealand is very unlikely, so facilities need not be vector proof. Further serological testing during post-arrival quarantine is not necessary.

1.10.2 Risk management measures

The OIE Code [9] notes that a country may prohibit the importation of horses and semen from countries or zones infected with AHS. However, such a policy is not considered necessary to protect New Zealand from AHS because the absence of recognised vectors and climatic factors make disease establishment here extremely unlikely.

Demonstration of a disease-free region and/or a seasonal period of low risk should require the provision of animal health surveillance information supporting such a case.

Live horses

Either:

1. The horses were resident since birth, or at least the previous two months, in a country that is free of AHS according to the criteria within Article 2.1.11.2 of the OIE Code.

Or:

1. AHS occurs in the exporting country; *and*

2. Either: i) The horses were resident prior to export in an approved AHS free zone;
Or: ii) The date of export and the 28 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export;
Or: iii) The horses were kept in a pre-export isolation facility for the 28 days (35 days in the case of donkeys) prior to export, and protected from vectors during this period and during movement to the port of export. (Protection from vectors shall comprise confinement within arthropod-proof stables at all times, with the exception of officially supervised exercise sessions between the period 2 hours after sunrise and 2 hours prior to sunset. Prior to these sessions the horses shall receive prophylactic treatment with an approved insect repellent.); *and*

3. The horses were subjected to an indirect ELISA or CFT for AHS during the 28 days prior to export on two occasions not less than 21 days apart, and demonstrated a negative, stable or declining antibody titre; *and*

4. The horses were showing no clinical signs of AHS on the day of export; *and*

5. Upon arrival in New Zealand the horses were subjected to a minimum 14 day period of post-arrival quarantine prior to biosecurity clearance.

Horse semen

Either:

1. The donor stallions have been resident since birth, or at least the two months prior to collection, in a country that is free of AHS according to the criteria within Article 2.1.11.2 of the OIE Code.

Or:

1. AHS occurs in the exporting country; *and*

2. Either: i) The donor stallions were resident prior to collection in an approved AHS free zone;
Or: ii) The date of collection and the 28 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident; *and*

3. The donor stallions were subjected to indirect ELISA or CFT for AHS during the 28 days after semen collection, on two occasions not less than 21 days apart, which demonstrate a negative, stable or declining titre; *and*

4. The donor stallions were showing no clinical signs of AHS on the day of collection.

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2 Vesicular stomatitis

2.1 Aetiology

Vesicular stomatitis (VS) is an OIE List A disease caused by a group of viruses within the family Rhabdoviridae, genus Vesiculovirus. Two antigenically distinct viruses are recognised: New Jersey (NJ) and Indiana (I). [1] A number of other serotypes have been reported in South and Central America.[2, 3] Isolates from the USA tend to be more similar to Mexican isolates of the same year than isolates from the USA in different years.[4]

2.2 Susceptibility

VS infects Equidae, cattle, swine, white-tailed deer, and numerous species of small mammals in the tropics. [3] Sheep and goats appear to be refractory to infection. [2]

VS is of regulatory importance because it is clinically indistinguishable from foot and mouth disease (FMD) in FMD susceptible species (horses are resistant to FMD). [1]

VS commonly infects humans in endemic areas causing mild, flu-like symptoms. [3, 5]

High rates of seroprevalence are reported in susceptible species within the endemic areas. [3]
A study during the 1997 outbreak in the USA found 1.3% of susceptible animals on the premises surveyed were seropositive and 31% of these were Equidae. [4]

2.3 Distribution

VS is confined to the Americas, and occurs in endemic and epidemic forms. The disease tends to be endemic in low-lying areas with tropical climates, heavy rainfall and large insect populations. The endemic zone is from Panama south to Venezuela and Peru, with pockets of endemic infection in Mexico and the USA. [3, 5] Endemic VSNJ formerly occurred in the south east USA, but now appears confined to Ossabaw Island, on the south-east coast of Georgia. [6]

Epidemic VS appears to occur as a result of explosive spread northwards to Mexico and the USA and southwards to Brazil and Argentina from the endemic zones. The epidemics are seasonal; they occur in summer and sharply decrease in cold weather (typically the first frost in temperate zones). [2, 3] Epidemics in the USA occur principally in the south-western states and along the Mississippi valley. During outbreaks clinical cases appear to follow natural drainages, but there is some distant spread and large populations of susceptible animals within the infected area may be excluded. Spread does not always appear to be related to animal movements. [5, 6, 7]

VS was recorded in France in 1915 and 1917, and South Africa in 1886 and 1887. [5] The last record of VS in Canada was 1949. [8]

VS has never been recorded in New Zealand.

2.4 Clinical signs

The incubation period in horses is short, typically 1-3 days [2, 3] but possibly up to 21 days. [1]

Morbidity is variable, although often high (up to 90%) within a herd. Mortality in horses is rare. Affected horses exhibit depression and a transient fever. The typical lesions are vesicles, which are very short lasting and rupture within hours. Healing commences within 4 days of rupture and is rapid, with most animals being clinically normal within 3-10 days of onset of signs. [1, 2, 3, 4, 6]

Subclinical infection is common, with only 10-15% of infected horses developing clinical signs. [5]

2.5 Transmission

The epidemiology of VS, and particularly the importance of various modes of transmission, is incompletely understood. [2, 3, 5, 6, 7]

VS virus is present in saliva, vesicular exudate and epithelium of open vesicles. Direct spread occurs within a herd, presumably by virus contact with cutaneous or mucosal abrasions. While arthropod transmission is known to occur, no vertebrate amplifying host species (i.e. one in which virus circulates in sufficient quantities to infect a biting arthropod vector) has been found. *Aedes* spp. mosquitoes, *Lutzomyia* spp. sandflies and *Simulium* spp. blackflies can transmit VS. A wide range of other mosquitoes and biting flies are capable of becoming infected. How arthropods become infected and how they pass on infection to vertebrates is unknown. [2, 3, 5, 6, 7, 9]

It has not been determined whether North American VS epidemics occur following climatic conditions which favour a northward extension of insects' range from endemic areas or whether the virus is maintained within a reservoir host within the USA. [7]

Iatrogenic transmission on hands and equipment may occur during outbreaks. [3]

Although no reports of transmission of VS via artificial insemination have been found, virus has been isolated from horse semen.[10]

2.6 Diagnosis

Definitive diagnosis is by virus isolation on cell cultures, with identification by electron-microscopy, indirect ELISA, CFT or IFT. [3, 5]

The diagnostic techniques prescribed by the OIE for international trade are a liquid-phase blocking ELISA, a competitive ELISA, VN and CFT. The CFT is noted as being of low sensitivity and frequently affected by anti-complementary or non-specific factors. [5]

VN and CF titres rise quickly,[3] and can be detected within 2 weeks of infection. [2] VN titres persist but CFT titres decline to low levels within 2-4 months. [3]

2.7 Immunity

While cattle are susceptible to reinfection 30-60 days after recovery, horses may remain refractory for longer periods. [3]

A variety of vaccines have been developed, but it is not yet clear whether they provide protection from disease. [5]

2.8 Treatment

There is no effective treatment.

2.9 Risk assessment

2.9.1 Release assessment

Live horses imported from the VS endemic and epidemic zones of the Americas could be infected with VS. In the epidemic zones, such as in the USA, there is a seasonal pattern to disease occurrence. The likelihood of an infected horse being imported would increase during summer and autumn, and would be negligible in the winter in many temperate regions.

The prevalence of infection in a herd of horses within the endemic zone may be high. It may also be high during disease outbreaks in the epidemic zones. Subclinical infection is common, and so clinical freedom in individual horses does not provide a good assurance of freedom from infection.

The OIE considers the incubation period to be 21 days for the purposes of international trade. The length of the infectious period for horses is not known, but as there is no viraemia horses can probably be considered to no longer be infectious once all lesions have healed, typically within 10 days of their appearance.

Importation of horse semen probably presents very little risk of VS introduction if the stallion is free from lesions at the time of collection.

2.9.2 Exposure assessment

While outbreaks of VS have occurred in countries outside the Americas, these have apparently been self-limiting and no endemic cycles have established.

New Zealand's arthropod fauna contains species related to those that have been experimentally implicated in VS transmission. However, as there is no viraemia in infected horses, arthropod vectors are unlikely to become infected by ingestion of blood from any imported infected horse. Nevertheless, because the means by which arthropods become infected remains uncertain, the possibility of mechanical transmission by insects must be considered should a horse with VS lesions be imported. The ability of VS to transmit by direct contact also suggests that a restricted outbreak might occur within a herd should an infected animal be introduced. Iatrogenic transmission is a

further possibility.

Despite the possible exposures discussed above, the likelihood of endemic cycles establishing here is considered to be remote. New Zealand's temperate climate and the fact that such cycles do not occur outside of the Americas support such a conclusion.

2.9.3 Consequence assessment

The clinical similarity of VS to FMD could mean that an imported case would lead to suspension of a wide range of commodity exports until the nature of the disease was confirmed. If a diagnosis of VS was confirmed, some trading partners might impose restrictions on agricultural exports, including live animals, genetic material and meat products. The equine, cattle and deer agricultural export industries could be severely affected by trade restrictions, and trade-related consequences could last for up to 2 years until New Zealand once again met the OIE criteria for VS freedom.

The potential consequences of an imported case of VS are therefore likely to be severe, although largely indirect and associated with an adverse impact on agricultural exports.

New Zealand conducts no routine serological surveillance for VS. The presence of VS seropositive horses would have no impact for animal health surveillance.

2.9.4 Risk estimate

Although VS is unlikely to establish endemic cycles in New Zealand, horses infected with VS virus could be imported from endemic areas during periods of virus transmission, and some transmission to other animals in New Zealand could occur. An imported case could lead to a loss of New Zealand's health status as a VS-free country, with the potential for trade consequences for important agricultural export sectors. Measures to ensure infected horses are not imported are warranted. Semen presents a very low risk so long as stallions are free from clinical signs at the time of collection.

2.10 Risk management

2.10.1 Risk management objective

All horses should be protected from infection during the pre-export period, and recently infected horses should be ineligible for export.

Horses that have been resident in a VS free country for a minimum of 21 days prior to export do not present a risk of disease introduction.

Horses from VS infected regions should be isolated for 21 days. The risk of arthropod transmission suggests pre-export isolation premises should preclude contact with arthropod vectors of disease.

Serological testing during isolation will determine if horses have been recently infected. The LP-

ELISA, c-ELISA and VN are acceptable tests. The CFT has low sensitivity and should not be used. If serologically positive horses are detected, all horses should be re-tested not less than 14 days subsequently. All horses should demonstrate negative, stable or declining titres, because seroconversion or rising titres may indicate circulating virus, presenting the possibility of direct transmission between isolated horses.

When horses are imported from VS infected areas, post-arrival quarantine for a minimum 21 days duration will further reduce the likelihood of infected horses being released into New Zealand. Quarantine facilities should be arthropod-proof.

All horses should be clinically free from the signs of VS, and this also applies for stallions from which semen is to be collected for importation. Semen collection centres should have been free from VS during the minimum 21 days prior to collection.

2.10.2 Risk management measures

The OIE Code [(11)] defines a VS free country in Article 2.1.2.2.

The USA is in the epidemic zone of VS. Epidemics occur regularly, but not annually, and follow a seasonal pattern. During these outbreaks and in the intervening periods the United States Department of Agriculture conducts surveillance, monitors spread, and declares and notifies infected areas in order to safeguard agricultural exports. Although there is no international standard for VS free zones, the lower risk posed by importation of horses from areas of the USA where no cases have occurred for two years should be recognised. Such horses should not be subject to the pre-export isolation or post-arrival quarantine recommendations for other VS infected countries.

Live horses

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free of VS according to the criteria in Article 2.1.2.2. of the OIE Code.

Or:

1. When importing from countries where VS occurs but approved surveillance systems are in place to provide rapid detection and on-going monitoring:
Either: i) the horses were resident since birth, or at least the 21 days prior to export, in a part of the territory of the country where VS has not occurred during the previous 2 years;
Or: ii) The date of export and the 21 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident; *and*

2. During the 21 days prior to export the horses were subjected to a LP-ELISA, c-ELISA or VN test for VS. In the case of any positive result, all in-contact horses should be re-tested not less than 14 days subsequently. The results of testing should indicate all horses have negative, stable or declining titres; *and*

3. The horses were showing no clinical signs of VS on the day of export.

Or:

1. When importing from countries where VS occurs, the horses were kept in a pre-export isolation facility for the 21 days prior to export, and protected from vectors during this period and during movement to the port of export; *and*

2. The horses were subjected to a LP-ELISA, c-ELISA or VN test for VS during the 21 days prior to export. In the case of any positive result, all horses should be re-tested not less than 14 days subsequently. The results of testing should indicate all horses have negative, stable or declining titres; *and*

3. The horses were showing no clinical signs of VS during the 21 day period prior to export and on the day of export; *and*

4. Upon arrival in New Zealand the horses were subjected to a minimum 21 day period of post-arrival quarantine in an insect-proof facility prior to biosecurity clearance.

Horse semen

Either:

1. The donor stallion has been resident since birth, or at least the previous 21 days, in a country that is free of VS according to the criteria within Article 2.1.2.2. of the OIE Code.

Or:

1. The donor stallions were kept on premises during the 21 days prior to semen collection where VS has not occurred during that period; *and*

2. The donor stallions were showing no clinical signs of VS on the day of semen collection.

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3 Venezuelan equine encephalomyelitis

3.1 Aetiology

Venezuelan equine encephalomyelitis (VEE) is an OIE List B disease caused members of the genus Alphavirus of the family Togaviridae. VEE is closely related to the viruses which cause Eastern and Western equine encephalomyelitis (EEE, WEE). [1, 2, 3, 4]

Within the VEE complex there are six antigenic subtypes (I-VI), containing a number of antigenic variants. Antigenic variants I-AB and I-C are associated with epidemic cycles in horses. The other variants within subtype I and the other subtypes are associated with endemic cycles involving rodents, mosquitoes and birds, and are not normally pathogenic for horses. [1, 2, 3, 4] However, outbreaks of equine encephalomyelitis in Mexico between 1993 and 1996 were caused by endemic VEE viruses. [5]

The epidemic VEE viruses probably arise through point mutations and natural selection amongst the endemic viruses maintained in sylvatic cycles. [5]

3.2 Susceptibility

VEE viruses infect Equidae, humans, birds, rodents, dogs, bats, rabbits, marsupials and non-human primates. [2] In humans the disease is often fatal. [1, 2, 3, 4]

3.3 Distribution

VEE viruses are restricted to the Americas. Epidemics of VEE have occurred in many Central and South American countries, but there have been no occurrences in the USA since 1972. Chile and Argentina have not recorded epidemics of VEE. [1, 6]

Endemic VEE viruses exist in the tropical and subtropical Americas, including the Florida everglades, Mexico, Central America and northern South America. Foci tend to involve specific VEE subtypes and variants restricted to particular areas of tropical wet forest. In these areas endemic VEE viruses cycle year round as a result of evenly distributed rainfall. [1]

VEE has never occurred in New Zealand.

3.4 Clinical signs

VEE tends to occur in epidemics. All the equine encephalomyelitis viruses produce from mild to severe encephalitic diseases that are clinically indistinguishable. During epidemics of VEE horses develop fever and depression, followed by a range of nervous signs culminating in recumbency, convulsions and death.[4] Infections of horses with endemic VEE viruses may be subclinical or result in mild transient fever, although nervous signs can also develop.[5]

Fever occurs 12-24 hours and neurological signs 5-6 days after infection.[4] For the purposes of

international trade the OIE defines the infective period for VEE as 14 days and the incubation period as 5 days. [7]

Mortality rates in horses differ with the strain of the virus. During epidemics it may be 40-80%. [1, 8]

3.5 Transmission

Transmission requires insects to act as vectors. During epidemics VEE is transmitted by many species, while endemic VEE tends to cycle between *Culex* spp. mosquitoes and vertebrates. [5] At least 41 species from 11 genera of mosquitoes have been found to be naturally infected, including the genera *Culex*, *Aedes*, *Psorophora* and *Deinocerites*. [2] Blackflies may also play a role in transmission. [5]

Infection of horses with epidemic VEE viruses results in high titre viraemia, and horses are the most important amplifying host. Normally, titres in other mammalian hosts are not sufficient to infect vectors, but these other species may act as sentinels.[8] The period of viraemia in horses is from the onset of pyrexia at 12-24 hours post-infection until the production of neutralising antibodies at about 5-6 days post-infection, the time when neurological signs appear. [4]

While no report of transmission by artificial insemination has been found, the high viraemia that occurs in horses suggests semen collected from viraemic animals could be infective.

3.6 Diagnosis

Definitive diagnosis relies on virus isolation on cell culture from blood or post-mortem samples, with identification by CFT, HI, plaque reduction neutralisation test (PRN), or by immunofluorescence. [4] RT-PCR assays for VEE identification will probably become available in the near future. [9]

Presumptive diagnosis can be achieved through demonstrating seroconversion or a rising antibody titre using CFT, HI, PRN and IgM capture ELISA. Specific antibodies against epidemic VEE variants can be detected using the PRN test and by IgM capture ELISA. [4]

3.7 Immunity

VEE vaccines are available and widely used in endemic areas in combination with WEE and EEE vaccines. Equine VEE vaccines probably do not protect against endemic VEE viruses.[4]

3.8 Treatment

There is no specific treatment. [1]

3.9 Risk assessment

3.9.1 Release assessment

The risk of VEE must be considered when horses are imported from Central and South America. Horses infected with epidemic VEE develop high titre viraemias, and so present a real risk of spreading virus if imported. The distribution of endemic VEE viruses is well documented, and horse infections and movements probably do not play a significant role in endemic VEE epidemiology. Epidemics attract international attention, particularly when they occur outside the known endemic areas. Animal and public health surveillance systems in the Americas monitor VEE outbreaks and activity, and provide a sound basis for determining country health status.

There is a high seroprevalence in horses within the endemic areas, and there may also be high prevalence of infections during epidemics. However, the infective period is short because viraemia ends with the production of neutralising antibodies around 1-2 weeks after infection. Vaccination of horses in endemic areas and in areas at risk of epidemics reduces the risk of importing horses viraemic with epidemic VEE viruses (though vaccination may not protect against endemic viruses).

The high viraemia suggests semen collected from recently infected stallions could contain virus, but there are no reports of artificial insemination being a pathway for transmission.

3.9.2 Exposure assessment

Horses develop high titre viraemia and act as amplifying hosts during epidemics of VEE. Importation of a viraemic horse has the potential to lead to infection of insects and transmission to other horses, although this would be dependent on capable insect vectors being present here. The potential of New Zealand insect species to act as vectors of VEE has not been tested, but *Culex* spp. with proven arbovirus vector competence do occur here and VEE viruses are able to infect a wide-range of insect species.

Nevertheless, it seems unlikely that endemic VEE cycles would establish here, as such cycles have never established outside of the Americas, nor in temperate areas of the Americas.

3.9.3 Consequence assessment

Although establishment in New Zealand is unlikely, an imported clinical case of equine encephalomyelitis could result in adverse consequences. Many countries prohibit the importation of horses and semen from VEE infected countries.

VEE is an important zoonosis and there would be public health implications from any occurrence in New Zealand.

New Zealand conducts no routine serological surveillance for VEE. There is therefore no justification for excluding VEE seropositive horses.

3.9.4 Risk estimate

Although VEE is unlikely to establish endemic cycles in New Zealand, infected horses could be imported from endemic or epidemic areas during periods of virus transmission, and transmission to other animals and humans in New Zealand could occur. The most significant risk would be from horses infected with epidemic VEE viruses. Any clinical cases could result in New Zealand temporarily losing its health status as a VEE-free country. This, in turn, could have significant trade consequences for the equine industries. Measures to ensure infected horses are not imported are warranted. The risks associated with semen imports are uncertain, and measures for donor stallions are warranted.

3.10 Risk management

3.10.1 Risk management objective

All horses should be protected from infection during the pre-export period, and recently infected horses should be ineligible for export.

Horses that have been resident in a VEE free country for a minimum of 21 days prior to export do not present a risk of disease introduction.

Horses imported from VEE infected countries should be isolated for 21 days in arthropod-proof facilities. The presence of viraemic horses within the isolation facility should be avoided, and could be achieved by requiring all horses entering isolation to have been previously vaccinated, or by serological testing during isolation. Effective vaccines for VEE are available and widely used, and the OIE Manual provides recommendations for vaccine manufacture and administration. [4] In the case of unvaccinated horses, the first serological test should be conducted not less than 7 days after entering isolation. Any positive test should result in the re-test of all in-contact horses not less than 14 days subsequently. All unvaccinated horses should demonstrate negative, stable or declining antibody titres. The CF, HI, PRN or IgM capture ELISA should be considered acceptable tests.

A minimum 7 day period of post-arrival quarantine in arthropod-proof facilities would further reduce the chances of a VEE infected horse being released.

Stallions from which semen is collected should have a negative, stable or declining antibody titre for tests applied on the day of collection and 14-21 days subsequently.

3.10.2 Risk management measures

The OIE Code [7] defines a VEE free country in Article 3.4.12.2. The definition includes that imports of horses from infected countries must follow the recommendations of Article 3.4.12.5. The measures within Article 3.4.12.5 are consistent with the risk management objectives discussed above.

The OIE Code [7] notes VEE free countries may prohibit the importation of horses or horse semen

from VEE infected countries. Such a measure is not considered necessary to safeguard New Zealand's health status.

Live horses

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free from VEE according to the criteria within Article 3.4.12.2 of the OIE Code.

Or:

1. When importing from countries where VEE occurs, the horses were kept in a pre-export isolation facility during the minimum 21 days prior to export, and protected from vectors during this period and during movement to the port of export; *and*

2. Either i) The horses were fully vaccinated against VEE (two doses given 2-4 weeks apart as a primary regime, followed by annual revaccination, using an inactivated vaccine for VEE either alone or in combination with EEE and WEE) not less than 60 days and not more than 1 year prior to export;
Or ii) The horses were subjected to the CF, HI, PRN or IgM capture ELISA for VEE, not less than 7 days after entering pre-export isolation. If any positive results were recorded, all horses were subjected to a repeat test not less than 14 days subsequently. The results must indicate all horses had negative, stable or declining antibody titres; *and*

3. The horses were showing no clinical signs of VEE during pre-export isolation and on the day of export; *and*

4. Upon arrival in New Zealand the horses were subjected to a minimum 7 day period of post-arrival quarantine in an insect-proof facility.

Horse semen

Either:

1. The donor stallions have been resident since birth, or at least the previous 21 days, in a country that is free from VEE according to the criteria within Article 3.4.12.2 of the OIE Code.

Or:

1. When importing from countries where VEE occurs, the donor stallions were subjected to the CF, HI, PRN or IgM capture ELISA for VEE on the day of collection and between 14 and 21 days after collection of semen for export. The results must indicate the donor stallion had a negative, stable or declining antibody titre; *and*

2. The donor stallions were showing no clinical signs of VEE on the day of collection and for the subsequent 21 days.

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4 Eastern and Western equine encephalomyelitis

4.1 Aetiology

Eastern and Western equine encephalomyelitis (EEE and WEE) are OIE List B diseases caused by a complex of viruses of the genus *Alphavirus* in the family Togaviridae. The viruses are closely related to the VEE viruses. [1, 2, 3, 4]

4.2 Susceptibility

In North America the main endemic cycles of transmission of both EEE and WEE involve passerine birds and specific mosquitoes. Foci of endemic cycles involving other vertebrates also occur. The species involved in WEE endemic cycles in South America have not been established. [2]

During epidemics a wide range of domestic and wild birds and mammals (as well as snakes, turtles, tortoises and frogs) become infected, and a much wider range of mosquitoes and other arthropods become involved in transmission. Horses and humans may be infected during these epidemics, which typically occur from mid-summer to late autumn. [1, 2]

The incidence of confirmed equine encephalitis cases in the United States between 1972 and 1993 ranged from four to 409 cases of EEE and eight to 703 cases of WEE per year. [1]

In humans, EEE causes severe disease with approximately 65% mortality and a high level of permanent sequelae. WEE is usually mild in adults, but more severe in children. Mortality is approximately 3-14%. [4]

4.3 Distribution

Equine encephalomyelitis viruses are restricted to the Americas. [1, 2, 3, 4] WEE occurs in the western states of the USA and the western provinces of Canada, as well as in Mexico, Central and South America. Typically WEE in horses occurs as sporadic cases over a wide geographic area. [4] Epidemics with high attack rates may also be recorded, such as in California in 1952 when 1,120 horses were affected out of a population of around 100,000. [1]

EEE occurs in the eastern and southern states of the USA (but also extends into the midwestern states and overlaps with WEE), Quebec and Ontario in Canada, Mexico, the Caribbean, and Central and South America. [4]

There is a marked seasonal incidence of both diseases as a result of vector activity. The great majority of cases occur in mid to late summer in temperate climates or the wet season in tropical climates, when insect populations are highest. [4] The first frost ends transmission cycles in temperate areas by killing mosquito populations. [1] The means by which the EEE and WEE viruses survive over the winter has not been established. [1, 2]

Neither EEE nor WEE have been recorded in New Zealand.

4.4 Clinical signs

All the equine encephalomyelitis viruses produce from mild to severe encephalitic diseases that are clinically indistinguishable. The incubation period is 5-14 days. Clinical signs may be as mild as a transient fever, or may progress to nervous signs and death within 24-48 hours. [4]

Morbidity in horses varies widely and is dependent upon seasonal conditions and prevalence of insect vectors. The mortality rate differs with the strain of the virus. With WEE it is usually 20-30%. With EEE it is between 40-80%. [5]

4.5 Transmission

In North America, the primary vector of WEE virus is the mosquito *Culex tarsalis*, and the endemic cycle involves passerine birds. *C. tarsalis* is not found in South America, and the species involved in endemic WEE cycles there have not been established. [1, 2]

The primary vector of EEE is the mosquito *Culiseta melanura*, again with maintenance of infection in passerine birds. Foci of infection involving other vertebrate and mosquito species also occur. [1, 2]

Infections of other animals and birds occur when mosquito populations build up in summer and autumn and their feeding habits change. Widespread infection of other animals and birds then leads to infections transmitted by other mosquitoes, ticks, blood-sucking bugs, chicken mites and lice. [2, 5]

While tropical climates allow for year-round transmission between birds and mosquitoes, the mechanisms by which the viruses over-winter in temperate areas remains to be determined. [2] Migratory birds appear not to be involved in spreading EEE or WEE. [2, 5]

Horses and humans are normally regarded as dead-end hosts because viraemia is of insufficient titre to re-infect mosquitoes. However, there is evidence that in rare cases of EEE high titre viraemia may occur in horses. [1, 2]

4.6 Diagnosis

Although the clinical signs of EEE, WEE, VEE, Borna disease, Japanese encephalitis and rabies in horses are indistinguishable, a presumptive diagnosis based on season, location, clinical signs, and the known distribution of these viruses can generally be made. [4]

Definitive diagnosis is by virus isolation on cell culture from post-mortem brain samples, with identification by direct immunofluorescent staining or a neutralisation test. [4]

Experimental infection with EEE and WEE in horses leads to a detectable antibody response in 5-10 days, [5] and high seroprevalence in the general equine population results from widespread

inapparent infection, vaccination and cross-reactions between EEE and WEE. Paired convalescent sera are necessary to make a diagnosis based on serology. CFT, HI, PRN and IgM capture ELISA tests are available. [4]

4.7 Immunity

Immunity after natural infection is long-lasting, and is passed in colostrum to foals. [2, 5]

Inactivated EEE and WEE vaccines are commercially available, and considered safe and highly effective. They are widely used in endemic areas. The OIE Manual provides recommendations for vaccine manufacture and administration. [4]

In the USA, 46.3% of establishments with horses less than 12 months of age, 57.2% of establishments with brood mares, and 63.2% of establishments with other horses over 12 months of age vaccinate against encephalitis. [6]

4.8 Treatment

No specific treatment is available.

4.9 Risk assessment

4.9.1 Release assessment

The risk of horses being infected with EEE or WEE must be considered when horses are imported from the Americas. Variations in seasonal incidence mean the risk of infection varies during the year, and will approach zero during winter in temperate regions.

Widespread vaccination and vector control programmes have led to a declining incidence of disease in North America. [1] Widespread use of vaccines contributes to high seroprevalence, making an estimate of natural incidence of infection difficult. Historical data indicate that incidence may be high during epidemics.

No infective period can be described for horses. They are considered a dead end host because viraemia of sufficient titre to infect mosquitoes does not occur. The only exception may be rare cases of high titre viraemia associated with EEE.

No reports of EEE and WEE infectivity in semen of horses or transmission by artificial insemination have been found. Such transmission is considered very unlikely because of the lack of viraemia.

4.9.2 Exposure assessment

Horses are generally considered as dead end hosts, with the exception of the rare cases of high titre viraemia associated with EEE. *Culiseta melanura*, the mosquito vector in endemic cycles of EEE, is not found in New Zealand. Neither are the other mosquito species which are considered

important in EEE and WEE epidemiology. For these reasons there is probably no chance of an imported case leading to further cases here.

4.9.3 Consequence assessment

The consequences of a single clinical case in an imported animal would relate principally to the disease investigation. The clinical signs of equine encephalomyelitis are suggestive of a number of different viruses, some of which have implications for international trade. Exports of horses and their semen could be stopped until a definitive diagnosis was established. Actions by trading partners would probably not continue once a diagnosis was established, because further cases would not be expected.

EEE and WEE are zoonoses, so the public health implications of imported cases must be considered, in particular by persons investigating imported cases. Mosquito transmission from an imported infected horse to humans is highly unlikely.

4.9.4 Risk estimate

Even though the risk of introducing EEE and WEE is low, the trade implications of imported cases warrant safeguards for live horses. Safeguards against EEE and WEE in semen are unnecessary.

4.10 Risk management

4.10.1 Risk management objective

In order to prevent importation of clinical cases of EEE and WEE, horses should be protected from infection during the pre-export period.

For importation from countries outside of the Americas, a declaration of residency for the 21 days prior to export on premises where no cases of equine encephalomyelitis have occurred is appropriate. This measure acknowledges that there are other causes of infectious encephalitis to which horses are susceptible, and is considered appropriate to account for the risk of exposure to such agents in the pre-export period.

For countries in North, Central and South America, clinical freedom on the day of export also provides an effective safeguard alone, because of the short incubation period. The risk of horse being infected with EEE or WEE in the 1-2 weeks prior to export could be managed by vaccination. Effective vaccines for both EEE and WEE are available.

4.10.2 Risk management measures

The OIE Code [7] recommends safeguards based on clinical freedom, and either 3 months premises freedom, 21 days insect-proof isolation, or vaccination. These measures are consistent with the discussion above, although MAF suggests that the widespread availability of vaccines makes these more practical than insect-proof isolation.

Live horses

Either:

1. In the case of all horses, they were kept during the 21 days prior to export on premises where cases of equine encephalomyelitis have not occurred during that period; *and*

2. The horses were showing no clinical signs of equine encephalomyelitis on the day of export.

Or:

1. When importing from countries in the Americas, the horses were vaccinated against EEE and WEE (two doses given 2-4 weeks apart as a primary regime, followed by annual revaccination, using an inactivated vaccine for EEE and WEE either alone or in combination with VEE) not less than 60 days and not more than 1 year prior to the scheduled date of export; *and*

2. The horses were kept during the 3 months prior to export on premises where EEE or WEE have not occurred during that period; *and*

3. The horses were showing no clinical signs of equine encephalomyelitis on the day of export.

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5 Equine infectious anaemia

5.1 Aetiology

Equine infectious anaemia (EIA) is an OIE List B disease caused by a lentivirus of the family Retroviridae. [1, 2, 3, 4]

5.2 Susceptibility

All species, breeds and age-groups of Equidae are considered susceptible to infection and act as the reservoirs. EIA virus does not replicate in any other animals or insects. [4]

Donkeys may be resistant to clinical signs of EIA. Three donkeys inoculated with EIA virus remained asymptomatic for 1 year with lower viraemia than recorded in ponies and horses. [5]

5.3 Distribution

EIA occurs worldwide. [2, 3, 4, 5]

In Australia EIA occurs with a low sporadic incidence and is confined to certain regions. [6] In Victoria the disease has not been recorded since 1978 (pers. comm. Patricia Ellis, Natural Resources and Environment, Victoria, Australia, 27 April 1998). EIA is notifiable in Queensland, New South Wales, Victoria, South Australia and Western Australia. Surveillance data from the Australian National Animal Health Information System indicate that between July 1993 and December 1997 a total of 6,834 horses were tested for EIA with 21 positives, all reported from Queensland (pers. comm. Robyn Martin, AQIS, 1 July 1998). In 1999 cases in horses from New South Wales were recorded after several years absence. Follow-up surveillance indicated that the prevalence in that state is very low (pers. comm. Peter Kirkland, Chief Virologist, Elizabeth Macarthur Agricultural Institute, 17 August 1999).

The USDA tested 1.31 million horses for EIA between October 1995 and September 1996. The survey found 0.11% positive horses, compared with 0.16% the previous year. Highest prevalence was in the southern-central states of Arkansas, Louisiana, Mississippi, Oklahoma and Texas. [7] Approximately 1,600 horses in the USA test positive for EIA each year [8] within the USDA control programme. [9]

In June 1999 the first ever case of EIA was recorded in New Zealand in a horse imported from New South Wales, Australia. No transmission occurred, and stamping out procedures were successfully implemented.[10]

5.4 Clinical signs

The incubation period is 1-3 weeks, but may be as long as 3 months. [3]

A febrile reaction is typically the first clinical sign detected, with ataxia, jaundice, oedema, petechial

haemorrhages in the mucosae and anaemia following. Acute infections may result in death within 10-14 days of onset of signs. Other horses appear to recover, but will typically enter a 2-3 week cycle of relapse and recovery. Death may also occur during relapses. Relapses may continue for a year and then cease. [4, 11]

5.5 Transmission

Once infected with EIA horses probably remain carriers for life. The disease tends to be manifested in slowly spreading outbreaks after the introduction of a subclinically infected horse. Blood is the principal infective agent, but all tissues, secretions and excretions may contain the virus, including milk, semen and urine. [3] Foals will become infected in utero or via the milk of their infected dam. [11]

Mechanical transmission by blood-feeding insects is the most important pathway for spread. In the USA, tabanid flies, including the horse flies *Tabanus* spp. and *Hybomitra* spp. and the deer flies *Chrysops* spp., are considered the most important vectors. *Stomoxys calcitrans*, the stable fly, is another important mechanical vector. [2, 3, 5, 11]

Iatrogenic transmission occurs. EIA virus survives for up to 4 days on hypodermic needles. [2, 5, 11] Contaminated twitches, bridles and gags have been implicated. [4, 11]

Viraemia precedes the onset of clinical signs by 2-7 days. [4] Titres vary widely between horses, and are often reported as highest during bouts of illness, [3, 5] but clinical signs are not a reliable indicator of the amount of circulating virus. [12]

A variety of factors affect insect transmission, including distance, insect feeding behaviours, interruption of insect feeding, and virus titres. Virus infectivity in insects is lost in 4 hours, and so transmission occurs only if an insect feeding on a viraemic horse is interrupted and moves to continue feeding on another horse. As a result, spatial barriers of more than 100 metres reduce mechanical transmission by insects. [2, 5]

5.6 Diagnosis

Virus isolation is not usually necessary because the high rate of carriers makes serological diagnosis a reliable indicator of health status. [3]

Circulating antibody may take 2-3 weeks, and occasionally up to 45 days, to reach a detectable titre. Titres then remain consistently high. The agar gel immunodiffusion test (AGID, or Coggin's test) is a simple and reliable test, and is the OIE's prescribed test for international trade. The competitive ELISA (c-ELISA) is also considered reliable, although positive results are usually confirmed using the AGID, because false positives occur. [3]

5.7 Immunity

There are no vaccines commercially available for EIA. [3]

Passive immunity is transferred to foals. Their serum levels become negative at 65-182 days.[11]

5.8 Treatment

No specific treatments are available. [11]

5.9 Risk assessment

5.9.1 Release assessment

EIA occurs essentially worldwide, and so the risk of introduction must be considered whenever horses and semen are imported. The prevalence of EIA varies between countries, and between areas within an infected country, and the risk of importing an infected horse will vary accordingly. EIA is notifiable in most countries.

Infected horses are considered lifelong carriers. Subclinical infections are common, and most chronically infected horses experience periods of remission.

In the absence of any risk management measures prior to export, the likelihood of an EIA infected horse being imported would be primarily dependent on the prevalence of infection in the exporting country or area.

5.9.2 Exposure assessment

Competent insect vectors of EIA are present in New Zealand. In particular, *Stomoxys calcitrans* occurs in association with horses. This suggests that EIA could spread if an infected horse were imported, although the risk of this would be dependent on the time of year and location of the imported horse. Observations in Australia indicate that climate and ecological factors may be conducive to a low sporadic prevalence of EIA within certain areas.

The presence of EIA virus in semen of viraemic horses suggests that spread by artificial insemination should be considered possible.

5.9.3 Consequence assessment

Introduction and establishment of EIA would result in direct adverse impacts from the initial investigation and efforts to control or eradicate the disease, and the clinical effects of disease. Impacts would be confined to the equine industries.

Indirect consequences would probably be limited, as EIA is present in most countries New Zealand exports horses and semen to. Exports to Australia would probably continue, with a testing component to pre-export measures. The export trade in equine blood products could be affected, requiring increased herd of origin surveillance for EIA in order to appropriately certify product health status.

5.9.4 Risk estimate

The risk of imported horses or semen being infected with EIA is primarily dependent on the prevalence of EIA in the area of origin. If introduced, EIA could spread to other horses here, but establishment may be geographically limited by equine population density, the time of year, vector distribution and climate. Establishment of EIA could have significant consequences for the equine industries. Measures to ensure infected horses and semen are not imported are warranted.

5.10 Risk management

5.10.1 Risk management objective

Any seropositive horse, and semen derived from seropositive horses, should be ineligible for importation. Horses should be protected from infection in the pre-export period.

Pre-export serological testing using the AGID or c-ELISA will detect seropositive horses. The test regime must account for the lag phase between infection and development of detectable antibody titres, which may be up to 45 days. Horses would need to be protected from infection from the period 45 days prior to testing until the time of export.
Premises of origin freedom from cases of EIA would provide a spatial barrier to decrease the risk of insect transmission. Requiring EIA to be notifiable in the exporting country will ensure appropriate official records are available for reference.

Pre-export measures for donor stallions should be consistent with the live horse recommendations.

The risk of introducing EIA into California was modelled for imports from low (0-0001-0.010%), medium (0.011-0.50%) and high (0.51-1.5%) prevalence countries. Pre-export measures modelled were clinical freedom and negative AGID pre-export test. Post-arrival measures modelled were a negative AGID test during 3, 7 and 14 days of post-arrival quarantine. The model predicted no infected animals would arrive from low prevalence countries. From medium prevalence countries, six infected animals would arrive and one would be released after 3 days of quarantine, but none would be released after 7 days. From high prevalence countries, nine infected animals would arrive, one would be released after 3 or 7 days quarantine, and none would be released after 14 days. [(13)]

A direct comparison with the risk of introduction into New Zealand is not possible because the data were specific to imports into California. However, the study indicates that post-arrival quarantine is of little additional benefit during imports from low-prevalence countries, such as Australia, because the risk of importing an infected horse is already very low. When importing from medium to high prevalence countries, post-arrival quarantine for periods of 7-14 days, with repeat serological testing during this period, will reduce the risk of infected animals being introduced.

5.10.2 Risk management measures

The OIE Code [14] recommends that importing countries require certification of clinical freedom from EIA, 3 months premises freedom, and negative serology during the 30 days prior to export. These measures are consistent with the pre-export measures discussed above. The Californian risk analysis suggests post-arrival quarantine and testing would further reduce the risk during importations from medium and high prevalence countries. This is considered justified for New Zealand, in order to maintain our status as free from EIA.

New Zealand horses often make brief visits to compete in Australia. If the visit is less than 21 days serological testing will be of limited use because seroconversion will probably not occur during this period. Under these circumstances, the only practical measure to manage the EIA risk is premises freedom declarations. Good records of movements while in Australia, and keeping such movements to a minimum, would assist export-certifying veterinarians to verify this requirement.

Live horses

1. EIA is a notifiable disease in the exporting country; *and*

2. The horses were kept for the 3 months prior to export on premises where EIA has not occurred during that period; *and*

3. The horses were subjected to the AGID or c-ELISA test for EIA during the 21 days prior to export, with negative results (unless being re-imported into New Zealand from Australia after a visit of less than 21 days); *and*

4. The horses were showing no clinical signs of EIA on the day of export; *and*

5. When the prevalence of EIA in the exporting country is assessed as medium to high, upon arrival in New Zealand the horses were subjected to a minimum 7 day period of post-arrival quarantine, during which time they were tested for EIA using the AGID or c-ELISA with negative results, prior to biosecurity clearance.

Horse semen

1. EIA is a notifiable disease in the exporting country; *and*

2. The donor stallions were kept on premises during the 3 months prior to collection where EIA has not occurred during that period; *and*

3. The donor stallions were subjected to either the AGID or c-ELISA test for EIA not less than 21 days after entry onto the semen collection centre with a negative result; *and*

4. The donor stallions were showing no clinical signs of EIA on the day of collection.

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6 Equine influenza

6.1 Aetiology

Equine influenza (EI) is an OIE List B disease caused by distinct subtypes of influenza A viruses in the family Orthomyxoviridae. The subtypes are designated subtype A Equine 1 (with the haemagglutinin and neuraminidase combination H7N7) and subtype A Equine 2 (H3N8). Antigenic drift occurs within these subtypes, particularly in the haemagglutinin. [1, 2]

EI virus isolates are identified according to the location and year of outbreaks they cause, for example influenza A/equine-1/Prague/56 and influenza A/equine-2/Miami/63. Recent outbreaks have mostly been influenza A/equine-2. [1]

Minor antigenic drift has been identified within subtype 1, whereas more extensive antigenic drift has been detected in subtype 2. Occasional major or subtype changes, called antigenic shifts, occur as a result of recombination with other influenza viruses. [1, 2]

6.2 Susceptibility

All Equidae are susceptible to EI, and the features of the disease are similar in horses, donkeys, mules and zebras. [3, 4]

Horses of all age-groups are susceptible. As maternal antibodies decline risk increases, and between the ages of 2-6 months foals are considered to be most susceptible. Most cases of disease occur in horses younger than 2 years, as older animals tend to be at least partially immune. [4]

Most outbreaks occur during the summer months. This is probably due to an increase in horse movements at this time of year, as there is no evidence that prevalence is influenced by climatic conditions. [3]

6.3 Distribution

Equine influenza occurs widely throughout the world. Australia and New Zealand have the only major horse populations so far unaffected. [1, 3, 4]

In North America and Europe there have been several major outbreaks in the last 30 years, and continual smaller outbreaks. The disease was introduced into South Africa in 1986 as a result of introduction of horses from the USA, and led to severe economic losses particularly in the racing industry.[5] Introduction into India in 1987 resulted through imports of horses from France. In 1989 there was a major outbreak in China that appears to not have been related to horse imports.[1] In 1997 a major outbreak occurred in the Phillippines, with 1,800 horses affected, 90% of these within 3 weeks. [6]

6.4 Clinical signs

The incubation period is 1-5 days. Clinical signs resolve in 1-3 weeks. [1, 3, 4]

The virus infects the epithelial cells of the respiratory mucosa, particularly in the upper respiratory tract. [1, 3, 4] Viraemia, if it occurs, is mild and brief. [4]

Fever, coughing and mucopurulent nasal discharges are the clinical signs in fully susceptible horses. Secondary pneumonia may result in death, but mortality is normally very low. The 1989 Chinese epidemic was a notable exception, with morbidity rates of 80% and mortality rates of 30% in unvaccinated rural horses. In partially immune horses, clinical signs are usually mild and indistinguishable from other common respiratory infections, or infection may be subclinical. [1]

6.5 Transmission

EI is a highly contagious disease that is transmitted directly from acutely infected to susceptible horses. Horses remain infectious for up to 7-10 days. No long term carrier state has been demonstrated. [1, 3, 4]

Rapid transportation of horses over long distances by air is attributed as a key factor in the spread of EI in the last two decades. [3, 6]

The virus is present in aerosols created by the coughing of infected horses. [1, 3] Aerosol spread over 32 metres has been recorded. Longer-distance aerosol or wind-borne transmission has been suggested as a possibility and suspected on epidemiological evidence.[3]

Observations during the 1986 South African outbreak suggested personnel, contaminated transport vehicles and other equipment contributed to the rapid and wide distribution. [1, 3, 5]

No reports have been found describing EI infectivity in semen or transmission by artificial insemination.

6.6 Diagnosis

Definitive diagnosis is by virus isolation. Typing of new isolates is by haemagglutinin inhibition using specific antisera. [2]

Two antigen capture ELISAs have been developed to detect viral nucleoprotein in nasopharyngeal swabs. [1, 6] Relatively low sensitivity and specificity and the short duration for which infected animals will give positive results indicate these tests are probably more useful as a management tool during investigation of outbreaks, rather than as a screening test during trade.

The serological test most commonly used is HI. Single radial haemolysis (SRH) may be a more sensitive test in vaccinated animals. The OIE Manual describes both procedures. Widespread vaccination and natural infection necessitate that serological diagnosis involve testing paired serum samples, 10-14 days apart, to demonstrate a rise in titre. [2]

6.7 Immunity

The response to natural infection with EI is an immunity that typically lasts for around 12 months. [1] Maternal antibodies are passed in the colostrum to foals, but decline to susceptible levels in 2-6 months. [4]

Vaccines are widely available and routinely used. Immunity is short-lived, and vaccination at short intervals (4-6 months) is necessary. A proportion of horses responds poorly to vaccination, requiring repeat vaccination to achieve protective titres of antibody. [2] The rate of poor responders is at least one in every 10 horses. [6]

In the USA, 46.5% of establishments with horses less than 12 months of age, 61.2% of establishments with broodmares, and 63.0% of establishments with other horses over 12 months of age vaccinate against EI. [7]

The SRH test may be used to measure antibody response to vaccination. A protective titre of antibodies has been determined to be 150 mm^2 (equivalent to relative antibody concentration of 44) by SRH. [5] SRH antibody concentration was highly predictive of susceptibility to EI during a study of vaccinated and unvaccinated thoroughbreds.[8] However, although 94% of vaccinated yearlings had protective titres following vaccination this proportion had declined to less than 25% 4 months later.

Surveillance of emerging influenza viruses to monitor antigenic shifts allows vaccine strains to be kept up to date. Antigenic drift and shift compromise the efficacy of vaccines. [5] An expert panel [9] has noted that as a matter of urgency vaccines in use in Europe should contain representatives of the American viruses. The panel recommended vaccines against equine influenza contain a Prague/56-like virus as the equine-1 (H7N7) component; either Suffolk/89 or a Newmarket/2/93-like virus as the Euroasian equine-2 (H3N8) component; and a Kentucky/94-like virus as the American equine-2 (H3N8) component.

6.8 Treatment

There is no specific treatment, although hyperimmune serum may be used in some cases, particularly foals. [3, 4]

6.9 Risk assessment

6.9.1 Release assessment

The risk of EI introduction must be considered when live horses are imported from any country outside Australasia. Imports of semen do not present a risk of disease introduction.

Although international notification of EI outbreaks provides an important aid to assess the risk associated with a particular consignment, all consignments from infected countries present a risk because movement controls during outbreaks vary, and subclinical infections may go undetected.

The prevalence of infection in countries where EI occurs endemically cannot be determined by serological methods because of widespread vaccination. EI is considered highly contagious, and high morbidity is recorded on premises and in populations during epidemics.

The duration of the infective period is relatively short, and long-term carriage, if it occurs at all, is rare. Obvious clinical infection will occur within 1-2 days in groups of fully susceptible horses in close contact. However, the pattern and signs of infection may vary in partially immune animals such that no obvious clinical signs occur.

6.9.2 Exposure assessment

EI is a highly contagious disease transmitted directly between horses via the respiratory route. Aerosols are created during coughing. Humans, vehicles and equipment may also be important means of transmission during outbreaks.

EI has never been recorded in New Zealand. Vaccination is not practised. The equine populations here are fully susceptible.

Explosive disease outbreaks are likely within New Zealand's immunologically naive equine populations.

6.9.3 Consequence assessment

The consequences of EI entry and establishment in New Zealand are likely to be severe.

Direct affects would result from control efforts and clinical disease. High morbidity would be very likely, and mortality rates as seen during the Chinese outbreak could occur. If introduced, EI is unlikely to be eradicated and national control would probably rely on vaccination as it does in other parts of the world.

Indirect effects would severely impact the domestic equine industries. South Africa achieved control during the 1986 outbreak only after a country-wide ban on movements of horses.[5] A similar move in New Zealand would see race meetings and other events cancelled. The total losses resulting from cancelling horse gatherings in New Zealand for a 4 week period have been estimated to be approximately $175 million.[10] The export trade in live horses would be disrupted. Australia would probably introduce requirements for vaccination and post-arrival quarantine for New Zealand horses.

EI is the infectious disease that probably presents the most serious economic threat to the New Zealand equine industries. The total overall cost of direct and indirect effects is likely to be very high. These would probably be concentrated within the equine industries and the dependant subsidiary industries. Long term effects would particularly affect those sectors reliant on the export trade to Australia.

6.9.4 Risk estimate

The likelihood of EI being introduced into New Zealand through live horse imports is high, and an outbreak would have serious and long-term consequences for the equine industries. Measures to ensure horses infected with EI are not imported are warranted, and the protection afforded by such measures should be commensurate with the magnitude of the consequences predicted if disease were to be introduced. Semen does not present a risk, and so no safeguards are necessary.

6.10 Risk management

6.10.1 Risk management objective

Risk management must rely on protecting horses from infection during the pre-export period. Detecting infectious horses in the pre-export period is presently not feasible, as there are no practical methods with appropriate sensitivity and specificity.

Horses imported from countries where EI does not occur, and which have adequate import controls and surveillance, do not present a risk.

When importing from countries where EI does occur, the risk management options include premises of origin disease freedom, vaccination, demonstration of protective titres, pre-export isolation and post-arrival quarantine.

Cases of EI on the premises of origin would increase the likelihood of infected horses moving into pre-export isolation. However, subclinical infections and the minor respiratory symptoms associated with infection in partially immune horses significantly reduce the assurance provided by safeguards relying on clinical detection of disease.

Vaccination remains the most important means of EI control in infected countries. This is despite well-recognised short-comings such as the high rate of poor responders and gradual erosion of vaccine efficacy through antigenic drift or shift, but this can be minimised by ensuring vaccines contain recommended strains providing immunity from circulating wild-type viruses. Ensuring that all horses are immunologically protected at the time of entry into pre-export isolation will significantly reduce the likelihood of introduction of virus and subsequent circulation within groups of isolated horses. The duration of immunity is probably no more than 4 months in a significant proportion of horses, so vaccination should occur within this period prior to isolation. Only horses with demonstrated protective levels of antibody should enter isolation. This can be achieved by requiring horses to be tested by SRH and demonstrate a protective level of antibodies (>150 mm^2 or 44 RAC) during the 30 days prior to entering pre-export isolation.

Vaccination of foals less than 2 months old will probably be ineffective as a result of maternal antibody interference. Up until 2 months foals are likely to be adequately protected if their dams are fully vaccinated, and so should be able to enter isolation unvaccinated on the basis of their dam's eligibility.

EI is shed for 10-12 days. Assuming a worst-case of an infected horse moving into isolation and infecting an in-contact horse at the end of the shedding period, the newly infected horse is likely to be finished shedding virus within 21 days of isolation. Notwithstanding the conservative OIE recommendation for 28 days isolation [(11)], a period of 21 days probably provides the maximum benefit to be gained from pre-export isolation in the case of EI.

Although EI can be transmitted by aerosol over short distances, pre-export isolation standards that prevent airborne spread are unrealistic. However, isolation should provide a minimum 100 metre separation between isolated and other horses at all times.

Post-arrival quarantine will further reduce the likelihood of infected horses being released, and would provide the most realistic opportunity to contain an outbreak in imported horses. A period of 14 days post-arrival quarantine is appropriate to account for the known EI virus shedding period. Once again, quarantine standards able to contain viruses spread in aerosol form are probably unrealistic. Low security facilities would provide an appropriate level of security provided there is a minimum 100 metre separation between quarantined and other horses.

Stress during transportation commonly leads respiratory signs in horses after arrival in New Zealand. Such cases are often impossible to clinically differentiate from mild EI infection. The antigen ELISAs are unsuitable as a routine pre-export screening test, but could be usefully applied in these circumstances and provide a basis for managing quarantine decisions. Any imported horse displaying respiratory signs such as coughing and sneezing should be confined in a closed stable until an antigen ELISA provides a negative diagnosis for EI.

6.10.2 Risk management measures

The OIE Code [(11)] provides criteria for a country to be recognised as EI free. The criteria include compulsory notification of all cases, no vaccination, no clinical cases for a minimum of one year, and serological surveillance to demonstrate freedom. All imports of horses have to comply with the provisions laid down in the Code. These include 28 days isolation and vaccination during the 2-8 weeks prior to export.

At the present time, New Zealand has not complied with the surveillance component of the OIE criteria for recognition as an EI free country. As such, recognition of other countries as being free from EI should be determined on the basis of their surveillance and import controls being equivalent to New Zealand's, rather than meeting the OIE criteria.

The discussion above has concluded that a 21 day period will probably provide the maximum benefit to be derived from pre-export isolation. The problems associated with vaccination have also been discussed and further measures to increase the efficacy of vaccination as a safeguard proposed, in particular SRH testing to determine that a protective antibody titre has been achieved.

Post-arrival quarantine has been discussed as a measure which would decrease the likelihood of horses shedding EI virus being released, while providing what would probably be the only opportunity to contain an outbreak in imported horses.

These measures differ from the OIE recommendations, and are considered to provide a higher level of protection. This is considered warranted because vaccination and isolation provides a significant risk that undetected cycling of virus in isolation groups could lead to infected horses being imported, and the very serious consequences such an event would have for New Zealand.

Live horses

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free from EI.

Or:

1. When importing from countries where EI occurs, the horses were kept for the 3 months prior to export on premises where EI has not occurred during that period; *and*

2. During the 4 months prior to export the horses (except for foals less than 2 months old when accompanied by their vaccinated dam) were vaccinated against EI using an approved inactivated vaccine either twice not less than 21 days apart, or once as a booster to a previous primary course of vaccination;
(**N.B.** Approved vaccines must contain a Prague/56-like virus as the equine-1 (H7N7) component; either Suffolk/89 or a Newmarket/2/93-like virus as the Eurasian equine-2 (H3N8) component; and a Kentucky/94-like virus as the American equine-2 (H3N8) component.) *and*

3. The horses were subjected to a SRH test during the 30 days prior to entering pre-export isolation (or upon entry into isolation) and demonstrated a protective level of antibodies against EI (>150 mm^2 or >44 RAC); *and*

4. The horses were kept for a minimum 21 day period prior to export in a pre-export isolation facility; *and*

5. Upon arrival in New Zealand the horses were subjected to a minimum 14 day period of post-arrival quarantine. No equidae should be allowed within 100 metres of the facility, and any quarantined horses exhibiting respiratory symptoms should be confined indoors until subjected to an antigen ELISA for EI with a negative result.

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7 Equine viral abortion

7.1 Aetiology

Equine viral rhinopneumonitis is an OIE List B disease caused by infection with either of two closely related alphaherpesviruses, equine herpesvirus-1 (EHV-1) and equine herpesvirus-4 (EHV-4). [1] Up until 1981 these viruses were considered to be subtype 1 and subtype 2 of EHV-1. Subsequently, most foetal isolates have been typed as EHV-1 and most respiratory isolates as EHV-4. [2, 3]

The terminology "equine viral abortion" is used in this risk analysis in order to distinguish two clinical syndromes caused by EHV-1, abortion and neurological disease, which have low sporadic occurrence in New Zealand, from equine rhinopneumonitis caused by EHV-4, which is very common.

Most EHV-1 viruses causing equine abortions are from two strains designated EHV-1B and EHV-1P by restriction endonuclease DNA fingerprints. [2, 3]

Other related equine herpesviruses also occur commonly worldwide. EHV-5 occurs in healthy horses, as does EHV-2. EHV-2 may also play a role as a primary pathogen causing respiratory disease and a rarely fatal lymphadenopathy of foals. [4] EHV-3 is a worldwide cause of equine coital exanthema. [5]

7.2 Susceptibility

EHV-1 and EHV-4 occur in all Equidae. On rare occasions EHV-1 has been isolated from cattle (abortion), llamas and alpacas (optic nerve neuropathy and blindness), captive gazelle (encephalitis), [2] and mice. [6]

7.3 Distribution

EHV-1 and EHV-4 are considered to have worldwide distribution. [1, 2, 3] Surveys using type-specific ELISA have demonstrated antibodies to EHV-4 in 95-100% and to EHV-1 in 3-79% of various groups of horses.[7] High prevalences of latent infection with EHV-1 and/or EHV-4 have also been demonstrated using PCR methods. [8]

A New Zealand survey using a type-specific ELISA detected antibodies to EHV-1 in 17-70% of horses in the various groups examined. The study concluded that probably all New Zealand thoroughbred studs have mares latently infected with EHV-1. [9] There have been four outbreaks and a number of individual cases of EHV-1 abortion in New Zealand since the first documented outbreak in 1975. EHV-1P occurs in this country, but EHV-1B has so far not been isolated. EHV-1B has been isolated from one outbreak of abortion in Australia. [10]

Another New Zealand study concluded that although EHV-1 infection was common it was not strongly associated with respiratory disease, whereas EHV-4 was involved in two out of five

outbreaks of respiratory disease. [11]

7.4 Clinical signs

EHV-1 can cause four clinical syndromes; respiratory disease, abortion, perinatal disease and myeloencephalitis. [2]

Following primary infection with EHV-1 or EHV-4 the incubation period ranges from 2 days to 2 weeks. Clinical signs may include fever, conjunctivitis, coughing and mild inflammation of the upper respiratory tract, and usually resolve within 2-5 days although nasal discharge and cough may last 1-3 weeks. Inapparent infection is common. [3]

Pregnant mares that abort usually display no respiratory or other clinical signs. Infection via the respiratory route may have occurred months previously. Most abortions occur at 6-11 months gestation. Earlier abortions are characterised by an autolysed foetus. In late abortions the placenta may be grossly normal and the foetus expelled alive, or both may show a marked inflammatory response and necrotic foci. [2, 3]

In a study involving 180 pregnant mares inoculated with EHV-1, 87% became viraemic and 42% aborted. The interval between onset of viraemia and abortion ranged from 6-81 days (mean 18.3 days). The mean duration of viraemia was 5.2 days, at 4-10 days post-inoculation. [12]

Stillbirths, weak and dying foals, and foals which develop respiratory distress and diarrhoea during the first weeks of life may result from EHV-1 infection during the last few days of gestation. [3]

Neurological disease caused by EHV-1 usually, but not always, follows abortion and/or respiratory disease by 7-10 days. Clinical signs vary from mild ataxia to complete recumbency with limb paralysis. [2] The course of the disease varies from 2 days to several weeks. The proportion of animals affected within a group varies from a single sporadic case to 90% morbidity. [3]

7.5 Transmission

Transmission is by inhalation of infected droplets and requires close contact, as herpesviruses tend to not survive well outside the animal body. [2, 3, 12]

Following primary infection, virus is shed from the nasopharynx for 7-14 days. Virus shedding following re-infection or re-activation of latent infections is usually transient (2 to 4 days). Virus is also shed in saliva, ocular discharges and faeces. A cell-associated viraemia occurs which may last weeks or months, and the virus then enters a latent phase. [3]

The biological phases in the life-cycle of EHV-1 are infection, respiratory shedding, latency and reactivation. Abortion in pregnant mares is probably incidental and accidental as a result of virus tissue trophisms making the placenta and foetus a perfect site for virus replication. [12] While epidemiological evidence suggests that the aborted foetus, placenta and fluids provide a source of

virus to in-contact mares, [3] there are no data on transmission of EHV-1 from an aborted foetus to other horses. [12]

Latent virus is present in the sensory nerve processes of the trigeminal ganglion [13] and in lymphocytes. No viral antigens are detectable, but viral DNA may be detected by PCR. [12]

No evidence has been found suggesting EHV-1 may be transmitted by artificial insemination.

7.6 Diagnosis

The clinical picture is highly characteristic. [1]

Virus may be isolated from nasopharyngeal swabs on equine cell cultures. Identification is by immunofluorescence or PCR methods. [1]

Because of widespread infection and vaccination, paired acute and convalescent serum samples are needed for serological diagnosis. CFT, SN and type-specific ELISA are available to detect antibody. [1]

7.7 Immunity

Immunity following natural infection of the respiratory tract is short-lived, and asymptomatic re-infection may occur within 3-4 months. Immunity following abortion is more durable, but unpredictable. Abortions do not normally occur in the same mares in consecutive years. [2] There is no clear relationship between the antibody status of mares and their likelihood of aborting. [3]

Both live attenuated and inactivated viral vaccines are commercially available. Duration of vaccine-induced immunity is short, and revaccination at frequent intervals is required. Vaccination is helpful in reducing the incidence of abortion in mares, but should not be considered a substitute for management practices known to reduce risks of abortion. [2, 3]

In the USA, 28.0% of establishments with horses less than 12 months of age, 54.9% of establishments with brood mares, and 42.8% of establishments with other horses over 12 months of age vaccinate against EHV. [14]

7.8 Treatment

No specific treatment is available.

7.9 Risk assessment

7.9.1 Release assessment

EHV-1 occurs worldwide and at high prevalence. All ages and classes of horses are susceptible to infection. Latent infections with subsequent reactivation and shedding occur. Inevitably, some

horses imported will be infected with EHV-1.

Importation of semen into New Zealand probably does not present a pathway for EHV-1 introduction.

7.9.2 Exposure assessment

Horses imported within 2 weeks of primary infection may be shedding EHV-1 in respiratory secretions. However, importation of a latently infected horse may also lead to exposure of New Zealand horses following subsequent reactivation of infection.

Importation of pregnant mares may represent a higher risk than imports of non-pregnant horses. Equine viral abortion may expose in-contact horses to high levels of virus in the aborted foetus, placenta and fluids.

7.9.3 Consequence assessment

Disease caused by EHV-1 occurs in New Zealand, though rarely. Studies have demonstrated a high prevalence of infection here. Different management of horses and/or variable reporting may be significant factors in explaining the low reported incidence of EHV-1 abortions relative to the high infection rates, as may EHV-1 strain differences. EHV-1 subtype B has not been isolated in this country, [9] although vaccines available here provide immunological cover for this strain (pers. comms. Elizabeth Sommerville, Rhone Merieux, and Bruce Graham, PacificVet, September 1996 and April 1997 respectively).

The consequences associated with importation of EHV-1 infected horses would be the direct consequences associated with equine viral abortion. If a susceptible population of pregnant mares were to be exposed to EHV-1 as a result of importation, the consequences of an abortion storm could be significant to the individual studs involved. It is important to note that such an exposure could also occur following movement of horses within New Zealand.

7.9.4 Risk estimate

EHV-1 already occurs here, though some strains do not. Imports of horses are likely to lead to further introductions and eventual exposures. The consequences of further introductions would be significant to individual establishments, although whether introductions of new strains would have significantly greater consequences is uncertain. Measures to reduce the risk during importation of horses are warranted, but should not be seen as a replacement for basic biosecurity measures on individual premises, particularly vaccination and the isolation of all new arrivals. Semen does not present a risk and so no safeguards are warranted.

7.10 Risk management

7.10.1 Risk management objectives

There are no practical measures to allow imports while ensuring infected horses, in particular horses with latent infections, are not imported. Safeguards could at best minimise the risk of importing horses that may have been recently exposed to infection with EHV-1 strains capable of causing abortion and/or neurological syndromes, which are rarely seen in New Zealand. Requiring the premises of origin to have had no clinical cases is probably the only practical measure available in this respect.

Vaccination provides variable protection, and will not clear latent infections. As such it can not be justified as an official zoosanitary requirement. However, vaccination of imported horses will decrease their susceptibility and the likelihood of active shedding. Vaccination can also be used to decrease the susceptibility of horses on premises receiving imported horses. In these respects, vaccination acts to reduce commercial risks and so is advisable for all horses to reduce the incidence of clinical disease associated with EHV-1.

7.10.2 Risk management measures

The OIE Code [15] recommends 3 months premises of origin freedom from cases of equine viral rhinopneumonitis and 3 months clinical freedom for the imported horses. The premises of origin safeguards are considered appropriate if made specific for equine viral abortion (EHV-1, including neurological disease).

Live horses

1. The horses were kept for the 3 months prior to export on premises where equine viral abortion (EHV-1, including neurological disease) has not occurred during that period; *and*

2. The horses were showing no clinical sign of equine viral abortion (EHV-1, including neurological disease) on the day of export.

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8 Equine viral arteritis

8.1 Aetiology

Equine viral arteritis (EVA) is an OIE List B disease caused by equine arteritis virus (EAV), the only member of the genus Arterivirus within the family Arteriviridae.[1, 2, 3] Although there is genetic variation in EAV isolates, and these may significantly vary in pathogenicity,[4] only one serotype is recognized. [3, 4]

A phylogenetic tree constructed from nucleotide sequence information provides evidence for continuing intercontinental transmission of EAV between Europe and North America. The New Zealand strains of EAV are related to American strains. [5]

8.2 Susceptibility

EAV infects horses, donkeys, mules and zebras.[3, 4, 6] All age groups and breeds of horses are susceptible. Prevalence of serological reactors and their titre increase with age. Differences in seroprevalence between breeds may indicate the importance of host genotype susceptibility or be related to specific management practices. [4]

8.3 Distribution

Despite the limited number of disease outbreaks, serological evidence confirms EAV is present worldwide. [3, 4, 7]

In the USA 15-30% of horses are seropositive to EAV. In Austria, 27% of horses are seropositive, and EAV has been identified in 3.8% of equine abortions. In France 1-3% of horses are seropositive. Seroprevalence in Ireland is 0.3%, and clinical disease has never been reported. In Germany seroprevalence was 24.8% in 1994, up from 1.8% in 1988. In the Netherlands, up to 45% of horses older than 4 years of age are seropositive. Seroprevalence in Sweden is 35% for standardbreds and 16% for Swedish warm-bloods. Seroprevalence in the UK has increased to 2-3%, from rates of 0.5% prior to 1993 when the first outbreak of disease was recorded there. [7]

Disease attributable to EAV infection has never been recorded in New Zealand. A survey in 1988 demonstrated seroprevalence of 3% and 54% amongst thoroughbred and standardbred stallions respectively. There are five known EAV carrier stallions here, all standardbreds. A voluntary control programme has been operating within the standardbred industry. [8]

8.4 Clinical signs

There are three clinical syndromes associated with EAV infection: an influenza-like illness of adult horses, an interstitial pneumonia of very young foals, and abortion in mares. The majority of infections are subclinical. [3, 4, 7, 9]

The incubation period is 3-14 days (6-8 days following venereal infection). Pyrexia may last 1-9

days. The clinical signs vary widely among individual horses and between outbreaks. [3]

Abortions in pregnant mares may occur any time beyond 2 months of gestation (pers. comm. Peter Timoney, Gluck Equine Research Center, 31 July 1998). Different strains of EAV probably differ in abortigenicity. [3]

Infections in stallions do not result in long-term effects on semen quality, although short-term reduction associated with pyrexia may occur. [3]

8.5 Transmission

Transmission of EAV occurs through the respiratory, venereal and transplacental routes. [3]

EAV is readily transmitted via the respiratory route through direct contact with infectious nasopharyngeal secretions from acutely infected horses. Blood, faeces, ocular secretions, urine and vaginal fluids also contain virus.[3] In experimentally infected animals the virus can be recovered from the serum for up to 11 days, from the nasopharynx for 14 days, and from the buffy coat, kidney and urine for 19 or more days. [4]

Infections in stallions may lead to a carrier state characterised by shedding in the semen. Short term (2-5 weeks), intermediate term (3-8 months) and long term (1-10 years) carrier states are recognised. The mean frequency of the chronic carrier state is approximately 35%, although this varies between breeds. [3] A study of 561 seropositive stallions found a mean rate of 40.8% carriers, over 70% of which were standardbreds. [10] Spontaneous clearance of the carrier state occurs, with no evidence for reversion to a shedding state. Output of EAV by shedders appears to be testosterone-dependent, but is continuous (rather than intermittent as previously believed). [3, 10] Chronic virus shedding has not been observed in geldings. [3]

Vaccination of mares prior to mating with known shedder stallions has been demonstrated to reduce the likelihood of transmission. [11]

8.6 Diagnosis

The samples for virus isolation include nasopharyngeal and conjunctival swabs or blood from acute infections, and the semen of carrier stallions. The OIE recommends that seropositive stallions be tested by virus isolation on the sperm rich fraction from two semen samples. [2]

The OIE also recommends that shedder stallions can be identified by test mating to two seronegative mares, which are tested for seroconversion 28 days after mating. [12] Approximately 85-100% seronegative mares bred to shedding stallions will seroconvert. [3]

Semen shedders can also be identified by detecting EAV nucleic acids in semen. A reverse transcriptase PCR provides comparable results to virus isolation. [2] A nested PCR assay has also demonstrated full agreement with virus isolation in experimental situations. [13] Neither PCR assay has been adopted by the OIE for international trade, although an expert report to the OIE

considered the nested PCR assay a sensitive and reliable alternative to virus isolation in cell culture. [8] The major advantage PCR assays have over traditional virus isolation methods is speed, as they can be completed in 48 hours, whereas virus isolation requires incubation for 1 week for each cell culture passage.

The VN test is the serological test prescribed by the OIE for use during international trade. [2] The VN test detects antibodies within a week after natural infection. [3] Widespread subclinical infection and vaccination mean that serological diagnosis relies on the demonstration of rising titres. A four-fold rise over two tests taken 14 days apart is considered significant. Reading the test requires subjective interpretation, so consistency is very important. An inter-laboratory comparison [14] of the VN test found that while there was generally excellent qualitative agreement regarding positive and negative samples, there was a great deal of variation in the quantitative results of endpoint titres. Serial tests to determine the presence of stable or declining titres should be performed at the same laboratory, preferably on the same day in a side-by-side fashion.

Although a wide variety of ELISAs have been developed to detect serum antibodies and these are being applied in laboratories around the world, none are sufficiently validated to be accepted by the OIE. [14]

8.7 Immunity

Neutralising antibody titres resulting from natural infection may remain high for years, or possibly life. They provide protective immunity, although EVA has occasionally been induced in horses with positive titres. Decline to negative levels a few years after initial infection, in the absence of further exposure, may also occur. [3]

Live attenuated and inactivated vaccines are available. After two initial injections as a primary course the duration of immunity is over 1 year. Detectable neutralising antibodies develop in the majority of horses within 3 weeks of vaccination. [2] Vaccination may not prevent infection in all mares mated to carrier stallions, but greatly reduces the likelihood of clinical symptoms and titres in the nasopharynx capable of leading to subsequent respiratory transmission. [11]

In the USA, 0.4% of establishments with horses less than 12 months of age, 2.5% of establishments with brood mares, and 1.8% of establishments with other horses over 12 months of age vaccinate against EVA. [15]

8.8 Treatment

There is no specific treatment.

8.9 Risk assessment

8.9.1 Release assessment

EVA occurs worldwide. Seroprevalence studies indicate that infection is common in many

countries, and approximately 35% of seropositive stallions may be long-term carriers. There may be an increased risk associated with imports of certain breeds, such as standardbred and warm-blood horses.

Subclinical infection is very common. Acutely infected horses remain infectious for up to 19 days. Natural infection is followed by a long-lasting immunity, and vaccination also reduces the risk of contracting acute infection and subsequent shedding.

Stallions that are shedding virus in their semen present the greatest risk of EVA introduction, during both live animal and semen imports. Every semen sample collected from such stallions will contain EAV.

8.9.2 Exposure assessment

Acutely infected horses will shed EAV for a short time only, but during this time will expose in-contact horses to infection. This is the only means by which mares and geldings could introduce infection. Horses that will not be used for breeding in New Zealand, such as competition horses, also present a risk only during the acute stage of infection.

The greatest exposure risk results from shedder stallions. Importation of a shedder stallion or his semen would lead to infection in inseminated mares.

Exposure of New Zealand horses to EAV could lead to endemic cycles of subsequent respiratory shedding, further acute infections, and potential long-term persistence in shedder stallions.

Industry breeding management practices such as artificial insemination create an increased exposure risk for the standardbred and warm-blood industries. Higher seroprevalence in standardbreds in New Zealand may reflect this exposure risk, or may be the result of higher genotypic susceptibility.

8.9.3 Consequence assessment

EAV is present in New Zealand, although disease has not been recorded here. Measures for EVA are imposed during exports of horses and semen from New Zealand.

At the present time, EAV appears to be present only in the standardbred industry. The voluntary industry control programme could eventually lead to eradication. In the absence of disease, the present consequences of EAV in New Zealand are limited to those in the standardbred industry resulting from compliance with control scheme measures.

New introductions of EAV could have direct impacts associated with clinical disease, in particular abortions, and may delay or preclude eventual eradication. The origin of the virus introduced, and its pathogenicity, would determine its clinical manifestations. Like New Zealand, Australia has not recorded clinical cases of EVA and maintains import restrictions (pers. comm. Sarah Kahn, AQIS, with Barry O'Neil, 30 July 1998). EAV strains more pathogenic than those that already occur here are unlikely to be introduced through importation of horses from Australia.

Introduction of EAV to other breeds in New Zealand would require that industry groups consider adopting the controls currently being voluntarily applied within the standardbred industry.

8.9.4 Risk estimate

Although EAV occurs here, clinical disease does not, and a control programme is operating. Introduction of more pathogenic strains, or introduction into other breeds, would have adverse consequences. Measures during importation of horses and semen are warranted.

8.10 Risk management

8.10.1 Risk management objectives

Horses infected with EAV should not be imported, and susceptible horses should be protected from infection in the pre-export period. The epidemiology of EVA allows these objectives to be achieved in different ways for stallions and other classes of horse.

Measures during importation of mares, geldings and horses imported for competition purposes need only ensure the absence of acute infection. This could be achieved by prior vaccination, or by demonstration of negative, stable or declining antibody titres in the pre-export period using the OIE recommended VN test.

Measures during importation of stallions must ensure the absence of acute infection and the semen carrier state. Seronegative stallions will not be infected. Vaccinated stallions will be protected from infection, but vaccination will not eliminate a pre-existing semen carrier state. Any unvaccinated seropositive stallion should be tested by either virus isolation or test matings. A negative test at any stage during the year prior to export should be sufficient to determine a stallion's health status, because seropositive stallions that are not semen carriers will not be susceptible to re-infection or revert to shedder status.

Protection from acute infection in the pre-export period is only necessary for seronegative horses, and this could be achieved by requiring premises of origin freedom from EVA in the pre-export period, including the presence of known shedder stallions.

Measures similar to those recommended for imported stallions are appropriate for semen donors.

8.10.2 Risk management measures

For EVA, the OIE Code [(12)] recommends that all live horses should have negative, stable or declining antibody titres, or have been vaccinated immediately following a negative serological test. All unvaccinated seropositive stallions should be tested to ensure they are not semen shedders. These measures are consistent with the discussion above.

European Union directive 95/329/EC details a variety of EVA vaccination practices that would

ensure horses are not incubating or already infected at the time of vaccination or in the ensuing period prior to developing a protective antibody titre. In the case of mares and geldings, vaccination need only be completed to ensure a protective titre is achieved by 21 days prior to export.

Imports from Australia have historically only required stallions to be subjected to a test for EVA, while mares and geldings have been subjected to individual and premises of origin clinical freedom requirements. The lack of clinical manifestations of EVA in Australian horses suggests that strains present there are of similarly low pathogenicity as New Zealand strains. There is no evidence that new cases of EVA infection have resulted from horses imported from Australia, and so the present arrangements should continue.

Live horses

Either:

1. When female, castrated male and competition horses are imported, the horses were kept for the 3 months prior to export in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The horses were subjected to a VN test for EVA during the 28 days prior to export which demonstrated a negative titre;
Or: ii) The horses were subjected to two VN tests for EVA during the 28 days prior to export, on blood samples taken at least 14 days apart which demonstrated a negative, stable or declining titre;
Or: iii) The horses were vaccinated against EVA not more than one year nor less than 21 days prior to importation in accordance with the vaccine manufacturer's recommendations. (N.B. horses from Australia exempt this requirement).

Or:

1. When uncastrated male horses are imported, the horses were kept for the 3 months prior to export in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The horses were subjected to a VN test for EVA during the 28 days prior to export which demonstrated a negative titre;
Or: ii) The horses were vaccinated against EVA under official veterinary control and have been re-vaccinated at regular intervals (at least annually);
(**N.B.** Approved programmes for initial vaccination are as follows:
a. vaccination on the day a blood sample was taken which was subjected to the VN test with a negative result;
b. vaccination during a period of isolation of not more than 15 days, commencing on the day a blood sample was taken which was subjected to the VN test with a negative result;
c. vaccination when the animal was at an age of 180 to 270 days during a period of isolation, during which two blood samples taken at least 10 days apart were subjected to the VN test and demonstrated a negative, stable or declining antibody titre.)
Or: iii) The horses are seropositive to EVA, and were found not to be a semen carrier

during the one year prior to export;
(**N.B.** Approved methods for determining semen carriers are as follows:
a. test mating to two mares that were subjected to SN tests with negative results on two blood samples, one collected at the time of test mating and the other 28 days after mating;
b. virus isolation on cell culture carried out on the sperm rich fraction of two separate semen samples with negative results.)

Horse semen

1. The donor stallions were kept for the 3 months prior to collection in premises where EVA has not occurred, and where EVA shedder stallions are not known to be present, during that period; *and*

2. Either: i) The donor stallions were subjected to a VN test for EVA not less than 21 days after entering the semen collection centre which demonstrated a negative titre;
Or: ii) The donor stallions were vaccinated against EVA under official veterinary control and have been re-vaccinated at regular intervals (at least annually);
(**N.B.** Approved programmes for initial vaccination are as follows:
a. vaccination on the day a blood sample was taken which was subjected to the VN test with a negative result;
b. vaccination during a period of isolation of not more than 15 days, commencing on the day a blood sample was taken which was subjected to the VN test with a negative result;
c. vaccination when the animal was at an age of 180 to 270 days during a period of isolation, during which two blood samples taken at least 10 days apart were subjected to the VN test and demonstrated a negative, stable or declining antibody titre.)
Or: iii) The donor stallions are seropositive to EVA, and were found not to be a semen carrier during the one year prior to collection;
(**N.B.** Approved methods for determining semen carriers are as follows:
a. test mating to two mares which were subjected to SN tests with negative results on two blood samples, one collected at the time of test mating and the other 28 days after mating;
b. virus isolation on cell culture carried out on the sperm rich fraction of two separate semen samples with negative results.)

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9 Horse pox

9.1 Aetiology

Horse pox is an OIE List B disease that occurred in Europe until the early twentieth century. Currently the only poxvirus of any clinical significance in horses is the orthopoxvirus that causes Uasin Gishu disease in parts of Africa. Poxviruses also occur in horses with mild skin lesions that resemble the human condition *molluscum contagiosum*. [1, 2]

9.2 Susceptibility

Uasin Gishu disease is presumed to be transmitted from a wildlife vector to horses. The host range is unknown. [1]

9.3 Distribution

Historically horse pox was recorded throughout Europe. [1, 2, 3] No European OIE member country has reported cases in recent years. [4]

Cases with lesions similar to Uasin Gishu disease have been recorded in Kenya, Burundi, Zambia and Zaire. [1, 2]

Poxviruses have been seen in horses with a condition resembling *molluscum contagiosum* in South Africa [2] and the USA. [1]

9.4 Clinical signs

Pox viruses cause generalized skin lesions. Lesions resemble papillomas, and occur over many parts of affected horses. They may appear and disappear intermittently for years. Other horsepox infections have been described as "grease", a condition of swollen, exudative skin around the fetlocks. [1]

9.5 Transmission

Little is known about transmission, but it is presumed to be by direct contact and fomites. The role of biting insects is unknown. [1, 2, 3]

9.6 Diagnosis

Diagnosis is made on clinical signs, with confirmatory visualisation of virus particles in skin specimens by electron microscopy or by virus isolation on cell cultures. [1, 2]

9.7 Immunity

No vaccines are available.

9.8 Treatment

Local astringent treatment may be palliative. [2, 3]

9.9 Risk assessment

9.9.1 Release assessment

While importation of horses from regions where horse pox viruses are known to have occurred presents some risk, the low prevalence of infection and obvious clinical signs suggest that infected horses are unlikely to be imported.

No information has been found regarding transmission via artificial insemination.

9.9.2 Exposure assessment

Direct contact with imported infected horses would lead to exposure of horses in New Zealand.

9.9.3 Consequence assessment

The consequences of introduction and establishment of horsepox viruses here would include direct disease affects, control efforts and any export restrictions imposed by trading partners.

9.9.4 Risk estimate

The risk of imported horses introducing horse pox is low, but if this did occur there would be adverse consequences. Measures during importation of live horses are warranted. Semen probably does not present a risk, so long as stallions are clinically healthy at the time of collection. No specific measures are necessary.

9.10 Risk management

9.10.1 Risk management objective

Horses should be free from the clinical signs of infection at the time of export, and have been protected from recent exposure during the pre-export period.

9.10.2 Risk management measures

The OIE Code [5] recommends premises freedom and clinical freedom on the day of export. These measures are consistent with the objective noted above.

Live horses

1. The horses were kept for 3 months prior to export in premises where cases of horse pox

have not occurred during that period; *and*

2. The horses were showing no clinical sign of horse pox on the day of export.

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10 Japanese encephalitis

10.1 Aetiology

Japanese encephalitis (JE) is an OIE List B disease caused by a virus of the genus *Flavivirus* in the family Flaviviridae. [1, 2, 3] JE virus is a member of a complex of viruses in this genus, which includes West Nile encephalitis, St Louis encephalitis, Murray Valley encephalitis, and Kunjin virus, all of which cause sporadic cases of human encephalitis. [4] Louping ill and Wesselsbron disease are further examples of flaviviruses.

Many animal species have antibodies to the flaviviruses endemic in a particular area, although the species clinically affected tend to be much more limited. Even fewer species are important in the epidemiology of disease.

10.2 Susceptibility

The natural cycle of JE virus involves waterbirds (such as herons and egrets) as the reservoir, pigs as the primary amplifying hosts, and transmission by mosquitoes. Horses and humans are susceptible to clinical disease in the form of encephalitis, but are dead-end hosts and are not important epidemiologically. [3]

Inapparent infections and very occasional cases of clinical disease also occur in cattle, sheep, and goats. Inapparent infections occur in dogs, cats, rodents, bats, snakes and frogs. [4]

Japanese encephalitis is an important zoonosis, with about a quarter of cases being fatal. [4]

10.3 Distribution

JE virus occurs throughout eastern Asia. [3, 4] The number of cases and deaths in humans and horses in Japan has been declining steadily since a mass vaccination campaign was begun in 1948. Between 1968 and 1990 only 15 clinical cases (10 fatal) were detected in horses. [1]

JE virus occurs in Papua New Guinea and the islands of Torres Strait [5] and in 1998 was detected for the first time in northern Queensland on mainland Australia (pers. comm. Sarah Kahn, AQIS, to Barry O'Neil, 6 August 1998).

10.4 Clinical signs

While most infections in horses are without clinical signs, three syndromes are recognised. In the transient type, fever is followed by uneventful recovery in 2-3 days. In the lethargic type there is a fluctuating fever, with mild nervous signs normally followed by recovery in one week. In the hyperexcitable form there is high fever with acute nervous signs, which lead to collapse and death. [4, 5]

The incubation period in horses is 8-10 days, [4] although the OIE Code [6] states that the incubation

period should be considered as 21 days. Clinical signs last 2-9 days. The mortality rate in clinically affected horses is generally about 5% but may be as high as 40% in severe outbreaks. [4]

10.5 Transmission

JE virus is an arbovirus transmitted by mosquitoes. Twenty-eight mosquito species have exhibited competence for infection in either field or laboratory studies. However, only a few species found in endemic areas are sufficiently abundant, have long enough flight ranges, and have the breadth of host feeding preferences to become natural vectors. *Culex tritaeniorhynchus* is an important vector throughout most of Asia. *Cx. vishnui, Cx. fuscocephala, Cx. gelidus, Cx. annulus* and *Cx. annulirostris* are important in the Asian countries where they occur. [3]

The transmission cycle between waterbirds and mosquitoes occurs mainly in tropical rice growing areas, which provide suitable habitat for both. *Cx. tritaeniorhynchus* and *Cx. vishnui* breed predominantly in rice paddies and are therefore the most important rural vectors. Build-up of infected mosquitoes eventually leads to infections in other mammalian hosts. The importance of pigs as food in many tropical rice growing areas of Asia ensures a continual supply of susceptible young pigs. Pigs act as amplifying hosts, developing a viraemia that lasts about a week. Solid immunity then develops and terminates the viraemia.[3]

In temperate areas there is a seasonal pattern of occurrence, with most cases in late summer and autumn. In tropical areas the virus circulates year round. [3, 4]

There is no evidence that JE is naturally transmissible directly from animal to animal. Transmission in pigs has been demonstrated experimentally through semen. Mechanical transmission by biting insects is not known to occur. [5]

Horses and humans are considered dead-end hosts, because a viraemia of sufficient titre to infect mosquitoes does not develop. [3, 4, 5]

10.6 Diagnosis

Serology is the principal means of diagnosis in the live animal. It requires demonstration of a four-fold rise in the titre of specific antibody. Serological tests generally suffer from a lack of specificity, leading to considerable cross-reaction with other flaviviruses. The test with highest specificity is the VN. [2]

10.7 Immunity

In horses, as in most animals and humans, there is a lifelong immunity after natural infection. Passive immunity is passed through colostrum and lasts 2-3 months. [5]

Vaccines are available for use in horses, but will interfere with serological diagnosis.[2]

10.8 Treatment

Treatment is supportive only.

10.9 Risk assessment

10.9.1 Release assessment

The risk that a horse might be infected with JE virus must be considered when horses are imported from Asia and, possibly, northern parts of Australia.

The prevalence of JE in horses in most areas is unknown. Vaccination programmes have reduced the incidence in Japan. In sub-tropical and temperate areas there is a seasonal pattern of occurrence, and the greatest risk occurs during late summer and autumn. The incubation period is short and, while clinical disease is easily recognised, the majority of infections in horses are probably subclinical.

There are no reports suggesting that there is a risk of JE introduction in horse semen.

10.9.2 Exposure risk

Horses do not develop viraemia of sufficient titre to infect mosquitoes, and are considered dead-end hosts. Direct transmission does not occur so there is essentially no risk that importation of an infected horse would lead to further cases in other livestock or humans.

While *Culex* sp. mosquitoes exist in New Zealand, none of those species involved in JE transmission cycles in Asia occur here. The temperate climate and lack of suitable rice paddy environments suggest that there is very little risk of endemic cycles establishing in this country.

10.9.3 Consequence assessment

The consequences of a single imported case would probably be the direct costs associated with disease investigation and indirect costs associated with disruption of trade over the short period until investigation established a diagnosis.

The differential diagnosis for equine encephalitis includes a number of diseases associated with public health risks and significance during international trade. A case in an imported horse would initiate an exotic disease investigation. Exports of live horses and semen could be stopped until a diagnosis was established. If JE were diagnosed in an imported horse, exports would probably resume without further measures being imposed.

Importation of seropositive horses, either as a result of previous infection or vaccination, would have no adverse consequences.

10.9.4 Risk estimate

Horses infected with JE could be imported from endemic areas, and this is more likely during the summer and autumn months when importing from temperate and subtropical zones. JE is not transmitted from infected horses, and so would not establish if infected horses were imported. Clinical cases in imported horses could cause short-term adverse consequences, and measures to reduce the likelihood of such cases are warranted. Imports of horse semen do not present a risk, and so safeguards are not necessary.

10.10 Risk management

10.10.1 Risk management objective

Horses should be protected from infection during the pre-export period, so that at the time of importation no horse is incubating or showing clinical signs of JE.

A minimum period of 21 days in insect-proof pre-export isolation provides a high level of assurance that horses would not be incubating disease at the time of importation. This is not necessary in temperate areas during periods when virus transmission has been demonstrated not to occur.

Vaccination of horses is commonly practised in endemic areas, and in Japan has lead to a large reduction in clinical cases. Vaccination prior to isolation would reduce the likelihood of clinical cases in isolated horses, an event that would disrupt export of all horses in the isolation group.

Premises of origin freedom from JE probably provides little additional benefit, considering infections are commonly subclinical.

10.10.2 Risk management measures

The OIE Code [6] recommends that horses imported from endemic areas be isolated for 21 days in insect proof facilities where no cases of JE have occurred during that period, and show no clinical sign on the day of export. Isolation from swine is also recommended, but the justification for this measure is difficult to understand considering JE will not be transmitted in the absence of insect vectors.

While not recommended by the OIE, vaccination is a readily available measure that would be very effective in reducing the likelihood of clinical cases.

The occurrence of JE in mainland Australia is cause for concern considering the number of horses imported from Australia. AQIS monitors JE virus activity in the islands of Torres Strait and in northern Australia through a programme of sentinel animals. Animal movement within the Torres Strait is controlled. [5] In response to the first human clinical case of JE in Northern Queensland during 1998, the Queensland Department of Health tested 400 human sera from mainland areas considered at greatest risk of incursion, with two positive results. The virus was also isolated from two sentinel pigs (pers. comm. Sarah Kahn, AQIS, to Barry O'Neil, 6 August 1998). These

outbreaks were probably caused by mosquitoes blown from endemic areas of Papua New Guinea, and there is no evidence that the virus has become established on the Australian mainland.[7]

The data from Australia suggest that animal and human health authorities there are conducting surveillance able to monitor the occurrence and spread of JE. The ability of AQIS to describe JE free areas of Australia should be acknowledged. Imports of horses from such areas do not present a risk, and so need not be subject to measures recommended for JE.

Live horses

Either:

1. The horses have been resident since birth, or at least the previous 21 days, in a country that is free of JE.

Or:

1. The horses have been resident since birth, or at least the previous 21 days, in a part of Australia where no cases of JE have occurred.

Or:

1. During importation of horses from countries where JE occurs, the horses were vaccinated against JE with an inactivated vaccine according to the manufacturer's recommendations during the 12 months, but not less than 60 days, prior to export; *and*

2. Either: i) The horses were kept for a minimum 21 days prior to export in a pre-export isolation facility and protected from insect vectors during that period and during transport to the port of export;
Or: ii) The date of export and the 21 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses were showing no clinical signs of JE on the day of export.

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11 Rabies

11.1 Aetiology

Rabies is an OIE List B disease caused by a virus in the genus *Lyssavirus* of the family Rhabdoviridae. Six distinct genetic lineages (serotypes 1-4, European bat lyssavirus 1, and European bat lyssavirus 2) can be distinguished in the genus. Subtypes of true rabies virus (serotype 1) may vary considerably in their pathogenicity. They are also classified on the basis of their origin e.g. canine, vulpine, etc. [1] In 1996 a further lyssavirus was isolated from fruit bats in Australia.[2]

11.2 Susceptibility

The viruses can infect all mammals, including humans, but are maintained in specific endemic cycles in typically carnivorous mammalian species in various countries. [1, 3]

11.3 Distribution

Rabies occurs in Europe, North and South America, Africa and Asia. [4]

Hawaii, Singapore, Norway, Sweden, Britain, Ireland, Australia, Fiji and New Caledonia are considered by MAF to be free of rabies. Rabies has never occurred in New Zealand.

11.4 Clinical signs

The incubation period of rabies is highly variable. The reported range in horses is 11 days to 25 months, but in the majority of cases it is less than 3 months. [3]

After onset of signs the course of disease in horses is very short, ranging from 1 to 7 days. Nervous signs progress and invariably lead to death or euthanasia. [3, 4]

11.5 Transmission

Transmission is by direct inoculation from an infected animal, particularly from bites and scratches. Dogs, foxes, skunks, raccoons and bats are the most frequently recognised source of infection, depending on the local epidemiological situation. [3, 4]

Domestic livestock are rarely a source of infection, although they do excrete virus in the saliva and chance transmission to humans may occur during handling. [3, 4]

11.6 Diagnosis

Diagnosis is made on clinical signs and brain histology. Viral antigen may be detected in fresh brain smears through florescent antibody and immunohistochemical tests. [1]

11.7 Immunity

Live attenuated and inactivated vaccines are available, and vaccination is commonly practised in endemic regions. (1)

In the USA, 10.3% of establishments with horses less than 12 months of age, 20.3% of establishments with brood mares, and 24.5% of establishments with other horses over 12 months of age vaccinate against rabies. (5)

11.8 Treatment

There is no treatment for clinical rabies. Prophylactic protection by vaccination or antiserum may be employed following suspected exposure. (1)

11.9 Risk assessment

11.9.1 Release assessment

The risk of an imported horse being infected with rabies is primarily dependent upon local epidemiological factors such as the prevalence of rabies and the cycles occurring in the exporting country, both of which affect the likelihood of pre-export exposure. While the clinical signs are obvious, the often long incubation period means horses could be imported prior to developing clinical signs.

No evidence for transmission in horse semen has been reported.

11.9.2 Exposure assessment

Exposure of humans handling imported horses presents the only probable exposure pathway.

11.9.3 Consequence assessment

The principal concern if a horse were to be imported and subsequently found to have rabies would be the public health consequences. Follow-up actions might include trace-back and prophylactic vaccination of potentially exposed persons.

The differential diagnosis for equine encephalitis includes a number of diseases associated with public health risks and international trade significance. A case of encephalitis in an imported horse would initiate an exotic disease investigation, and exports of live horses and semen could be stopped until a diagnosis was established.

11.9.4 Risk estimate

Horses imported from endemic areas could be incubating the disease. The consequences of an imported case would probably be confined to persons exposed to imported animals. Some

consequences associated with the disease investigation could also be expected. Measures during importation of live horses are warranted. Semen does not present a risk, and so no safeguards are necessary.

11.10 Risk management

11.10.1 Risk management objective

Horses should be protected from exposure to rabies during the pre-export period. This can be achieved by requiring horses to have resided in a country, zone or establishment where rabies has not occurred during the pre-export period.

11.10.2 Risk management measures

The OIE Code [6] recommends that imported horses should have been resident since birth or at least the 6 months prior to export in a country or establishment where no case was reported for at least the 12 months prior to export. These measures are consistent with the objective above. Accounting for very rare exceptionally long incubation periods is probably not feasible or necessary.

Surveillance systems to detect and report cases of disease will increase the value of safeguards which rely on the occurrence and distribution of cases. Most countries conduct monitoring and surveillance for human, domestic animal, and wildlife cases of rabies, and the World Health Organisation reports the situation in individual countries.

Vaccination undoubtedly provides an important means to protect humans and animals from rabies within endemic areas. As a zoosanitary measure during trade, it is applied during imports of potential reservoir carnivore hosts. Vaccinating imported domestic livestock such as cattle and horses, which are unlikely to lead to introduction and establishment, is not considered necessary. However, vaccination should be considered for persons with regular occupational exposure to such animals.

Live horses

1. The horses were kept since birth or for the 6 months prior to export in an establishment where no case of rabies was reported for at least the 12 months prior to export; *and*

2. The horses were showing no clinical sign of rabies on the day of export.

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12 Borna disease

12.1 Aetiology

Borna disease (BD) is caused by an RNA virus which has been placed in a separate family Bornaviridae within the order Mononegavirales. [1]

12.2 Susceptibility

BD occurs naturally in sheep, horses, cattle, cats, ostriches and possibly humans. The range of susceptible animals is broad, extending from chickens to primates. [1, 2, 3]

The zoonotic potential of BD is uncertain. [1, 2]

12.3 Distribution

There are areas in Germany, Switzerland and Austria where BD is endemic. [1, 2, 3, 4, 5, 6] Approximately 11.5% of horses in Germany may be seropositive. [3] Approximately 2% of clinically normal horses [5] and 4% of horses exhibiting nervous signs [6] in Austria may be seropositive. There were 21 reported cases of BD in horses in Switzerland between 1990-1995. [7] There is a seasonal fluctuation in cases, with a maximum in spring and early summer and a sharp decline in winter. [1]

BD virus occurs in the Swedish racing horse population and in Swedish cats. [8] BD virus has been recorded in cats with neurological disorders in Britain. [9]

Aside from the countries already mentioned, serological evidence of BD virus infection has been found in several other countries. The implications of serological findings on the actual distribution of BD virus are uncertain.

BD has not been recorded in New Zealand.

12.4 Clinical signs

BD in horses typically occurs as sporadic cases, with in-contact horses frequently found to be seropositive and free from clinical signs. [1]

The incubation period is highly variable, ranging from a few days to more than 12 months, but averaging 4 weeks. The disease lasts for 3 to 20 days and is usually fatal. [1]

BD is a slowly progressive polio-encephalomyelitis. A multitude of signs is observed and can typically be classified as depression and excitation, central sensory disturbances and motor disorders. [1] The mortality rate varies from 37-94%. [2] Recovered horses often have permanent sensory or motor disturbances. [1]

12.5 Transmission

The mode of transmission is uncertain. BD virus appears not to be readily transmitted beyond the endemic areas. No reservoir has been identified, nor has vertical transmission been demonstrated. [1, 2]

Viral RNA can be detected in conjunctival fluid, nasal secretions and saliva of seropositive healthy horses, which may act as virus reservoirs [1] and spread infection through direct contact or fomites and food. [2]

Based on the seasonal fluctuation of cases, arthropod vectors are suspected to play a role. [1, 2]

12.6 Diagnosis

Laboratory confirmation is required. Ante-mortem serology using IFT or ELISA will indicate previous exposure. CSF is also a reliable source of antibodies. Not all seropositive animals develop disease and not all diseased animals are seropositive. A study of 23 cases found 61% had serum antibodies. [1] Various viral antigen detection techniques are available for post-mortem diagnosis. [1, 10]

12.7 Immunity

There is no vaccine available. [1, 2]

12.8 Treatment.

None.

12.9 Risk assessment

12.9.1 Release assessment

Although clinical BD occurs in horses with low sporadic incidence and is largely confined to an endemic area in Central Europe, seroprevalence studies indicate infection occurs more frequently and more widely. The implications of results from serological surveys are uncertain. Both clinical cases and subclinically infected horses may present a risk of introducing BD.

Horses imported from endemic areas may be infected with BD virus, and the seasonal fluctuation of cases indicates a higher risk period during the spring and summer. Unrestricted movement of horses out of endemic areas, and the possibility that BD virus distribution is not properly defined, suggest that imports of horses from non-endemic areas may also present some risk.

The long incubation period and high percentage of subclinical infections suggest there is a risk of apparently healthy infected horses being imported.

No evidence has been found indicating that BD virus occurs in semen or is transmitted by artificial insemination.

12.9.2 Exposure assessment

The mode of transmission remains poorly understood. It appears BD virus is not readily transmitted outside the endemic areas because clinical cases in horses remain largely confined to these areas, despite no BD-specific control measures for movements of horses out of this area. This suggests that imports of infected horses would not lead to further cases here. However, healthy inapparent carriers may be important in spreading BD virus, and the postulated horizontal spread by direct contact to horses or sheep may pose a risk.

12.9.3 Consequence assessment

The consequences of an imported case of BD are difficult to assess. A single imported case would have direct effects associated with the disease investigation. The investigation would need to establish through trace-back and testing of in-contact animals whether transmission had occurred. If the animal had been imported some months previously (as is possible with diseases with long incubation periods), trace-back could be difficult. Active surveillance might be necessary in order to re-establish New Zealand's status as free from BD.

There is unlikely to be a significant response from trading partners to an imported case. Exports of horses from the known endemic areas occur without undue restrictions.

The public health implications of BD are uncertain. Persons involved in any investigation into an imported case would be obliged to take appropriate precautions considering the differential diagnosis for equine encephalitic disease.

12.9.4 Risk estimate

Uncertainties surrounding BD epidemiology make estimating the level of risk associated with imports difficult. While clinical cases appear confined to an endemic zone in central Europe, serologically positive animals are much more widespread and the true distribution of BD virus is unknown. Imports of horses from the known endemic area may lead to imported clinical cases, but whether spread to other animals would occur is uncertain. While a single imported case is unlikely to have significant impacts on the equine industries, the potential for spread and the full extent of public health implications are uncertain. Given these uncertainties, safeguards for live horses are probably warranted. In all likelihood semen does not present a risk, and so no safeguards are considered necessary.

12.10 Risk management

12.10.1 Risk management objective

Horses should be protected from exposure to infection in the pre-export period in order to minimise

the risk of importing clinical cases.

The only practical means to achieve this is to exclude horses recently resident within an area where clinical cases are known to have occurred. The seasonal occurrence of BD suggests that the status of an area should be defined by the occurrence of any case during the previous 12 months. Horses resident in free areas for a minimum of 3 months prior to export are unlikely to be incubating clinical disease.

There are no practical and reliable diagnostic tests for determining that a live animal is infectious or not. The present uncertainty regarding matching serological status with infective status suggests that serology should not be used as the basis for import decisions.

12.10.2 Risk management measures

The endemic area in Central Europe includes Switzerland, Germany, Lichenstein and Austria. Sweden should probably also be included in this group. Surveillance will allow more precise definition of endemic zones within these countries, and the prohibition should be applied at this local level to avoid unfairly discriminating against all horses in the countries overlapping the endemic area.

Live horses

1. The horses were resident during the 3 months prior to export in an area where no case of BD has occurred during the previous 12 months; *and*

2. The horses were showing no clinical sign of BD on the day of export.

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13 Equine encephalosis

13.1 Aetiology

Equine encephalosis (EE) is caused by an orbivirus in the family Reoviridae.[1] There are seven serotypes, all distinct from AHS virus.[2]

13.2 Susceptibility

Clinical disease occurs only in horses, although serological evidence suggests that some serotypes infect zebras and donkeys. All ages and sexes of horses are susceptible to infection, but clinical disease is more severe and mortality rates higher in weanlings and horses older than 7 years. [1]

13.3 Distribution

The disease has only been reported in South Africa and Botswana, although it probably also occurs in other parts of Africa. Outbreaks in South Africa occur irregularly at intervals of 2 or more years. At other times there is a sporadic incidence. There is a summer seasonal pattern of disease occurrence from December to June.[1, 2] The most recent outbreak of clinical disease in South African horses occurred in April 1999. [3]

Surveys have demonstrated a high prevalence of antibodies to EE in horses and donkeys distributed over the whole of South Africa, including the AHS free area. The highest prevalences (up to 73.2%), were in the warmer northern areas and the Eastern Cape had the lowest prevalence (28.4%).[4] Testing of 604 horse sera sent to Onderstepoort Veterinary Institute for different diagnostic purposes between 1994 and 1997 indicated that 75% of samples were seropositive, with provincial rates varying from 53.3% in Western Cape to 100% in Northern Province. [2]

EE has never been recorded in New Zealand.

13.4 Clinical signs

The vast majority of infections are subclinical. [2] Clinical syndromes associated with EE include acute death, an AHS-like syndrome, abortion and depression, and an inappetance, fever and jaundice complex. [2]

The incubation period is 2-6 days. A fluctuating fever lasts 1-5 days, with associated listlessness and inappetance. In severe cases this progresses to nervous signs, circulatory failure and death. The mortality rate is less than 5 %. [1]

13.5 Transmission

EE virus is an arbovirus transmitted by *Culicoides* midges. Although *C. imicola* is assumed to be a major vector,[1] EE is more widely distributed than *C. imicola*, suggesting that other species are involved in transmission. [2]

Koch's postulates have not been fulfilled for EE, despite experimental inoculation of horses, indicating EE virus may require other factors to cause disease. (2)

13.6 Diagnosis

Definitive diagnosis is by virus isolation on cell cultures or by intracerebral inoculation of mice. The SN test is used to make a positive identification. (1)

CFT, SN and indirect ELISA are used for serological diagnosis. All serotypes cross react on the CFT, but not with other orbiviruses (AHS, bluetongue and epizootic haemorrhagic disease of deer). On the SN test the serotypes do not cross-react. The indirect ELISA is used most regularly by Onderstepoort Veterinary Institute. (2)

13.7 Immunity

Previously infected horses are immune to re-infection with the same serotype, and may be partially immune to heterologous serotypes. Convalescent sera of infected horses have high titres of neutralising antibody for at least 1 year. (1)

No vaccines are available.

13.8 Treatment

Treatment is supportive only.

13.9 Risk assessment

13.9.1 Release assessment

Horses imported from Africa may be infected with EE. In South Africa the risk is probably confined to the summer months when transmission occurs.

The incubation period of clinical EE is short, and the clinical signs may be severe and obvious. However, the prevalence of subclinical infection is high.

While no information has been found regarding the infectious period, knowledge of other orbiviruses such as AHS and bluetongue suggest it is probably no longer than 28 days, and the immune response clears the viraemia.

No evidence has been found suggesting transmission by artificial insemination.

13.9.2 Exposure assessment

EE virus is spread by *Culicoides* midges, which do not occur here. A case of EE in an imported

horse would not lead to further cases in New Zealand.

13.9.3 Consequence assessment

The consequences of a single imported case of EE would probably be limited to short-term direct consequences associated with the disease investigation. Some indirect consequences associated with stopping exports of horses and semen until a diagnosis was established could occur. Consequences would be confined to the equine industries and would probably not continue after a definitive diagnosis was established.

13.9.4 Risk estimate

Horses infected with EE could be imported from Africa, but EE would not spread or establish in New Zealand. There could be short-term consequences affecting the equine industries associated with an imported clinical case. Measures to ensure infected horses are not imported are warranted. Semen probably does not present a risk, and so no specific safeguards are warranted.

13.10 Risk management

13.10.1 Risk management objective

Horses should be protected from infection during the pre-export period, and be tested to ensure that they have not been recently infected.

Isolation in arthropod-proof facilities would provide a high level of assurance that incubating horses will not be imported. An isolation period of at least 28 days should ensure that any horse infected prior to entering isolation is not infective at the time of export. Protection from infection may also be achieved if a seasonal period during which virus transmission by *Culicoides* vectors has been demonstrated not to occur can be defined.

Requiring all horses to demonstrate negative, stable or declining antibody titres during the pre-export period would provide further assurance that imported horses have not been recently infected. The CFT or indirect ELISA should be considered acceptable tests. Seropositive animals may remain susceptible to infection with heterologous serotypes of EE virus, however, and so pre-export isolation remains necessary.

13.10.2 Risk management measures

Live horses

Either:

1. The horses were resident since birth, or at least the previous 28 days, in a country that is free of EE.

Or:

1. EE occurs in the exporting country; *and*

2. Either: i) The horses were kept in a pre-export isolation facility for the 28 days prior to export, and protected from arthropod vectors during this period and during movement to the port of export;
Or: ii) The date of export and the 28 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses were subjected to the indirect ELISA or CFT for EE during 28 days prior to export on two occasions at least 21 days apart, and demonstrated a negative, stable or declining titre; *and*

4. The horses were showing no clinical sign of EE on the day of export.

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14 Louping ill

14.1 Aetiology

Louping ill virus is a member of the family Flaviviridae, genus Flavivirus. It is one of a serocomplex of 14 related tick-borne viruses given the term tick-borne encephalitis complex.[1, 2, 3]

14.2 Susceptibility

The disease mainly affects sheep. Humans, cattle, horses, pigs, deer, rodents and some bird and wild vertebrate species are also susceptible to infection.[1, 2, 3]

14.3 Distribution

Louping ill mainly occurs in Scotland, Northern England, Wales and Ireland. Endemic areas are also found in parts of Scandinavia and Europe. [2, 3] The disease is seasonal, with occurrence in spring through to summer and autumn, when ticks are active. [2, 3]

14.4 Clinical signs

Natural infections have been reported in horses, but the low seroprevalence suggests these are uncommon. Viraemia lasts for 1-4 days following infection. Progression to encephalitis is rare. [1]

In sheep the incubation period is 2-5 days. Viraemic titres peak in 2-4 days, with a corresponding fever, and decline with the development of circulating antibodies at 5-10 days. Nervous signs develop 6-8 days after infection, accompanied by a second temperature peak. Most infections in sheep are subclinical, and seroprevalence may be high in endemic areas.[2, 3]

14.5 Transmission

Louping ill is transmitted by the three-host sheep tick *Ixodes ricinus*. Only transstadial transmission occurs naturally. [2, 3]

Although experimental infection of horses has occasionally produced viraemia of sufficient magnitude to infect ticks, [3] natural infections of species other than sheep are not thought to lead to transmission to tick larvae or nymphs.[1, 3]

14.6 Diagnosis

Viraemic blood samples can be inoculated onto cell culture, with identification of isolates by SN tests. HI, CFT and ELISA are available for serological diagnosis. [1, 3]

14.7 Immunity

An antibody response detectable by the HI test occurs 5-10 days after infection. Immunity following infection is life-long. [2, 3]

Killed vaccines are widely used in sheep in endemic areas. [2, 3]

14.8 Treatment

Early cases may be treated with antiserum. [2]

14.9 Risk assessment

14.9.1 Release assessment

Horses could be infected with louping ill virus when imported from endemic areas during the summer and autumn transmission period. Seroprevalence in horses is low, and clinical disease is rare. The incubation period is short, and the clinical signs of encephalitis obvious. All of these factors suggest that the risk of healthy horses being infected when imported is very low.

Importation of horse semen does not present a risk.

14.9.2 Exposure assessment

Natural infections of horses probably do not produce viraemia of sufficient titre to infect ticks. *Ixodes ricinus* does not occur here. Transmission in New Zealand would not occur.

14.9.3 Consequence assessment

The only consequences associated with an imported horses subsequently developing encephalitis are likely to be those associated with the disease investigation, as discussed in the context of other encephalitic diseases. Transmission in New Zealand would not occur, so there would be no adverse consequences for the sheep industries.

14.9.4 Risk estimate

On rare occasions horses imported from endemic areas could be infected with louping ill, but louping ill would not be transmitted or establish in New Zealand. Infections that progress to clinical signs of encephalitis after importation would result in disease investigation to rule out other exotic equine encephalitides. However, clinical cases in horses are also rare. Horse semen does not present a risk.

14.10 Risk management

14.10.1 Risk management objective

Horses that are clinically affected with louping ill should not be imported. The low risk of infection, low risk of infection leading to clinical disease, and short incubation period all suggest a period of protection from infection prior to export is unnecessary.

14.10.2 Risk management measures

The risks associated with louping ill may be managed by requiring horses to be free from clinical signs of infectious or contagious disease on the day of export and during any period of preparation for export. Specific measures are not warranted.

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15 Hendra virus and Nipah virus

15.1 Aetiology

Equine morbillivirus pneumonia (EMP) was the term originally used to describe an acute equine respiratory syndrome which first occurred in Queensland, Australia, in 1994. [1, 2] The virus is now known as Hendra virus (HeV) and appears to represent a bridging genus between the Morbillivirus and Paramyxovirus genera. [3]

In 1999 a new paramyxovirus, now known as Nipah virus, was isolated during an outbreak of viral encephalitis in pigs and humans in Malaysia and Singapore. [4] Molecular studies show difference of 21% in the nucleotide sequence and 11% in the animo acid sequence to the HeV.[5] This suggests that Nipah virus is not a mutation of HeV, and it seems probable that the two related viruses have existed separately for some time. [6]

15.2 Susceptibility

The natural hosts of HeV are probably fruit bats (*Pteropus* spp., also called flying foxes).[6] Serological reactions were found in these species during epidemiological investigations following outbreaks of disease in horses and humans. [7, 8] Horses and humans are naturally susceptible, as demonstrated by the three outbreaks of disease which have probably resulted from spillover from the natural host. [6] Experimental infection of horses, cats and guinea pigs caused disease, whereas subclinical infection was demonstrated in fruit bats and rabbits.[9]

Nipah virus infection causes disease in humans, pigs and dogs. Approximately 100 persons have died in the 1998-99 outbreak in Malaysia out of 258 suspected infections. Cats, horses and flying foxes in the outbreak areas have been found with serological reactions. [5]

15.3 Distribution

EMP has occurred as three outbreaks in Queensland, Australia: two in 1994 and one in 1999.[3, 6, 7, 10, 11] One of the 1994 outbreaks was diagnosed retrospectively. [12]
Pteropus spp. fruit bats have a wide distribution around much of coastal Australia and throughout South East Asia. [8] If fruit bats are the natural reservoir, the actual distribution of HeV may be greater than suggested by outbreaks to date.

Subsequent to the 1994 outbreaks, serological surveillance was undertaken in Queensland. This has included targeting horses on properties in the location of the outbreak, properties where unexplained respiratory and neurological disease has been reported, and properties where horses have contact with wildlife. A total of 4,726 horses were sampled during this surveillance, all with negative results for HeV antibodies. [6]

Outbreaks of Nipah virus have occurred in pigs in five states of peninsular Malaysia; Perak, Negeri Sembilan, Johor, Malacca and Kelantan.[5, 13] Human cases in Singapore are thought to have been associated with pig meat products imported from Malaysia.

HeV and Nipah virus have not been recorded in New Zealand, nor anywhere else other than discussed above.

15.4 Clinical signs

The clinical signs during outbreaks of HeV in horses were those of acute respiratory disease. The incubation period varied between 5 and 8 days, and was followed by anorexia, depression and a high fever, with rapid progression to acute respiratory signs and eventual death with copious frothy nasal discharge. Of 14 horses infected during one of the 1994 outbreaks, seven died, four recovered, and three apparently experienced subclinical infection. The seven seropositive horses were destroyed. Only three horses were involved in the other 1994 outbreak and that of 1999, and all three died. [6]

Three humans infected with HeV developed a serious influenza-like illness and progressive encephalitis. One person died during the acute illness, one person recovered and remains healthy, [2, 10] and the other apparently recovered from an acute illness but died from progressive encephalitis approximately 1 year subsequently. [7]

Experimental infection of four horses with HeV produced clinical signs in three horses within 11 days. Signs were consistent with those seen during outbreaks. [14]

It is not known whether two horses found to be seropositive to Nipah virus exhibited clinical signs at the time of infection. Infections in pigs caused mild to severe respiratory signs, including a "one mile cough" (so called because it could be heard from a long distance), followed by convulsions and other neurological signs in some animals. Mortality was low but morbidity high. Infected humans developed from mild to severe clinical signs. Severe cases progressed from disorientation to coma and death. Some humans showed no apparent clinical symptoms. [5]

15.5 Transmission

Transmission of HeV is assumed to be direct and require close contact. Transmission studies involving fruit bats, cats and horses suggest that the virus is not highly contagious. Transmission to horses did not occur when they were in contact with inoculated horses and fruit bats, but did occur in one of three horses in contact with an inoculated cat. Transmission from horses to cats could not be demonstrated. Horses could be infected by the oronasal route, and excreted HeV in urine and saliva. [14]

In one of the 1994 outbreaks 14 horses in contact with infected horses did not become infected. [3]

Transmission of Nipah virus in pigs has been demonstrated by oral and parenteral inoculation, and in-contact pigs also became infected. Virus multiplication in tonsils and respiratory epithelium suggests transmission by pharyngeal and bronchial secretions. [5]

15.6 Diagnosis

HeV infection may be diagnosed by virus isolation, PCR or indirect immunoperoxidase staining on post-mortem specimens. Serological diagnostic techniques include SNT and ELISA. [1, 6] The ELISA test has relatively low specificity, and this has caused some problems during export of horses to countries that have adopted it as a pre-export screening test in response to the events described above.

Little is known about the serological response to HeV infection. One horse that recovered from clinical signs was found to be seropositive to SNT with virus present in the spleen when euthanased at 21 days following experimental intranasal inoculation. [14]

Nipah virus infection is diagnosed by virus isolation, or by serology using IgG or IgM capture ELISA or SNT. [5]

15.7 Immunity

Unknown.

15.8 Treatment

None.

15.9 Risk assessment

15.9.1 Release assessment

Horses imported from Queensland could be infected with HeV, but the incidence of outbreaks and the absence of any detectable seroprevalence in the general equine population suggest a very low risk. The clinical manifestation of HeV when transmitted from its wildlife reservoir to horses or humans is typically acute and severe, although subclinical infections in horses have also been recorded. Natural transmission to horses appears to be a very rare.

The incubation period of HeV in horses is probably no more than 11 days. Virus is recoverable from urine, saliva and tissues for at least 21 days, but transmission from horses to to other animals (including other horses) has not been demonstrated.

Horses imported from Malaysia could possibly be infected with Nipah virus. Serological surveillance in Malaysia suggests the likelihood of such an event is very low. Little, if anything, is known about clinical signs, incubation period and infectivity in horses.

As more becomes known about HeV and Nipah virus epidemiology, the endemic area for these viruses may need to be considered as the range of the reservoir, fruit bats, potentially encompassing other areas in Australia and South East Asia.

Transmission of HeV or Nipah virus via semen has not been investigated, although the likelihood of a stallion being infected, clinically healthy and having semen collected for export must be considered remote.

15.9.2 Exposure assessment

Transmission of HeV from horses to other animals has not been experimentally demonstrated. During one outbreak some horses in contact with infected horses did not become infected. Transmission from horses to humans in close association with them probably occurred during outbreaks, based upon epidemiological evidence, and presents a public health risk for animal handlers.

While much remains unknown, experience in Australia and the absence of fruit bats from New Zealand suggest that while an imported case might lead to a limited outbreak here, potentially involving human cases, establishment of HeV is very unlikely.

There is insufficient information to draw conclusions on whether a horse infected with Nipah virus would lead to transmission and establishment here. The absence of epidemiological evidence for the involvement of horses in transmission cycles in Malaysia and the absence of fruit bats here again suggest this is unlikely.

15.9.3 Consequence assessment

Any imported case or outbreak of HeV or Nipah virus in New Zealand would result in an investigation involving animal and public health authorities. The investigation would focus on the infected premises with trace-back of in-contact horses and humans. This would involve short-term direct consequences for the affected persons and properties. Public health consequences could be severe, as both viruses cause fatal zoonotic infections.

An imported case is likely to result in immediate reaction from trading partners. In response to the Australian and Malaysian outbreaks there was a suspension of trade in live horses, with gradual re-establishment of exports under additional testing measures.

15.9.4 Risk estimate

The low incidence of disease outbreaks and the low seroprevalence in horses both suggest the likelihood of horses infected with HeV being imported from Australia is very low. The disease incidence of Nipah virus in Malaysia is much higher, but only two horses are known to have been infected. Fruit bats do not occur here, so HeV or Nipah virus would probably not establish if an infected horse were imported, although there could be an outbreak of disease involving horses and humans in contact with the imported animal. The consequences of importing clinical cases could be severe, and would include the disease investigation, public health consequences, and trade measures for horse exports. Measures to reduce the likelihood of infected horses being imported are warranted. Semen probably does not present a risk.

15.10 Risk management

15.10.1 Risk management objective

Horses clinically infected or incubating HeV and Nipah virus should not be imported. Horses should be protected from infection in the pre-export period.

The short incubation period and obvious and acute clinical signs associated with HeV infection in most horses make clinical freedom the most important safeguard.

The serological response to HeV has not been well defined, although limited data suggest that by the time a detectable titre of antibody is produced the horse will probably have manifest acute respiratory disease. Persistence of infection and the possibility of the carrier state require further investigation. The benefit of serological testing in the absence of clinical signs would probably only be to ensure that recovered or subclinically infected horses were not imported.

HeV titres and tissue distribution during the acute phase of clinical disease were much greater than those recorded in a horse that had recovered from clinical disease. Virus was not recovered from horses that had been subclinically infected. Transmission from horses with acute disease to other animals could not be demonstrated.[14] This suggests that the likelihood of subclinically infected or recovered horses transmitting infection is probably negligible, and that safeguards aimed at preventing any such event (i.e serological testing) are unwarranted.

Similar investigations into Nipah virus have not yet been undertaken in horses. Considering the greater distribution and incidence of infection in Malaysia, serological testing of horses for Nipah virus is probably warranted. Either the IgG or IgM capture ELISA or SNT should be considered acceptable.

Protection from infection in the pre-export period could be achieved by measures to restrict the area or premises from which imports are permitted, or by requiring horses to undergo a period of pre-export isolation. The outbreak areas appear to be confined to particular states in the affected countries, although the environmental factors that contribute to outbreaks have not been well-defined. The presence of a reservoir host, suggested to be fruit bats, may be a contributing factor. Excluding access by fruit bats to horses being prepared for export could reduce the risk of infection in the pre-export period.

Outbreaks of disease in horses are rare and acute events that receive widespread publicity, international attention, and investigation by animal and public health authorities in Australia and Malaysia. Infection with HeV is notifiable in all states and territories of Australia (pers. comm. Tim Buick, AQIS, 22 June 1999). The control and eradication programme for Nipah virus during the 1998-1999 outbreak has involved mass culling of infected pigs and widespread surveillance in many species. [5]

So long as HeV and Nipah virus remain notifiable and there is a swift response to disease outbreaks, a premises of origin disease freedom statement covering the 3 month period prior to

export provides the most practical option to achieve protection from infection in the pre-export period.

15.10.2 Risk management measures

Live horses

Either:

1. The horses were resident since birth, or at least the previous 3 months, in a country that is free of HeV and Nipah virus.

Or:

1. The horses were imported from Australia, where infection of horses with HeV is a notifiable disease; *and*

2. During the 3 months prior to export the horses were kept on premises where infection of horses with HeV has not occurred during that period; *and*

3. The horses were showing no clinical signs of infection with HeV on the day of export.

Or:

1. The horses were imported from Malaysia, where infection of horses with Nipah virus is a notifiable disease; *and*

2. During the 3 months prior to export the horses were kept on premises where infection of horses with Nipah virus has not occurred during that period; *and*

3. During the 30 days prior to export the horses were tested for Nipah virus using either the IgG or IgM capture ELISA or SNT, with negative results; *and*

4. The horses were showing no clinical signs of infection with Nipah virus on the day of export.

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16 Getah and Ross River viruses

16.1 Aetiology

Getah virus (GV) and Ross River virus (RRV) are closely related arboviruses that are members of the Semliki Forest antigenic complex of the Alphavirus genus within the family Togaviridae. [1, 2, 3, 4, 5, 6]

16.2 Susceptibility

GV causes disease in horses and, possibly, neonatal pigs. Subclinical infection occurs in humans, monkeys, cattle, water buffaloes, goats, dogs, rabbits, domestic fowl and night herons. [1]

RRV causes disease in humans and laboratory mice. Serological evidence indicates RRV causes clinical disease in horses, although virological confirmation is lacking. Subclinical infection with RRV occurs in a wide range of marsupials, mammals and birds. [4, 5]

16.3 Distribution

GV caused epidemics in horses in Japan in 1978, 1979 and 1983. Serological evidence indicates it is distributed throughout North East and South East Asia. [1, 2, 3, 6] Virus has been isolated from mosquitoes in Australia and antibodies detected in humans and cattle there during the 1960s, [6] but it has not been detected since and is considered exotic to Australia.[1] An outbreak has been described in thoroughbred horses in India, and a survey indicated seroprevalence in the group of 17%. [7]

RRV occurs throughout Australia, including Tasmania, and also in Papua New Guinea, the Solomon Islands, New Caledonia, Fiji, American Samoa and the Cook Islands. [4, 5]

16.4 Clinical signs

Most infections of horses with both GV and RRV are subclinical. Clinical signs of infection in horses with GV include a biphasic fever lasting 1-4 days, with debility and inappetance. There may be a papular skin rash on the neck, chest and thighs, and oedema of the limbs. Recovery occurs within a week, and mortality is low. [1] Clinical signs in experimentally infected horses developed within 2-3 days, and there was complete recovery in most horses within a week. [6]

RRV may be associated with a condition in horses involving muscle and joint stiffness, limb oedema and nervous signs. Experimental inoculation of horses has only resulted in a very mild clinical syndrome. [4]

16.5 Transmission

Both GV and RRV are transmitted by mosquitoes. *Culex tritaeniorynchus* is the main vector of GV in tropical areas and *Aedes vexans nipponi* in cooler northern climates. [6, 8] Many other

species of mosquitoes have been demonstrated to be competent laboratory vectors. There are similarities between the epidemiology of Japanese encephalitis and GV. In tropical rice growing areas the natural transmission cycle of GV is probably between mosquitoes and pigs.[6] In areas where pigs are not present in large numbers, GV may be maintained in cycles involving horses and mosquitoes.[6, 7] Viraemia has been detected for 1-5 days after experimental infection of horses.[8] Epidemics in Japan occur between July and September, the period of peak mosquito vector activity.[6]

GV is also excreted from the nasal mucosa and horses have been shown to be susceptible to experimental intranasal infection. This suggests direct aerosol transmission may occur, although it is not thought to be significant in natural cycles.[6]

A wide variety of *Culex* and *Aedes* spp. is capable of transmitting RRV, although efficiency varies considerably. The role horses play in RRV transmission has been investigated through experimental inoculation of 11 horses. Viraemia in ten was low or undetectable, and in one was relatively high and lasted 5 days.[4]

16.6 Diagnosis

Diagnosis of GV is by intracerebral inoculation of whole blood or serum into suckling mice, or by virus isolation on cell cultures. Identification of isolates is by VN tests.[1] Serological diagnosis is by HI, CFT, VN or ELISA, although cross-reactivity among alphaviruses occurs.[8]

RRV in human patients can be diagnosed serologically by HI, but the antibody response in horses is unreliable. Virus isolation has no role in routine diagnosis.[4]

16.7 Immunity

VN antibodies to GV appeared within 2 weeks of experimental infection[8] and within 2-4 days of clinical signs in an outbreak in horses.[7] How long such antibodies remain and whether they prevent viraemia on re-infection is not known. An inactivated GV vaccine has been used in racehorses in Japan, with some apparent success.[8]

6.8 Treatment

There is no specific treatment.

16.9 Risk assessment

16.9.1 Release assessment

The risk of horses being infected with GV must be considered when importing from Asia. Although isolated once in Australia, GV infection and clinical disease in horses has not been reported there. High seroprevalence in some Asian countries indicates infection is common and widespread. Most infections are probably subclinical. Clinical disease in horses has only been reported in Japan and

India. Epidemics in Japan follow a seasonal pattern related to climatic effect on activity of mosquito vectors. The incubation period is very short, probably only a few days, and horses probably remain infectious for a short period only. As with other Alphavirus infections, a sterile immunity probably follows natural infection, although this has not been proven.

The risk of horses being infected with RRV must be considered when importing from Australia and the South Pacific. While seroprevalence studies indicate widespread and common infection in humans, the prevalence in horses is unknown. Limited experimental data indicate a low percentage of horses develop significant viraemia, and that it is of short duration in those few that do. The relationship between serological antibody response and viraemia is uncertain.

No evidence has been found regarding GV and RRV presence in semen or transmission by artificial insemination.

16.9.2 Exposure assessment

Neither of the two most important mosquito vector species for GV are present here, although the vector potential of endemic mosquitoes is uncertain. New Zealand's temperate climate is similar to Japan's, suggesting that climate would not be a barrier to virus establishment. The possibility of direct transmission of GV suggests the potential for limited spread on infected premises. However, the likelihood of GV establishing endemic cycles here is probably remote given the absence of the most important vector species.

Limited data suggest that RRV viraemic titres capable of re-infecting mosquitoes are rare and short-lived in horses. A relatively high volume of horse imports (approximately 500 per year) from Australia has not led to cases of RRV or GV here. Neither have human movements, despite these being suspected to have lead to epidemics of RRV in the Pacific Islands. [(4)]

Three of the introduced mosquito species present in New Zealand can be infected with RRV but probably do not transmit infection efficiently. A fourth recently introduced species, *Aedes camptorhynchus*, is an efficient vector but probably has a limited distribution and may soon be eradicated.

16.9.3 Consequence assessment

The consequences of imported cases of GV, or an outbreak on premises following importing infected horses, would probably be confined to the disease investigation and any subsequent attempts at control. Outbreaks of GV in Japan have caused significant disruption in the racing industry, mainly as a result of control measures. The export trade in horses and semen would probably not be significantly affected, as few countries impose restrictions on horse movements for GV.

The presence of RRV in Australia has not led any country to impose restrictions on the importation of horses from that country. The mild clinical syndrome in horses does not cause significant impacts within the Australian equine industries. The consequences of imported cases of RRV in horses

would probably be related only to its zoonotic potential. Given that RRV is just as likely to infect humans arriving from Australia, and given that the number of humans far exceeds the number of horses imported annually, even this potential zoonotic risk must be considered minor.

16.9.4 Risk estimate

Horses infected with Getah group viruses could be imported from Asia, Australia and the South Pacific. Subclinical infection of horses with GV is common, but outbreaks of disease appear rare. Importing subclinically infected horses would probably not result in adverse consequences, because transmission to other animals would be unlikely. Given the short incubation period and course of disease, the risk of importing clinical cases of GV is probably only significant when importing from areas experiencing epidemics. The likelihood of spread from an imported case is probably low, and establishment of endemic cycles even lower. The consequences would mainly be associated with the disease investigation and any control measures. Measures are warranted to ensure horses clinically infected with GV are not imported.

Importing horses infected with RRV is very unlikely to lead to further cases here, and a single imported case is unlikely to have significant consequences. Given the history of importations, the lack of significance of horses in RRV epidemiology, and higher volume trans-Tasman human movements (that probably present a greater risk), measures for RRV during imports of horses are not warranted.

In neither case is semen likely to present a risk, so no measures are considered necessary.

16.10 Risk management

16.10.1 Risk management objectives

Horses should be free from clinical signs of GV at the time of export, and have been protected from infection during the pre-export period.

The risk is most significant when importing horses from areas at risk of epidemics. In such cases, a 14 day period of pre-export isolation in insect vector proof facilities will provide appropriate protection, but is probably not necessary during seasonal periods in which virus transmission has been demonstrated not to occur. Premises of origin freedom from cases will provide further protection.

16.10.2 Risk management measures

Live horses

Either:

1. The horses were resident since birth, or at least the previous 21 days, in a country in which clinical cases of Getah virus have not occurred during the 12 months prior to export.

Or:

1. The horses were kept for the 21 days prior to export on premises where GV disease has not occurred during that period;

2. Either: i) The horses were kept for a minimum 14 days prior to export in an insect-proof pre-export isolation facility, and were protected from insect vectors during transport from the pre-export isolation facility to the port of export;
Or: ii) The date of export and the 14 days immediately prior were during an approved period when virus transmission has been demonstrated not to occur as a result of seasonal climatic influence on vector activity in the area where the horses were resident and prepared for export; *and*

3. The horses showed no clinical signs of GV on the day of export.

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6.2 BACTERIAL DISEASES

17 Anthrax

17.1 Aetiology

Anthrax is an OIE List B disease caused by the spore-forming, Gram-positive, rod-shaped bacterium *Bacillus anthracis.* [1, 2, 3]

17.2 Susceptibility

All warm-blooded animals are susceptible, including humans and occasionally birds. [2]

17.3 Distribution

Anthrax occurs worldwide, [1, 3] most commonly in the tropics and subtropics. [2] New Zealand's last case of anthrax occurred in 1954. [4]

17.4 Clinical signs

Anthrax may take peracute, acute and subacute to chronic forms. [1 , 2, 3, 5] Horses are most likely to manifest the acute form. The manifestation may vary with the route of infection, but typically will result in death within 48-96 hours. [1] The OIE Code considers the incubation period for anthrax to be 20 days. [6]

17.5 Transmission

Infected animals have large numbers of bacteria in the blood. Haemorrhage before death and the opening of carcasses cause sporulation and environmental contamination. Anthrax spores remain viable in favourable conditions for at least 50 years. Animals are exposed to the spores by ingestion, inhalation or inoculation through the skin, such as by biting insects. [2]

No reports have been found suggesting transmission of anthrax by artificial insemination.

17.6 Diagnosis

Diagnosis is based on typical clinical signs, with confirmation by visualisation of *B. anthracis* in polychrome methylene blue stained smears from tissues and blood, or culture on 5% blood agar incubated overnight. [1]

A nested PCR has been developed to detect spores in environmental samples, and is between 10^4 and 10^7 times more sensitive than culture. Other antigen detection tests include the Ascoli test and immunofluorescence. [1]

Although AGID, CFT, indirect microhaemagglutination, ELISA and electrophoretic

immunotransblot methods have been described to detect antibodies to vaccination, there is no standardised serological test for the detection of anthrax in animals. [1]

17.7 Immunity

Vaccines produced from live spores from the variant $34F_{(2)}$ strain are available. Initial vaccination of horses requires two doses one month apart and annual boosting. [1]

17.8 Treatment

Antibiotics and anti-anthrax serum may be effective if given early, but severely ill animals are unlikely to recover. [2, 3, 4]

17.9 Risk assessment

17.9.1 Release assessment

The possibility that horses could be infected with anthrax must be considered whenever they are imported because the disease probably has a worldwide distribution. However, as anthrax in horses has a very short incubation period and death within 1-3 days is the most likely outcome, importation of horses that are incubating the disease is unlikely.

Semen does not present a pathway for introduction of anthrax.

17.9.2 Exposure assessment

Importation of a horse infected with anthrax would potentially result in exposure of in-contact animals and humans. Spores could also contaminate soils, resulting in on-going risk of exposure for many years.

17.9.3 Consequence assessment

A case of anthrax in imported animals, and any spread to in-contact animals or humans, would have significant direct and indirect consequences. The disease investigation and control effort would include management of the risks to public health. Restrictions on movement of animals and animal products from infected properties would be necessary. Some trading partners might temporarily impose measures on exports of animals and animal products, including meat and dairy products, as has occurred during outbreaks in Australia.

17.9.4 Risk estimate

The likelihood that horses will be incubating anthrax at the time of importation is very low because of the low incidence, short incubation period and obvious acute clinical signs of disease. However, a case of anthrax in livestock here, imported or otherwise, would have serious animal and public health consequences, so measures to manage this risk are warranted. Measures during importation

of semen are unnecessary.

17.10 Risk management

17.10.1 Risk management objective

Horses should be protected from infection during the pre-export period, and be free from clinical signs on the day of export.

Protection from infection during the pre-export period can be effectively achieved by requiring that no cases of anthrax have been reported from the premises of origin.

17.10.2 Risk management measures

The OIE Code [6] recommends a minimum 20 day period of premises freedom from cases of anthrax prior to export. Adherence to this recommendation is appropriate.

Live horses

1. The horses were kept during the 20 days prior to export on premises were anthrax has not occurred during that period; *and*

2. The horses were showing no clinical sign of anthrax on the day of export.

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18 Leptospirosis

18.1 Aetiology

Leptospirosis is an OIE List B disease of animals and humans caused the spirochaete *Leptospira*. There are seven pathogenic species of *Leptospira* with approximately 200 distinct serovars classified into 23 serogroups. Differentiation between serovars belonging to a particular serogroup is by cross-agglutination tests. [1]

18.2 Susceptibility

Leptospira have a wide host range, including a large number of mammals and poikilothermic vertebrates such as amphibians and reptiles. [2] The epidemiology of leptospirosis can be complex, with serovars having differing affinities for animal hosts and this affinity varying in different regions. [3]

Leptospirosis is an important zoonosis. [1, 2, 3, 4]

Serovars adapt to infect one or more maintenance animal hosts. Such hosts are typically characterised by high susceptibility, long-term kidney infection and urinary shedding. Serovars will also infect incidental (or accidental) hosts, which are characterised by low susceptibility but more severe disease if infected.[3] This should be appreciated as a generalisation as it probably oversimplifies the complex epidemiology of leptospirosis.

Rodents and other wild animals may be important as maintenance hosts for many leptospiral serovars and act as sources of infection for domestic animals. However, some serovars are maintained in domesticated animals. [2, 3, 4]

Horses are probably able to act as a maintenance host for serovar *bratislava.* There is a consistently high worldwide seroprevalence and a renal carrier rate of approximately 8%. The risk of infection increases with population density and other group management factors, and does not disappear when horses are managed in high biosecurity groups (such as on equine studs). Rodents, hedgehogs, pigs and dogs may also maintain serovar *bratislava,* and horses may be infected through exposure to these animals. [5]

Horses are susceptible as incidental hosts to a wide spectrum of other *Leptospira* serovars. [5] The predominant serotype infecting horses varies from country to country, and results are also dependent on the leptospiral antigens used in the survey.[6] A survey in Kentucky examining cases of equine abortions and using serological diagnosis only (versus isolation by culture) found the majority of cases were caused by serovar *pomona (*subtype *kennewicki),* followed by *grippotyphosa* and *bratislava.*[4] A study in Ireland assessed the strains causing abortion and jaundice in horses by culture and immunofluorescence, and found isolates from six serogroups*:* Australis (serovar *bratislava*), Ballum (*arboreae*), Canicola (*canicola*), Icterohaemorrhagiae (serovar not determined), Pomona (*pomona*) and Sejroe (*hardjo*).[6]

18.3 Distribution

Leptospirosis occurs worldwide. [1, 2, 3, 4] Infection is more common in areas or seasons when the climate is warm and humid, soils are alkaline and there is an abundance of surface water.[4] The distribution of particular serovars varies.

The serovars isolated from animals in New Zealand are serovar *hardjobovis,* serovar *pomona* (including subtype *kennewicki*), serovar *copenhageni,* serovar *ballum,* serovar *tarassovi* and serovar *balcanica.* [7]

Leptospirosis occurs in horses in New Zealand. In 1989 a recently aborted mare showed a very high titre to serovar *copenhageni*. A serological survey following up this report examined 762 equine sera samples from three sources (submissions to various veterinary laboratories covering various periods). Specific microscopic agglutination test (MAT) reactions (assumed to be titres of 25% agglutination at 1:200 serum dilution or more) to serovars *copenhageni* (16 horses, or 2.1%), *pomona* (8 or 1.0%), *hardjo* (11 or 1.4%), *bratislava* (18 or 2.4%), *tarrasovi* (3 or 0.4%) and *ballum* (1 or 0.1%). [8] Low titre reactions (50% agglutination in 1:100 serum dilution) were recorded in 245 samples (32%), including several low titre reactions to serovars *canicola* and *grippotyphosa,* considered exotic to New Zealand.

Serovar *pomona (kennewicki*) was isolated during an investigation into an outbreak of leptospirosis abortion on a New Zealand Thoroughbred stud in 1997 following introduction of a carrier mare imported from Australia. Serological evidence also suggested infection with serovar *bratislava.* [9, 10] Follow-up surveys found that in-contact mares had serological titres to serovars *copenhageni, hardjo, pomona* and *bratislava.* [9]

Equine sera samples (n= 590) held in the serum bank at the Central Animal Health Laboratory were tested for leptospirosis in 1998. Titres to serovar *bratislava* were detected in 50 samples and serovar *pomona (kennewicki)* in one sample (total seroprevalence of 8.6%).[11]

The serovars *canicola, grippotyphosa* and *icterohaemorrhagiae* have not been recorded in livestock in New Zealand.

18.4 Clinical signs

Most infections of horses with *Leptospira* are subclinical, although the very young and pregnant mares are particularly susceptible to clinical disease. [5]

Acute disease in pregnant mares is characterised by abortions, stillbirth and premature live birth. In young foals there may be fever, jaundice and death from interstitial nephritis. [5]

Chronically recurrent ophthalmia or uveitis may be a sequel to equine leptospirosis. [5]

18.5 Transmission

Infected animals shed leptospires in the urine during the later clinical phase and during the convalescent and recovery phases. Shedding persists for variable periods. Chronically infected carrier animals play an important role in maintaining infection. Aborted foetuses also are a source of infectivity. [2, 3, 4]

Horses may shed leptospires in urine for up to 4 months, but the level of leptospiruria is low.[4] The role horses play in maintenance and transmission of leptospirosis has not been fully elucidated. [5]

Leptospires contaminate pasture, feed and drinking water. Survival in the environment depends upon conditions of moisture, temperature and pH. The portal of entry into the body is through cutaneous or mucosal abrasions. Neonatal infection, probably in utero, has been recorded. [2, 3, 4]

In cattle, transmission is also possible through coitus or by artificial insemination.[2] Excretion of leptospires in semen has not been reported in stallions. [5]

18.6 Diagnosis

While identification of leptospires in blood is considered diagnostic, isolation is not always possible because bacteraemia is transient and not always accompanied by clinical signs. Demonstration of leptospires in the genital tract, kidney or urine indicates an animal is or was a carrier. Demonstration in tissues of aborted foetuses is diagnostic. Isolation by culture followed by identification provides a definitive diagnosis of infecting serovars. [1] Serovar *bratislava* is amongst the most difficult of *Leptospira* serovars to isolate (pers. comm. William Ellis, Department of Agriculture for Northern Ireland, 25 March 1998).

A PCR-based method for detecting serovar *pomona (kennewicki)* in samples of equine urine, eye fluid and leptospira cultures has recently been described. [12]

The MAT using live antigens is the most commonly employed serological diagnostic test, and is the test prescribed by the OIE for use during international trade. For optimum sensitivity the test should employ serovars of all the serogroups known to occur in the exporting country as well as those for which the species under test is known to act as a maintenance host. The MAT applied in this way may allow identification of the serogroup of the infecting serovar, although caution during interpretation is advised because of serovar cross-reactivity. The MAT is of most use as a screening test for a group of animals, and has limitations in its ability to detect a chronically infected individual. Paired sera demonstrating a rising titre are indicative of a recent infection. ELISAs have also been developed, and in general are quite sensitive but lack serovar specificity. [1]

The antibody titre is in no way indicative of the renal carrier state. [5]

18.7 Immunity

Agglutinating antibodies are detectable in horses 9 days after exposure. [3]

While inactivated leptospiral vaccines for sheep, cattle, deer, pigs and dogs are available, none are

licensed for horses. [5] Nevertheless, in the USA, 0.9% of establishments with horses less than 12 months of age, 2.8% of establishments with brood mares, and 2.5% of establishments with other horses over 12 months of age vaccinate against leptospirosis. [13]

Vaccination is not an effective means of eliminating leptospires from the kidneys of chronically infected animals. [1]

18.8 Treatment

The OIE Code [14] recommends when importing domestic livestock (including horses) that the animals be injected with 25 mg dihydrostreptomycin per kg of live body weight twice with an interval of 14 days, with the second injection within 24 hours of shipment.

Dihydrostreptomycin is no longer available for animal treatments in the USA, and streptomycin use may also be limited in the near future (pers. comm Lisa Ferguson, USDA, 4 September 1998). In Australia, these antibiotics may not be available for general use for much longer, although they will probably remain available for certain uses under permit where necessary on grounds of human health, animal welfare and trade. [15]

Leptospira are sensitive in vitro to penicillin G, ampicillin, ceftizoxime, erythromycin, kanamycin, streptomycin and tetracyclines. In-feed medication with tetracyclines has eliminated leptospiruria in pigs. [3] Recent studies in the USA have established the use of a long acting oxytetracycline at 20 mg/kg is highly effective in eliminating the chronic carrier state of serovar *hardjo* in cattle (pers. comm. Carole Bolin, USDA, 1 June 1999).

There are virtually no data on treatment of equine leptospirosis, so extrapolation from other species is required. Streptomycin, amoxycillin and oxytetracycline have been suggested to eliminate the carrier state. [5]

18.9 Risk assessment

18.9.1 Release assessment

The specific *Leptospira* serovars of concern to New Zealand are the known exotic animal pathogenic serovars, in particular serovars *canicola, grippotyphosa* and *icterohaemorrhagiae.* Surveys in horses here indicate they are already widely exposed to endemic serovars. Horses are known to be susceptible to infection with the exotic serovars. [4, 6] The prevalence of such infections is unknown.

Leptospirosis occurs worldwide, although the distribution of serovars varies. High seroprevalence rates in horses indicate high levels of exposure. An estimated 8% of horses may be renal carriers of serovar *bratislava.*

The period of renal shedding during incidental infection of horses with the exotic serovars is unknown. Experience in other livestock species indicates that incidental hosts shed leptospires in the

urine for short periods only, typically associated with acute clinical signs. However, acute clinical signs are rarely reported in equine leptospirosis.

18.9.2 Exposure assessment

The occurrence during 1997 of abortions in mares in contact with an imported mare clearly indicates the potential for exposure of horses to leptospirosis as a result of imports.

Of potentially greater importance would be exposure of maintenance host species in New Zealand to exotic serovars, which could lead to the establishment of endemic infection. The Norway rat (*Rattus norvegicus*) is readily infected by a low dose of serovar *icterohaemorrhagiae*, and then sheds large numbers in urine for long periods. Serovar *canicola* readily infects and is maintained in dogs.

While there are no data to indicate transmission via horses semen, extrapolation from other species suggests it should be considered as a possibility.

18.9.3 Consequence assessment

The consequences for individual premises of importing leptospirosis carriers were illustrated by the outbreak in a stud in 1997. They included a number of abortions, treatment of all in-contact horses, and a significant imposition on stud management during the investigation. [9]

The case referred to above was suspected to have involved *Leptospira* serovars already present in New Zealand. Introduction of exotic serovars would probably have similar consequences for individual premises. However, there would potentially also be consequences for other livestock industries and for public health. The magnitude of such consequences is difficult to predict because it would rely on the interaction of exotic serovars with hosts in the New Zealand environment. Such interactions may not conform with experience with endemic serovars.

18.9.4 Risk estimate

Horses infected with exotic *Leptospira* serovars could be imported leading to introduction and establishment in New Zealand. Semen collected from infected stallions may also present a risk. Aside from the consequences for individual premises affected by an outbreak of equine leptospirosis, the consequences of introduction may include impacts on other livestock industries and public health. Measures to ensure live horses and semen are not infected with exotic *Leptospira* serovars are warranted.

18.10 Risk management

18.10.1 Risk management objectives

Imported horses and semen should be free from infection with the exotic *Leptospira* serovars. Measures should be required during importations from countries where exotic *Leptospira* serovars occur.

Developing an effective but practical safeguard for leptospirosis is complicated in that serology does

not provide an indication of renal carrier status; serological diagnosis is not able to precisely define infecting serovars; there is a lack of specific data regarding treatment; antibiotics recommended by the OIE are not available in some countries; and treatments may have some undesirable effects.

Culture is not a sensitive technique to diagnose the carrier state. However, future developments in diagnostic techniques for equine leptospirosis may allow the introduction of further alternatives, such as application of PCR assays for urine specimens.

At the present time, MAT serology and antibiotic treatment are probably the only practical safeguards that can be applied, and should be considered as alternative options.

18.10.2 Risk management measures

Detectable titres of agglutinating antibodies appear soon after infection in horses, and persist longer than the likely period of urine shedding. However, it is widely recognised that the MAT has limitations as a screening test in individual animals, and results are best interpreted at a herd level. Cross-reactivity between serovars confounds serovar-specific serological diagnosis. Using the MAT to determine whether an animal is infected with a certain serovar based on low titre differences in serogroup agglutination would not be reliable (pers. comms. Carole Bolin, USDA, 1 June 1999; and David Miller, USDA, 28 April 1999). Nonetheless, negative serology probably provides a strong assurance that the horse is not currently infected, and therefore provides a useful pre-export measure. If used in this way, requiring serology to be negative (<50% agglutination) at the 1:200 titre may provide the most appropriate interpretation (pers. comm. David Miller, USDA, 28 April 1999).

The OIE treatment recommendation provides the basis for New Zealand's leptospirosis safeguards in all domestic livestock species. Specific data indicating the efficacy of alternative treatments, such as oxytetracycline or amoxycillin, in eliminating the renal carrier state are required. Oxytetracycline has been demonstrated to be effective in eliminating the chronic carrier state of serovar *hardjo* in cattle. Considering that use of dihydrostreptomycin and streptomycin in horses is also reliant on extrapolation from other species, use of oxytetracycline as a pre-export treatment for horses is probably reasonable.

Localised tissue reactions to intramuscular antibiotics are a side-effect, so there may be a reluctance to treat horses that are expected to be fit for competition very soon after arrival. Flexibility in the treatment period, such as allowing treatments during the 30 day period prior to export, may account for these concerns.

Streptomycin and dihydrostreptomycin are not classed as performance enhancing or inhibiting drugs for horses in New Zealand. Analytical testing regimes for competition horses are unlikely to detect these antibiotics, and even if detected they would not be considered of any consequence. However, procaine penicillin is commonly combined with streptomycin and dihydrostreptomycin in antibiotic preparations, and would be detected (pers. comm. Murray Brightwell, Equine Branch New Zealand Veterinary Association, 15 March 1999).

Freedom from clinical cases on the premises of origin of the horse, ascertained by due enquiry and/or examination of any relevant records (such as private veterinary practitioner records of disease investigations), provides some level of assurance that horses have not been in contact with known sources of infection in the pre-export period. The high rate of subclinical infection and potential exposure to wildlife sources means premises freedom from clinical cases is not in itself a complete safeguard.

European Union legislation (Council Directive 92/65/EEC, through reference to the provisions of Directive 90/429/EEC) requires the addition of an effective combination of antibiotics, in particular against leptospires and mycoplasmas, to be added to the semen after final dilution. This measure should provide sufficient safeguard against the risk of leptospirosis in equine semen.

Live horses

1. The horses were kept for the 3 months prior to export on premises where clinical cases of leptospirosis in livestock have not occurred during that period; *and*

2. During the 30 day period prior to export:
Either: i) The horses were subjected to the MAT employing antigens from serogroups representative of serovars known to infect horses in the exporting country and *Leptospira* serovars *canicola, grippotyphosa* and *icterohaemorrhagiae*, with negative results (<50% agglutination at the 1:200 titre);
Or: ii) The horses were injected with dihydrostreptomycin or streptomycin (at a dose rate of 25 mg/ kg of live body weight) on two occasions with an interval of not less than 14 days;
Or: iii) The horses were injected with long-acting oxytetracycline (at a dose rate of 20 mg/ kg of live body weight) on two occasions with an interval of not less than 14 days.

Horse semen

1. An effective combination of antibiotics, in particular against leptospires and mycoplasmas, must be added to the semen after final dilution. This combination must produce an effect at least equivalent to the following dilutions:
not less than: 500 IU per ml streptomycin,
500 IU per ml penicillin,
150 mg per ml lincomycin,
300 mg per ml spectinomycin.
Immediately after the addition of the antibiotics the diluted semen must be kept at a temperature of at least 15 °C for a period of not less than 45 minutes.

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19 Contagious equine metritis

19.1 Aetiology

Contagious equine metritis (CEM) is an OIE List B disease caused by the bacterium *Taylorella equigenitalis*. All isolates are serologically and morphologically similar but colony variations are related to virulence, with large colony variants being more virulent. [1, 2, 3]

In 1998 an organism phenotypically indistinguishable from *T. equigenitalis* was isolated from healthy donkeys in California and Kentucky. Transmission occurred during natural and artificial service, and infected animals seroconverted to the CFT for *T. equigenitalis.* DNA sequence analysis indicated differences (97.6% homology) between the American isolate and previously known isolates, and the new isolates have been proposed to be considered a new species in the genus *Taylorella* and named *T. asinigenitalis.* [4]

19.2 Susceptibility

CEM is an infection of horses, although donkeys are also susceptible. [1, 2]

19.3 Distribution

CEM was first described in the UK in 1977, after which it was diagnosed throughout Europe, North America, Australia and other countries.[1] Several countries appear to have successfully controlled CEM, including Australia (last recorded case 1980).[5] There were several outbreaks of CEM in Britain during 1996. [6] CEM occurs in Japan. [5] CEM has never been reported in South Africa,[2] or New Zealand.

A PCR test for *T. equigenitalis* was applied to populations of horses in the Netherlands and in other countries including the UK and Iceland. Detected prevalence was higher than suggested by clinical signs and culture.[7] It has been hypothesised that virulent *T. equigenitalis* emerge incidentally from non-virulent strains which inhabit the equine reproductive tract as commensals and do not interfere with fertility.[8]

In another study the culture-PCR test (application of the PCR assay to a sample taken from the primary inoculation site of a swab culture, in order to detect the presence of *T. equigenitalis* that are not forming visible colonies because of overgrowth by contaminants) was applied to 217 swabs from 174 horses submitted for CEM culture to the Animal Health Trust. All samples were negative, leading to a conclusion that the prevalence of PCR-positive but culture-negative horses in the UK is low. [9]

19.4 Clinical signs

Clinical cases are manifested as mucopurulent vaginal discharge 2 to 12 days after infection. There is inflammation of the endometrium causing temporary infertility. Mares may exhibit more than one

episode of disease in a short period of time. Most recover uneventfully but become carriers of *T. equigenitalis*. Carriage may result in poor fertility, but does not always affect conception. Abortion occurs on rare occasions. [1, 3]

Many primary infections of mares are subclinical. Stallions are invariably asymptomatic.[1]

19.5 Transmission

T. equigenitalis is a true venereal pathogen. Infected mares and carrier stallions act as reservoirs, with intermittent shedding of the organism in exudates and venereal excretions for long periods following remission (or absence) of clinical signs. CEM is highly contagious and can also be spread indirectly on fomites contaminated with genital exudates, such as equipment used for genital examinations or to wash and tail-bandage mares. [1, 2, 3]

Foals born to infected dams may be infected at parturition, becoming long term asymptomatic carriers. [1, 8]

Transmission from infected stallions via smegma has also been recorded. Infection can be transmitted by artificial insemination. [10]

19.6 Diagnosis

Diagnosis is by culture and can be difficult because of the fastidious nature of *T. equigenitalis*. In stallions the principal sites of colonisation are the urogenital membranes of the urethra, urethral fossa and penile sheath. In mares the principal sites of colonisation are the urogenital membranes of the clitoral sinuses and fossa, urethra and cervix. Long-term persistence of the organism in the uterus has been observed on rare occasions. [1]

The OIE recommends that a quality control system should be in place before a laboratory is approved to undertake testing for CEM. The OIE refers to the swabbing protocol recommended by the Horserace Betting Levy Board (HBLB). The British Equine Veterinary Association and the HBLB make recommendations for CEM diagnosis based upon classification of mares as high or low risk as determined by breeding history. The sites of swabbing in the mare are the clitoral sinuses and fossa, the cervix and on at least one occasion a swab from the endometrium during oestrous. The cultures obtained from the cervix and the endometrium are less contaminated than those from the external genitalia, where overgrowth from other organisms may prevent a diagnosis. High-risk mares are subjected to swabbing and culture on three occasions. All stallions are swabbed at the start of the breeding season on two occasions. [1, 7, 11]

Serological tests do not detect all carriers and should not be considered a substitute for culture. [1]

PCR tests have been developed and applied either directly to swabs or as a means of identifying culture isolates. [7, 8, 12]

19.7 Immunity

Neither prior infection nor vaccination is protective. Serum antibody declines rapidly after natural infection. [1]

19.8 Treatment

Carriers of *T. equigenitalis* can be treated using systemic antibiotics combined with disinfectant washing of exposed genital membranes. Treatment may take weeks, and repeated washing may be required. A significant number of mares can be refractory even to several courses of treatment. [2] Infections in stallions also may not be cleared by washing with chlorhexidine, and such treatments may have a negative effect on fertility because of toxic action of the disinfectant on semen. Treatment with enrofloxaxin, on the otherhand, was found to be effective in stallions. [8]

Nonetheless, treatment is now recognised by the HBLB as usually being effective, and this has lead to a reduction in the period that a mare is considered high risk from 5 years to 2 years following treatment, if during that period the mare has had a foal which has tested negative for CEM. [13]

19.9 Risk assessment

19.9.1 Release assessment

New Zealand is at risk of CEM introduction during importation of mares, stallions and horse semen from countries where the infection occurs. Even in such countries, the prevalence of CEM is probably low. The presence of control programmes in exporting countries, such as the HBLB programme in the UK, reduces the risk of infected animals being exported.

Infected stallions are always asymptomatic. Mares may or may not show acute clinical signs. Asymptomatic long-term carriage may occur in both stallions and mares.

The health status of foals under the age of 12 months can be considered the same as the dam. However, because of the potential for transmission during parturition, the health status of foals should be assessed in situations in which the dam is unavailable for testing.

19.9.2 Exposure assessment

CEM is a venereally transmitted disease. Infected horses imported for breeding purposes would be likely to expose other horses to infection during either natural or artificial service. Imported infected semen is also likely to infect inseminated mares.

Horses imported on a temporary basis to compete are not likely to introduce CEM so long as they do not have sexual contact with New Zealand horses. Likewise, importation of geldings will not lead to CEM introduction and spread.

19.9.3 Consequence assessment

The consequences of CEM introduction would be confined within the equine industry. Direct consequences associated with reproductive wastage and control efforts would probably be significant. Indirect effects associated with trading partners imposing measures would have a significant impact on exports. Trade with Australia, a CEM free country, would be disrupted, and measures would be imposed on exports of breeding horses and semen to other countries.

19.9.4 Risk estimate

Mares and stallions imported for breeding from infected countries could introduce and spread CEM leading to significant adverse consequences for the equine industries. Semen collected from infected stallions also presents a pathway for introduction and spread. Sexually immature horses might have been infected by their dam. Measures against CEM during importation of mares, stallions, foals and semen are warranted.

19.10 Risk management

19.10.1 Risk management objective

When importing from infected countries, mares, stallions and foals (when the health status of the dam is unknown) should be tested negative for CEM, and have been protected from infection in the pre-export period.

The diagnostic regime recommended by the HBLB and OIE is based on serial swab and culture from specified sites around the genitalia. Once PCR assays are properly validated and standardised, they may provide a useful alternative or adjunct to culture-based methods.

Protection from infection can most simply be achieved by requiring that horses have not been mated from the time of testing until export. Requiring horses during the 3 months prior to export to not visit premises where there are infected horses will further reduce opportunities for exposure.

Donor stallions from infected countries should be subjected to a regime of swabbing and culture. Testing during the breeding season in which the semen is collected should be considered acceptable, although if testing occurred prior to collection, the stallion should have been permitted to serve mares only of equivalent health status from the time of testing until collection.

Temporary importation of competition horses does not present a risk of exposing local horses so long as they are not used for breeding.

19.10.2 Risk management measures

There is no international standard for recognition of countries as free from CEM, so difficulty will arise in determining the status of countries that have previously reported infection but subsequently claim freedom. A cautious approach is warranted if surveillance data are lacking or equivocal.

The OIE Code [(14)] recommends importing countries require stallions and mares to show no clinical

signs, have had no contact with infected animals or premises, and to have had negative laboratory tests for CEM prior to export. These measures are consistent with the objectives above. Variations involving testing of foals, the number and timing of swabs, the use of an endometrial swab during oestrous, and measures for pregnant mares require further discussion.

If a horse has not been mated, the chances of it being a carrier are significantly reduced. However, the risks are not eliminated because the foal may have been infected by the dam at birth or from fomites. As horses are unlikely to have been bred prior to 2 years of age, the status of horses younger than 2 years should be assumed to be negative if the dam is available and has tested negative. If the dam is not available, young horses should be tested. However, fillies should only be tested by swabbing the less invasive sites of the external genitalia, and a deep cervical or endometrial swab should not be required.

Live horses should be swabbed for CEM on three occasions before it can be accepted that they are not infected. The USDA requires swabbing to be done over a 7 day period on days 1, 4 and 7, whereas AQIS requires swabbing on three occasions at intervals of 5-10 days.[15] Either regime should be considered acceptable.

In countries with approved Codes of Practice for CEM, such as the United Kingdom and the HBLB Code of Practice, there is a reduced risk of exposure to infected horses as a result of testing requirements for all horses used for breeding. Any testing in the breeding season prior to export may be used to fulfil the export test requirements, so long as there has been no sexual contact with horses not of equivalent health status since the time of the first swab considered to be for export purposes.

Requiring swabs on three occasions will ensure that in the case of stallions and low risk mares at least one swab is performed in the immediate pre-export period, and this is justified by the higher risk associated with live horse imports. When semen is imported, swabbing of stallions need only have occurred on at least two occasions, as required by the HBLB. Other factors, such as the addition of antibiotics to semen, mitigate the risk.

The OIE and HBLB swabbing sites include the endometrium and deep cervix on at least one occasion during oestrous. These sites are less heavily contaminated and swabbing there may increase the chance of successful culture by reducing bacterial overgrowth.[1] However, the endometrial swab is no longer a part of the USDA protocol for mares, following a recommendation from a panel of experts which concluded that it did not provide additional assurances (pers. comm. Lisa Ferguson, USDA, 20 March 1998). AQIS still requires the endometrial swab.[15] New Zealand should continue to follow the OIE and HBLB recommendations and swab the endometrium on at least one occasion, at least until more definitive proof on the benefits or otherwise of swabbing this site is forthcoming.

The endometrial swab is contraindicated in pregnant mares. AQIS allows a declaration of establishment of origin freedom from CEM for the 12 months prior to export and further post-arrival quarantine restrictions in its place when importing pregnant mares.[15] Pregnant mares remain under quarantine restrictions until after foaling when the endometrial swab can be performed. This practice could be adapted to the New Zealand context. A further acceptable alternative would be to require

both the stallion and the mare to have been tested for CEM prior to service in accordance with the requirements noted above.

Live horses

Either:

1. The horses have been resident since birth in a country that is free of CEM.

Or:

1. The horse is a gelding.

Or:

1. The horses were kept during the 3 months prior to export on premises where CEM has not occurred during that period; *and*

2. During the 60 day period prior to the export the horses have been tested for CEM by swabbing and culture on three occasions with a negative result for *Taylorella equigenitalis* in each case. The swabs may be taken on days 1, 4 and 7 over a 7 day period, or at 5-7 day intervals. (Horses less than 731 days of age are exempt testing if their dam is available for testing and is negative.) (In countries with approved Codes of Practice for CEM, any testing undertaken in the breeding season prior to export may be used to fulfil export requirements of testing on three occasions. The horse must be certified as not having had sexual contact with horses not of equivalent health status since the time of the first swab considered to be for export purposes.); *and*

3. The sites for swabbing are:
i) In stallions, from the prepuce, the urethral sinus, and the fossa glandis (including its diverticulum);
ii) In mares, from the mucosal surfaces of the urethra and the mucosal surfaces of the clitoral sinuses and clitoral fossa, and if the mare is greater than 731 days old, from the mucosal surfaces of the cervix, and the endometrium (on at least one occasion); *and*

4. Since the date of first swabbing for CEM testing, the animal has not been naturally mated except to horses of equivalent health status; *and*

5. In the case of pregnant mares:
Either: i) The stallion and mare were tested for CEM by swabbing on three occasions during the 60 day period prior to mating according to the protocols noted at points 2 and 3 above, and had no sexual contact with any other horses from the time of first swabbing until the time of last service (in the case of stallions, collection of semen for artificial insemination is permitted);
Or: ii) The pregnant mare has been swabbed and cultured prior to export, but the cervical and endometrial swab were not performed. After arrival in New Zealand, the pregnant mares must be held in a registered quarantine facility in New Zealand until the cervical and endometrial swab can be completed subsequent to foaling.

Horse semen

Either:

1. The donor stallions have been resident since birth in a country that is free of CEM.

Or:

1. The donor stallions were kept for the 3 months prior to collection on premises where CEM has not occurred during that period; *and*

2. During the breeding season in which the semen for export is collected the donor stallion has been tested for CEM by swabbing and culture on two occasions, with a 5-7 day interval, with a negative result for *Taylorella equigenitalis* in each case; *and*

3. The sites for swabbing are from the prepuce, the urethral sinus, and the fossa glandis (including its diverticulum); *and*

4. If testing occurs prior to collection of semen, since the time the first swab was taken until the time of collection the donor stallion has been permitted to naturally serve only mares of equivalent health status.

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20 Glanders

20.1 Aetiology

Glanders is a contagious and often fatal OIE List B disease of Equidae caused by the bacterium *Burkholderia mallei,* [1] until recently known as *Pseudomonas mallei*. [2, 3, 4]

20.2 Susceptibility

Horses, donkeys and mules are susceptible. [1, 2, 3] Glanders is a zoonosis, and 95% of human cases are fatal.[1] Human cases typically result from occupational exposure.

Glanders also occurs naturally in carnivores, rodents, cattle, sheep, goats and pigs. [3]

20.3 Distribution

Although once very widespread, glanders has disappeared from many countries. It persists in some areas of Eastern Europe, Asia (India, Pakistan, Turkey and China) and North Africa.[3]

20.4 Clinical signs

The incubation period for glanders varies from a few days to many months. [1, 3] For the purposes of the OIE Code, the incubation period is considered to be 6 months. [5] Acute and chronic forms are described. In horses glanders generally occurs in the chronic form, whereas in donkeys and mules the acute form is more common. [2, 3]

Acute glanders is characterised by fever, nasal discharges and ulceration of nasal membranes. There may be progressive respiratory signs, and swollen submandibular and other superficial lymph nodes may rupture to discharge pus. Bacteraemia occurs and may lead to death within weeks. [3]

Chronic glanders may progress over several years. Respiratory signs are mild. There may be enlarged submandibular lymph nodes and thickening of the skin in the extremities, particularly the hind legs. The skin form (farcy) is typified by enlarged lymphatics and the formation of nodular abscesses in the skin, which ulcerate and discharge pus. [3]

20.5 Transmission

Transmission is by ingestion, inhalation or inoculation. Nasal discharges, pus from cutaneous lesions, urine, saliva, tears and faeces are infective. *B. mallei* is an obligate parasite, and is easily destroyed by heat, sunlight and drying. [2, 3]

Asymptomatic carriers are very important in disease transmission. Chronic infection may persist with periodic shedding. [2, 3]

20.6 Diagnosis

Smears and culture from lesions may lead to a diagnosis, [1] although bacteriology is usually complicated by samples being highly contaminated. [6]

The tests prescribed by the OIE for use during international trade are the mallein test and the CFT. The mallein test involves inoculation of purified protein derivative. The lower eyelid (the intradermopalpebral mallein test) is the most reliable and sensitive location. The CFT has been used for many years but is not as sensitive as the mallein test. It is reported to be 90-95% accurate. Serum becomes positive 1 week after infection and remains positive for a long time in chronic cases. Healthy horses may have a CF titre for a variable period following the mallein test. [1]

A dot ELISA is also available for serological diagnosis.[1, 6] It is reported to be more sensitive than the CFT, and will detect antibody within 4-6 days of infection. [6]

20.7 Immunity

No vaccines are available. Recovered animals are not always immune to re-infection. [2, 3]

20.8 Treatment

Treatment is not recommended. Control measures normally include a stamping out policy. [2, 3]

20.9 Risk assessment

20.9.1 Release assessment

The limited geographical distribution of glanders probably most influences the likelihood of disease introduction. The prevalence of glanders in infected countries is unknown. The disease spreads rapidly once introduced onto premises where horses are present. The ability of surveillance systems to detect, notify and control such outbreaks in infected countries will determine whether infected horses are likely to be imported.

The incubation period may be long and, although the clinical signs are typically obvious, asymptomatic carriers are the most likely source of infection. The occurrence rate of such carriers is unknown.

There are no reports of transmission by artificial insemination. However, the shedding of bacteria in various secretions by subclinically infected horses and the multiple pathways by which transmission occurs suggest imports of semen from countries where glanders in endemic should be treated with some caution.

20.9.2 Exposure assessment

Direct horse to horse transmission by a variety of routes indicate multiple exposure pathways if an infected horse were imported.

20.9.3 Consequence assessment

Imported cases would lead to direct spread, and movement of horses may result in multiple infected properties prior to a diagnosis being made. Overseas experience in controlling and eradicating glanders indicate that this would be possible given the resources. Inevitably there would be trade measures imposed by New Zealand's trading partners on live horse and semen exports. Exports of other livestock species could also be affected.

As glanders is a serious zoonosis there would be public health consequences associated with an outbreak. These would principally concern those persons with occupational exposure to infected animals.

20.9.4 Risk estimate

Horses and semen imported from endemic areas could be infected with glanders, and this could result in an outbreak of disease here with significant consequences for the equine industries and for public health. Measures against glanders during importation of horses and semen are warranted.

20.10 Risk management

20.10.1 Risk management objective

Horses should be protected from infection during the period prior to export or semen collection, and be tested for glanders with negative results. The intradermopalpebral mallein test and either CFT or dot ELISA should be considered acceptable.

20.10.2 Risk management measures

The OIE Code [5] describes conditions for a country to be considered as free from glanders, and recommends 6 months residency for horses imported from such countries. Horses imported from infected countries should be subjected to 6 months premises of origin disease freedom and pre-export testing. These measures are consistent with the objectives above for live animals.

The Code [5] does not make recommendations when importing semen. However, the European Union, the USA and Australia do impose measures, suggesting that sufficient uncertainty with regard to transmission via semen remains for New Zealand to adopt a cautious position. Similar measures should apply for donor stallions as for live horses.

Live horses

Either:

1. The horses were resident since birth, or at least the previous six months, in a country that

is free of glanders according to the criteria within Article 3.4.8.2 of the OIE Code.

Or:

1. The horses were kept for the 6 months prior to export on premises where glanders has not occurred during that period; *and*

2. The horses were subjected to the intradermopalpebral mallein test, CFT or dot ELISA for glanders not less than 7 days after entering pre-export isolation, with a negative result; *and*

3. The horses were showing no clinical signs of glanders on the day of export.

Horse semen

Either:

1. The donor stallions were resident since birth, or at least the previous six months, in a country that is free of glanders according to the criteria within Article 3.4.8.2 of the OIE Code.

Or:

1. The donor stallions were kept for the 6 months prior to collection on premises where glanders has not occurred during that period; *and*

2. The donor stallions were subjected to the intradermopalpebral mallein test, CFT or dot ELISA not less than 21 days after entering the semen collection centre or after semen collection, with negative results; *and*

3. The donor stallions were showing no clinical signs of glanders on the day of collection.

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21 Bovine brucellosis (*Brucella abortus*)

21.1 Aetiology

Bovine brucellosis is an OIE List B disease of cattle characterised by abortions and caused by the bacterium *Brucella abortus*.[1] *B. abortus* may also infect horses, causing the conditions fistulous withers and poll evil. [2]

21.2 Susceptibility

Cattle are the main natural host species for *B. abortus*. The bacterium is an important zoonosis, and readily infects humans. Horses are naturally susceptible, as are camelids, pigs, sheep, goats and dogs. [2]

21.3 Distribution

B. abortus has had a world wide distribution but a number of countries have successfully eradicated it. These countries include Australia, Canada, Israel, Japan, Austria, Switzerland, Denmark, Finland, Norway, Sweden and the United Kingdom. [3] *B. abortus* has also been eradicated from New Zealand. [4]

21.4 Clinical signs

B. abortus tends to localise in bursae, tendons, muscles and joints of infected horses. The result is chronically discharging abscesses associated with fistulous withers and poll evil. Abortions in horses have also been described. [2]

21.5 Transmission

Transmission occurs via ingestion of material contaminated from aborted foetuses and membranes, or urine and faeces from a cow that has recently aborted. Cattle only shed large numbers of bacteria into the environment at or around the time of abortion. Cows remain chronically infected, and the disease spreads from farm to farm by live animal movements.[3]

Abortions in mares may pose a risk of transmission to cattle, as may discharges from fistulous withers or poll evil that contain high numbers of organisms. [2]

Investigation of the significance of horses in the epidemiology of *B. abortus* in the United Kingdom found seroprevalence varied between 5 -19% during the years 1976 to 1983 (depending on the year and the test). Experimentally infected horses did not excrete the organism in sufficient numbers to infect susceptible cattle with which they were in contact. Horses are probably infected by contact with infected cattle, but infection of cattle by horses is probably very unlikely. [5]

21.6 Diagnosis

Diagnosis is by bacteriological isolation of *B. abortus* from aborted foetuses or membranes in cattle, or from discharges from typical lesions in horses. (1)

Several serological diagnostic tests are available for brucellosis in cattle. They may be used in horses but have not been adequately validated.

21.7 Immunity

Vaccines are available for use in cattle, but are rarely used in horses. (6)

21.8 Treatment

Treatment of *B. abortus* infections in horses is difficult because the nature of the lesions means recurrence is commonly encountered. Options for treatment include excision, debridement and lavage of lesions, and antibiotics. Vaccination of horses using *B. abortus* strain 19 has been attempted as a treatment for fistulous withers.(6)

21.9 Risk assessment

21.9.1 Release assessment

Infection with *B. abortus* is a well-recognised syndrome in horses. The classical lesions would prevent any infected horse being certified for export as free from clinical signs of infectious or contagious disease. Horses incubating infection and not showing clinical signs might occasionally be imported. A seroprevalence study has indicated up to 19% of horses may have been exposed to *B. abortus*, although the number of horses harbouring subclinical infections at any one time is likely to be lower.

21.9.2 Exposure assessment

With the possible exception of horses with discharging lesions and mares which abort, horses probably do not excrete *B. abortus* in sufficient quantities to infect cattle. Horses with discharging lesions are unlikely to be imported and, if the lesions occurred after importation, would be likely to receive veterinary attention and treatment. The likelihood of imported infected horses leading to transmission to other horses or, more importantly, cattle is low.

21.9.3 Consequence assessment

Re-introduction of brucellosis into New Zealand cattle populations would have significant adverse consequences for the cattle industries, including trade measures on live cattle, genetic material, and some cattle product exports. *B. abortus* is a zoonosis and so there would also be public health consequences from re-introduction.

A diagnosis of *B. abortus* in an imported horse would probably not result in the significant trade-related consequences for the cattle industries if appropriate disease investigation and control

measures indicated the infection had not spread.

21.9.4 Risk estimate

Importation of horses is unlikely to lead to re-introduction of *B. abortus* into cattle in New Zealand. Requiring horses to be free from clinical signs of infectious or contagious disease on the day of export (or semen collection) will reduce the likelihood of clinical cases being imported. Specific measures aimed at detecting subclinical infections are not warranted.

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22 Melioidosis

22.1 Aetiology

Melioidosis is a bacterial infection caused by *Pseudomonas pseudomallei* (renamed *Burkholderia pseudomallei*). There are strains of varying pathogenicity. [1, 2]

22.2 Susceptibility

Melioidosis has a very wide host range. It is primarily a disease of rodents, but occurs in many domestic and wild animals, in fish, and in humans. [1, 2]

22.3 Distribution

Melioidosis has been reported in South East Asia, Australia, the Caribbean and Europe. Most cases occur during the wet season in low-lying swampy areas.[1, 2] The disease is recognised as an emerging problem, with countries within the known endemic zones (particularly South-East Asian countries such as Thailand) reporting increasing numbers of human cases. Cases have been reported in India and China in recent years, and in Africa and the Middle East. An outbreak occurred in France in the mid-1970's. Imported human cases occur in the United Kingdom. [3]

Melioidosis does not occur in New Zealand.

22.4 Clinical signs

Infection with *B. pseudomallei* causes a usually fatal septicaemia. At post mortem the principal feature is multiple abscesses in most organs. [1, 2] Melioidosis in horses normally manifests as an acute metastatic pneumonia with a fever. There may be colic, diarrhoea and lymphadenitis of the legs. The course is typically short, although horses may survive for several months. [2]

22.5 Transmission

Although originally considered a zoonosis with a reservoir in rodents, *B. pseudomallei* is now known to be a widely distributed environmental saprophyte. Most human and animal cases probably arise through exposure to contaminated soil or muddy water. Laboratory animals have been experimentally infected by ingestion, inhalation and insect bites, but evidence of naturally acquired infection by these routes remains anecdotal. Infected humans and animals excrete the organism in faeces, sputum, urine and pus, from whence new environments may become contaminated. The organism survives for long periods in water and soil in favourable environmental conditions, predominantly tropical and sub-tropical climates (i.e. warm and wet). However, whether the organism will multiply once introduced into a new environment is entirely unknown. The evidence for animal to human or animal to animal transmission is relatively weak. Whether a human or animal becomes infected and succumbs to disease after encountering *B. pseudomallei* in the environment will depend on the virulence of the strain, the immune status of the host, and the size of the inoculum. [4]

22.6 Diagnosis

The organism may be cultured from discharges. An allergic skin test using melioidin as an antigen is available, as are serological CFT and indirect haemagglutination tests. [1, 2]

22.7 Immunity

No information has been found.

22.8 Treatment

B. pseudomallei is sensitive to antibiotic combinations such as trimethoprim and sulfamethoxinazole, and novobiocin and tetracycline. [1]

22.9 Risk assessment

22.9.1 Release assessment

Melioidosis has a tropical and sub-tropical distribution. Disease causes obvious clinical signs in horses. The risk of horses infected with *B. pseudomallei* being imported prior to manifestation of clinical signs is probably low.

No evidence regarding transmission by artificial insemination has been found, and semen probably does not present a pathway for disease introduction.

22.9.2 Exposure assessment

The evidence for direct horizontal transmission is weak. Whether imported cases of melioidosis would lead to introduction and establishment is primarily a factor of whether climates and soil-types here would support not only survival, but multiplication of *B. pseudomallei*. If this occurred at all, it would be restricted to areas of New Zealand with favourable wet and warm climatic conditions, if there are such areas.

22.9.3 Consequence assessment

Introduction of *B. pseudomallei* into New Zealand could have significant direct and indirect consequences for the equine and other livestock industries, as well as for public health.

22.9.4 Risk estimate

There is a low risk that imported horses may introduce *P. pseudomallei* leading to establishment in particular areas of New Zealand and significant adverse consequences for the livestock industries and for public health. Measures to reduce the risk of infected horses being imported are warranted. Semen probably does not present a risk.

22.10 Risk management

22.10.1 Risk management objective

Horses should be free from clinical signs of melioidosis at the time of importation, and be protected from infection during the pre-export period. Requiring horses to originate from premises that have not recorded any case during the pre-export period will reduce the opportunities for infection prior to export.

22.10.2 Risk management measures

Live horses

During importation of all horses the following safeguards should be met:

1. The horses were kept during the 3 month period prior to export on premises where melioidosis has not occurred during that period; *and*

2. The horses were showing no clinical signs of melioidosis on the day of export.

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23 Equine salmonellosis (*Salmonella abortus equi*)

23.1 Aetiology

Salmonellosis is caused by infection with any of the more than 2,000 serotypes of species within the genus *Salmonella*, family Enterobacteriaciae. [1, 2, 3] *S. abortus equi* is an equine adapted organism, and is rarely isolated from any other animals. [4]

23.2 Susceptibility

Although salmonellosis occurs in humans and all animals, with some serotypes adapted to humans or specific animal host species, *S. abortus equi* occurs only in horses and donkeys. [1, 2, 3, 4]

23.3 Distribution

S. abortus equi used to be widely reported in the early 1900s, but is now rarely encountered. The distribution of the disease as reported to the OIE in 1992 (the last year for which the OIE collected distribution information for *S. abortus equi)* showed that the disease occurred in South America, North America, Asia, Europe and Africa.[5] The disease probably has a reducing prevalence in most areas, and is now rarely isolated in the USA [2] and South Africa.[4]

S. abortus equi has not been reported in New Zealand, although enteric salmonellosis occurs in livestock here. [6, 7]

23.4 Clinical signs

S. abortus equi infection in the pregnant mare causes abortion, typically in the seventh or eighth month of gestation, with retention of placenta and metritis as common sequelae. Foals carried to term may develop acute septicaemia in the first days of life, or develop polyarthritis 7-14 days later. Infection in the stallion causes fever, orchitis, with swelling in the prepuce and scrotum, and arthritis. [4, 8] *S. abortus equi* has also been isolated in cases of tendovaginitis, bursitis and pneumonia in horses.[4]

23.5 Transmission

Transmission of *S. abortus equi* is typically by ingestion of the organism on pasture or food contaminated with uterine discharges from carriers or recently aborted mares. Venereal transmission at coitus is also thought to occur, although the epidemiological role of the stallion is uncertain. In utero infections occur. The organism has not been recovered from faeces. [4, 8]

23.6 Diagnosis

Diagnosis is by isolation of *S. abortus equi* from an aborted foetus or membranes through culture on selective media. [1]

Serum agglutination tests on paired sera samples may allow significance to be attributed to be antibody titres. The SAT will not differentiate between serotypes unless performed by specialised reference laboratories using an assortment of reference antigens. (1)

23.7 Immunity

Inactivated vaccines against *S. abortus equi* are available,(2) and autogenous vaccines may be prepared and used in specific outbreaks.(4)

23.8 Treatment

Antibiotic therapy, preferably after determining drug sensitivity, is indicated.

23.9 Risk assessment

23.9.1 Release assessment

Although *S. abortus equi* has historically had a wide geographical distribution, the disease probably has a decreasing distribution and prevalence. The clinical syndromes of infection are typically easy to recognise. Subclinical carriers are important in spreading the disease.

23.9.2 Exposure assessment

Pregnant mares which abort and shed *S. abortus equi* onto pastures provide the main route by which New Zealand horses could be exposed to infection. The epidemiological role of the stallion is uncertain, and so artificial insemination using imported semen should be presumed to present a potential exposure pathway.

23.9.3 Consequence assessment

The consequences of *S. abortus equi* introduction would be confined to the equine industries. They would include the initial disease effects, such as abortion storms and high foal mortality rates, as well as the costs of investigation and control. Overseas experience suggests that the disease could be controlled and eradicated.

Measures against live horses and semen exports would probably be imposed by trading partners.

23.9.4 Risk estimate

Horses subclinically infected with *S. abortus equi* could be imported from endemic areas, leading to establishment in New Zealand and adverse consequences for the equine industries. Semen should also be presumed to present a pathway for disease introduction. Measures during importation of both live horses and semen are warranted.

23.10 Risk management

23.10.1 Risk management objective

Horses should be free from the clinical signs of infection at the time of importation or semen collection, and should have been protected from infection during the pre-export/pre-collection period. Requiring horses to originate from premises that have had no cases of disease will reduce the opportunities for infection.

23.10.2 Risk management measures

Live horses

1. The horses were kept during the 3 months prior to export on premises where equine salmonellosis (*S. abortus equi*) has not occurred during that period; *and*

2. The horses were showing no clinical signs of equine salmonellosis on the day of export.

Equine semen

1. The donor stallions were kept for the 3 months prior to collection on premises where equine salmonellosis (*S. abortus equi*) has not occurred during that period; *and*

2. The donor stallions were showing no clinical signs of equine salmonellosis on the day of collection.

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24 Lyme disease

24.1 Aetiology

Lyme disease is caused by infection with the spirochaete *Borrelia burgdorferi.* [1, 2]

24.2 Susceptibility

Disease occurs in cattle, horses, sheep, dogs and humans. The reservoir hosts for *B. burgdorferi* are rodents and white-tailed deer. [1, 2]

Lyme disease is an important zoonosis and accounts for over 90% of vector borne disease in the USA, where it was the ninth leading reported infection for 1995 with 11,700 human cases.[3]

24.3 Distribution

Lyme disease is distributed throughout the USA, Canada, Europe, Australia, Asia and in countries of the former USSR. The geographical distribution is closely related to the distribution of the various *Ixodes* spp. ticks that are important for disease transmission. [2]

Seroprevalence in horses in the USA is approximately 10%, but may be much higher on individual premises in endemic areas. [2]

Lyme disease does not occur in New Zealand.

24.4 Clinical signs

Subclinical infection of horses is much more common than clinical disease. In horses the clinical signs of infection are chronic weight loss, sporadic lameness, arthritis and neurological signs. Poly-arthritis, abortion and the birth of foals that are weak and die soon after birth also occur. [2]

24.5 Transmission

B. burgdorferi is transmitted by *Ixodes* spp. ticks. Other blood-sucking arthropods may be involved in transmission, but this has not been conclusively demonstrated. [1, 2]

B. burgdorferi is excreted in the urine, and direct spread via urine/mucosal contact has been demonstrated in some rodent reservoir hosts. [2] Clinically normal and naturally infected horses have been found to shed the organism in urine. [4]

Transplacental transmission occurs, and can cause mortality in foals. [2]

24.6 Diagnosis

In domestic animals and humans the organism is found in low numbers in blood and tissues, so is

difficult to isolate. Dark field examination or culture of samples such as blood, CSF, urine or colostrum may provide a diagnosis. [2]

Serological tests such as the indirect immunofluorescent antibody test, an ELISA and Western blot analysis are used most frequently. Subclinical infections are common, so test results must be interpreted in conjunction with clinical findings. [2]

24.7 Immunity

A vaccine is available for use in dogs, but none is available for other domestic animals. [2]

24.8 Treatment

Infection may be treated by long courses of tetracyclines or penicillin.[2]

24.9 Risk assessment

24.9.1 Release assessment

The widespread distribution of infection, high serological prevalence in some areas, and potential for subclinical infection combine to suggest a relatively high probability of *B. burgdorferi* being introduced into New Zealand. Considering species susceptibility and trade patterns and volumes, the most likely route of introduction is probably humans, followed by dogs and horses.

24.9.2 Exposure assessment

Tick species capable of transmitting Lyme disease do not occur on livestock in New Zealand, and so transmission is unlikely to occur here. There is a remote possibility that urine/mucosal contact could directly transmit the disease to other animals in close contact.

24.9.3 Consequence assessment

The consequences associated with a case of Lyme disease in an imported horse are likely to be minimal, once trace back had determined the case was imported. The potential for cases in animals in close contact might require investigation, such as a serological survey of in-contact animals. Assuming there was no spread, trade consequences for exports of animals and animal products would be unlikely. The public health consequences would be most significant for persons investigating imported cases and those with occupational exposure to imported horses.

24.9.4 Risk estimate

On rare occasions horses infected with Lyme disease might be imported from endemic areas, but the infection would be unlikely to spread here. An imported case would probably not have significant adverse consequences. Specific safeguards are not warranted.

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25 Q fever

25.1 Aetiology

The causative organism of Q fever is the intracellular Rickettsia *Coxiella burnetii*. [1, 2, 3, 4]

25.2 Susceptibility

Q fever is a zoonosis. [1, 2, 3, 4]

Sheep, goats and cattle are the most common reservoirs for spread of *C. burnetii* to humans. Cats also shed the organism, especially at the time of parturition. Dogs have infrequently been associated with transmission to humans. [2, 5]

A large number of other animal species are susceptible to infection, including horses, donkeys, water buffalo, camels, pigs, rabbits, mice, pigeons, ducks, geese and domestic fowl.[6]

25.3 Distribution

C. burnetii is widely spread throughout the world, and the epidemiology of transmission to humans varies from country to country. [5]

C. burnetii is not present in New Zealand. [7]

25.4 Clinical signs

In areas where *C. burnetii* is common, infection of domestic animals generally does not cause any significant clinical signs, although abortions and stillbirths may occur in cattle, sheep and goats. [2]

The symptoms of Q fever in humans vary from country to country and may reflect strain differences. A self-limiting febrile illness is the common manifestation in Australia. In Canada the disease may be a pneumonia or granulomatous hepatitis. A chronic form leading to endocarditis also occurs with varying incidence in different countries. [5]

25.5 Transmission

The most important route of human infection is inhalation of contaminated aerosols which result from environmental contamination by reservoir animals, particularly at the time of parturition. Occupational exposure to parturient animals or contaminated aerosols is a common finding in many epidemiological studies into Q fever. Percutaneous inoculation and ingestion are also potential routes of infection. Person to person transmission has been documented, but it is very unusual. There is no evidence for vertical transmission. [5]

Transmission amongst animals is believed to occur within two cycles, one involving wild animals and ticks and the other involving direct infection. Some species of ticks are essential in specific wildlife

cycles, in particular ticks from the *Ixodidae* and *Argasidae* families. However, there are several locations where Q fever flourishes but ticks are rare or non-existent. [6]

Like most animals, horses are susceptible to infection.[2] Out of 121 samples of equine sera submitted to a veterinary hospital in California for unrelated purposes, 31 (26%) were positive for antibodies to Q fever.[8] Serological surveys have also demonstrated infection of horses in Canada.[9] However, there are no reports suggesting that horses have been a source of infection for humans or other animals, despite several reviews of the epidemiology of Q fever in a variety of countries. [5, 10, 11, 12, 13, 14] In all likelihood, horses are not significant in the epidemiology of Q fever.

25.6 Diagnosis

The organism may be demonstrated in smears made from infective material, particularly the foetus, placenta and vaginal discharges soon after abortion in rumunants. Staining, immunofluorescence and PCR are available to confirm the presence of *C. burnetii*. [6]

Serological tests such as the CFT, IFAT, MAT, capillary agglutination, and ELISA are available. The CFT is the most commonly used. [1, 6]

25.7 Immunity

There is an inactivated vaccine for use in cattle, sheep and goats. [1, 6]

25.8 Treatment

Domestic animals are typically not treated. [6]

25.9 Risk assessment

25.9.1 Release assessment

The prevalence of horses previously exposed to *C.* burnetii, as indicated by serological reactions, may be as high as 26% in specific populations. This is not unexpected considering that the organism is often common in environments where livestock are kept. The relevance of this observation to the actual infective status of horses is unknown. Like most other animals, horses probably do not show clinical signs of disease. There are no data indicating that horses shed the organism, or for how long.

25.9.2 Exposure assessment

Horses have not been considered to be a source of infections in humans or other animals, suggesting they probably do not play a significant role in the epidemiology of Q fever.

Suitable tick vector species do not occur on livestock in New Zealand, further mitigating the risk of spread through ticks should an infected horse be imported.

Q fever occurs in Australia. Approximately 500 horses are imported into New Zealand annually from that country, as well as significant volumes of horse semen. There is no suggestion that this trade has lead to cases of Q fever here.

25.9.3 Consequence assessment

The principal concern regarding Q fever introduction and establishment relates to the risk of human infections, which typically occur through occupational exposure. New Zealand has a policy of maintaining a Q fever free status so as to avoid the public health consequences of introduction.

Significant consequences in the livestock industries are considered unlikely unless control and/or eradication were attempted.

25.9.4 Risk estimate

Horses are not considered important in the epidemiology of Q fever. Imports of horses or semen do not present a significant risk of introducing Q fever into New Zealand. No specific measures are considered necessary.

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26 Equine ehrlichiosis

26.1 Aetiology

Equine ehrlichiosis is caused by infection with the Rickettsial organisms *Ehrlichia risticii* and *E. equi*. Infection with *E. risticii* causes Potomac horse fever (also known as equine monocytic ehrlichiosis). Infection with *E. equi* causes equine granulocytic ehrlichiosis. [1, 2, 3]

26.2 Susceptibility

Natural clinical disease due to infection with *E. risticii* occurs only in horses. Natural infections occur in dogs, and cats, mice and non-human primates have been infected experimentally. [3] Serological evidence of infection has also been found in pigs and goats.[2]

E. equi occurs naturally in horses. Dogs, cats, sheep, goats and monkeys may be experimentally infected. [3]

26.3 Distribution

Potomac horse fever is known to occur in the USA, Canada and Europe, and may occur more widely. The incidence of disease is sporadic and seasonal, with most cases occurring in summer and autumn. [3]

Equine granulocytic ehrlichiosis is known to occur in California and northeastern USA. A study involving 13 cases of naturally acquired disease in northeastern USA noted all cases occurred in October through December. [4]

E. risticii and *E. equi* do not occur in New Zealand.

26.4 Clinical signs

Most infections with *E. risticii* and *E. equi* are probably subclinical. Serological studies in the USA have demonstrated seroprevalences to *E. risticii* as high as 30% in at risk populations of horses.[2]

Potomac horse fever is uncommon in horses less than 1 year of age, although peracute disease can occur in foals. Clinical signs in adult horses vary from mild to severe, manifesting as fever, anorexia, depression, laminitis and diarrhoea lasting up to 10 days. Mortality is approximately 30%, and is associated with rapid dehydration and hypovolaemic shock as a result of diarrhoea. Surviving animals recover uneventfully. [1, 2, 3]

Equine granulocytic ehrlichiosis is characterised by a nonspecific illness of variable severity. There may be fever, depression, icterus, anorexia and limb oedema. [1, 4] The incubation period in experimental cases was 1-9 days. [1]

26.5 Transmission

Both *E. risticii* and *E. equi* can be transmitted experimentally through inoculation of blood from infected horses.[1, 3]

The mechanism of natural transmission for *E. risticii* is unknown, although it does not appear to be contagious. Like most other rickettsial organisms, *E. risticii* may be spread by arthropod vectors, although none have been identified. The risk of infection is greater for horses housed on premises with a history of animals with prior infection. *E risticii* has been experimentally transmitted to horses by the oral route, although there is no epidemiological evidence for a faecal-oral cycle of transmission. [3] Experimentally infected horses remained carriers of *E. risticii* for at least 40 days. [2] Infection in horses eventually leads to a sterile immunity, which suggests they are not a reservoir. Potential reservoirs may be dogs, rabbits and/or foxes. [3]

Although the transmission cycle of *E. equi* has not been defined conclusively, black-legged ticks, *Ixodes scapularis*, are the most likely vector in the northeastern USA. Pathogen load and duration of infection in horses is influenced by differences in the immune response of the infected animal.[4]

No reports of transmission via semen have been found for either organism.

26.6 Diagnosis

The organisms can be visualised in stained smears or cultured from blood taken during the febrile period. [1]

E. risticii and *E. equi* are serologically unrelated. [1] The antibody response can be detected by ELISA or IFAT. Serology should be interpreted in conjunction with clinical signs as titres remain high for long periods following infection. Paired samples demonstrating a rising titre may be necessary in endemic areas. [3]

In 13 confirmed clinical cases of *E. equi* infection in horses (confirmation by PCR identification of ehrlichial DNA in white blood cells, and a four-fold increase in antibody titre in paired serum samples), antibody levels were low in the early weeks of infection, increased to a peak between 19-81 days (mean 48.2, median 40 days) and declined by 183-215 days. [4]

E. risticii diagnosis by nested PCR, culture and IFAT was compared using samples from an experimentally infected pony, experimentally infected mice, and field samples from horses showing clinical signs compatible with Potomac horse fever. The IFAT titre became positive 6 days post-inoculation in the pony. Culture from the pony was positive from day 1-28 post-inoculation, and the nested PCR was positive from day 1-32. [5]

26.7 Immunity

Recovered animals demonstrate resistance to challenge by inoculation of infectious blood, indicating infection is probably followed by a sterile immunity for both *E. risticii* and *E. equi*.[1, 3] The

duration of the protective immunity is unknown.

Inactivated whole cell adjuvanted vaccines are available for Potomac horse fever. They require two doses 3 weeks apart with revaccination at 4 month intervals during the disease season. [3]

In the USA, 4.0% of establishments with horses less than 12 months of age, 11.0% of establishments with brood mares, and 18.0% of establishments with other horses over 12 months of age vaccinate against Potomac horse fever. [6]

26.8 Treatment

Oxytetracycline administered intravenously at a dose rate of 6.6 mg/kg is the treatment of choice, and horses treated early respond well. [3]

26.9 Risk assessment

26.9.1 Release assessment

Horses imported from endemic areas could be infected with *E. risticii* and *E. equi*, and the risk would be greater during summer and autumn months. The risk would increase for horses with recent pre-export exposure to other infected horses, such as if cases had occurred on the premises of origin. The endemic areas are not accurately defined at present.

Although high seroprevalence in endemic areas indicates high infection rates, the period of infectivity is relatively short and is followed by a sterile immunity, which reduces the likelihood of importing an infectious horse.

No information has been found suggesting transmission by artificial insemination.

26.9.2 Exposure assessment

Transmission cycles for both organisms require further definition. *E. equi* is transmitted by *Ixodes* ticks, and the same may be true of *E. risticii*. The lack of such ticks on livestock in New Zealand suggests the risk of imported cases leading to establishment here is low, although there would be potential for spread through iatrogenic transmission.

26.9.3 Consequence assessment

The consequences associated with imported cases would include the direct effects associated with clinical cases and control efforts. Clinical cases would probably occur only in horses, and consequences would be confined to the equine industries. Some measures might be imposed on live horse exports by trading partners, such as Australia, where *Ixodes* spp. ticks occur.

26.9.4 Risk estimate

Horses imported from endemic areas could have been infected with *E. risticii* or *E. equi*, but the risk of their being infectious at the time of importation is probably low. There is uncertainty regarding key aspects of these organisms' epidemiology, but the absence of *Ixodes* spp. ticks from New Zealand livestock probably mitigates the risk of establishment here. The consequences of introduction would be confined to the equine industries. Measures against *E. risticii* and *E. equi* during importation of live horses are probably warranted. In all likelihood semen does not present a risk.

26.10 Risk management

26.10.1 Risk management objective

Horses should be free from clinical signs of infection with *E. risticii* and *E. equi*, and have been protected from infection in the pre-export period. Requiring a 3 month period of freedom from cases on the premises of origin will provide some protection.

High seroprevalence and the likelihood of a sterile immunity following infection indicate that excluding seropositive horses is unwarranted.

26.10.2 Risk management measures

The endemic areas of both *E. risticii* and *E. equi* are not well defined, and so all imported horses should be subject to the measures.

Live horses

1. The horses were kept during the 3 months prior to export on premises where equine ehrlichiosis (*E. risticii* and *E. equi*) has not occurred during that period; *and*

2. The horses were showing no clinical signs of equine ehrlichiosis on the day of export.

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6.3 FUNGAL DISEASES

27 Epizootic lymphangitis

27.1 Aetiology

Epizootic lymphangitis is an OIE List B disease caused by infection with the dimorphic fungal soil saprophyte *Histoplasma capsulatum* var. *farciminosum* (previously *Histoplasma farciminosum*). [1, 2, 3, 4]

27.2 Susceptibility

The disease affects horses and mules, and less commonly donkeys and camels. [1, 3, 4] In rare instances cattle and humans are reported to have been infected. [4]

27.3 Distribution

Although prevalent in the early twentieth century, particularly when large numbers of horses were grouped together for military operations, the disease is considered rare today. [5] However, exposure to the causative organism may be more common. [6] *H. farciminosum* is endemic in countries bordering the Mediterranean, particularly Italy and North Africa. It is also found in Central and Southern Africa, and in regions of Asia and Russia. [6]

Epizootic lymphangitis has never been recorded in New Zealand.

27.4 Clinical signs

Epizootic lymphangitis is a contagious, chronic disease of horses that persists for 3-12 months. Disease tends to occur as outbreaks. [4]

Clinical disease occurs as cutaneous, respiratory, ocular or asymptomatic forms. [6] The cutaneous form occurs most often in the lower limbs. The organism establishes subcutaneously leading to ulcers, then invades via the lymphatics causing them to thicken. There is swelling in the regional lymph nodes, and the lymphatic lesions may burst and discharge pus. [1, 2, 3, 4, 6] In the respiratory and ocular forms the pus-filled lymphatic lesions extend along the conjunctiva and nasal mucous membranes to the pharynx and trachea.[1, 2, 6]

Mortality does not exceed 10-15%. Recovered animals may become asymptomatic carriers, but such animals can usually be identified by fibrocalcific skin lesions at sites of healing. Such horses are positive to intradermal tests and serology. [6]

27.5 Transmission

The mode of transmission is not well established.[6] The skin form occurs when contaminated soil contacts traumatised skin. The conjunctival form is believed to be spread by flies in the genera

Musca and *Stomoxys*. Fomites such as grooming equipment may also spread the disease. [1, 3, 4]

27.6 Diagnosis

The organisms can be cultured or visualised in stained smears made from pus. [1] Serological diagnosis can be made by fluorescent antibody, agar gel immunodiffusion, ELISA, and haemagglutinating tests. A skin hypersensitivity test is available to detect cell-mediated immunity. [1] The fluorescent antibody and skin hypersensitivity tests are probably the most accurate and reliable. [6]

27.7 Immunity

Most animals that recover develop a solid immunity. [2] Vaccines are available and are widely used in endemic areas. [4] Vaccinated animals will be seropositive. [6]

27.8 Treatment

Treatment with intravenous sodium iodide or amphotericin B may be successful. [6]

27.9 Risk assessment

27.9.1 Release assessment

Horses imported from endemic areas could be infected with epizootic lymphangitis. However, the decreasing prevalence and distribution of the disease suggest the risk is low, and probably decreasing. The ability of veterinary services to monitor and control the disease will have a large bearing on the risk of exposed horses being exported. The disease runs a chronic course, and is associated with obvious clinical signs. As the organism is present in soil, contamination of hoofs or equipment could potentially also lead to its introduction.

There is no evidence to suggest that epizootic lymphangitis is spread by artificial insemination.

27.9.2 Exposure assessment

Direct and mechanical insect transmission suggest that if the organism were introduced in an infected horse or on soil-contaminated hooves or equipment, spread and establishment of the disease could occur in New Zealand. Establishment here would in part depend on the ability of the saprophytic stage finding suitable environmental conditions here.

27.9.3 Consequence assessment

Direct affects of introduction would result from clinical cases and from control efforts. The disease is chronic and so cases could take some time to manifest clinically. This could provide opportunities for spread on premises where imported horses were kept and create difficulties during the control effort for tracing in-contact horses. Trading partners would probably impose measures during exports of horses. Consequences would probably be confined to the equine industries, although there would be the possibility of infections in cattle and humans.

27.9.4 Risk estimate

Epizootic lymphangitis could be introduced through imports of live horses from endemic areas, leading to spread and establishment in New Zealand with adverse consequences for the equine industries. Measures during imports of live horses are warranted. Semen does not present a risk.

27.10 Risk management

27.10.1 Risk management objective

When horses are imported from areas where epizootic lymphangitis occurs they should be protected from infection during the period prior to export, and remain free from clinical signs of disease. Soil contamination of horses and equipment should be minimised.

The chronic and contagious nature of epizootic lymphangitis infection in horses, the obvious clinical signs (including the ability to identify recovered horses by the presence of healed lesions), and the tendency for disease to occur in outbreaks indicate that establishment of origin freedom from disease would provide a good assurance of protection in the pre-export period, provided the ability of veterinary services to detect and control cases is assured.

27.10.2 Risk management measures

Live horses

1. The horses were kept for the 3 months prior to export on premises where epizootic lymphangitis has not occurred during that period; *and*

2. The horses were showing no clinical signs of epizootic lymphangitis on the day of export.

References

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6.4 PROTOZOAN DISEASES

28 Piroplasmosis

28.1 Aetiology

Equine piroplasmosis (or equine babesiosis) is an OIE List B disease caused by the tick-borne protozoa *Babesia equi* and *B. caballi*. [1, 2, 3, 4] Within the family Piroplasmidae there are two genera: *Babesia* and *Theileria*. *B. caballi* is regarded as a true *Babesia*, because the intraerythrocytic division into two merozoites is typical of the genus. *B. equi*, however, frequently divides into four merozoites in erythrocytes and there is development in lymphocytes prior to the erythrocytic cycle, all in similarity with *Theileria* spp. [2]

28.2 Susceptibility

Equine piroplasmosis occurs in horses, mules, donkeys and zebra, but horses are most susceptible. [3]

28.3 Distribution

B. equi and *B. caballi* are widespread throughout tropical and subtropical zones, but *B. caballi* extends further north. Equine piroplasmosis is endemic in Africa, the Middle East, the Balkans, the countries of the former USSR, and Asia (excluding Japan). [4]

In Europe, equine piroplasmosis is not endemic in Ireland, the United Kingdom, the Netherlands, the Scandinavian countries and Germany. Most infections in these regions can be traced to imported horses. The disease is endemic in Portugal, Spain, France and Italy. Belgium, Switzerland, Austria, Poland and the Czech Republic are probably marginal areas.[4]

Both *B. equi* and *B. caballi* are endemic in Latin America, with the exception of southern parts of Chile and Argentina. *B. caballi* was introduced into Florida in 1959, and has become endemic in some adjacent states of the USA. [4]

Equine piroplasmosis has been recorded and apparently eradicated from Canada (last recorded case 1987) and Australia (1976). [5] The Australian cases resulted from imports of live horses from Texas in the 1950s and 1960s and from Spain on three occasions in the 1970s.[6]

Equine piroplasmosis has never been recorded in New Zealand.

28.4 Clinical signs

The incubation period is 12-19 days for *B. equi* and 10-30 days for *B. caballi*. [4] The disease may be peracute, acute, subacute or chronic. In the rare peracute form horses are found moribund or dead. In the acute form horses exhibit anorexia, depression and intermittent fever. There is anaemia, jaundice and haemoglobinuria, and some horses develop colic or petechial haemorrhages

on the conjunctivae. In the subacute form the clinical signs are mild and last only a few days. Horses that recover often develop chronic disease, with slow progressive loss of condition and transient fever. [4] Neonatal babesiosis in foals causes rapid onset of anaemia, severe jaundice and malaise. [3]

28.5 Transmission

Equine piroplasmosis is transmitted by ticks. The distribution and occurrence of the disease is dependent on the biology of these vectors in particular geographic regions. [4]

B. caballi is transmitted both trans-stadially and trans-ovarially by ten species within the genera *Rhipicephalus, Dermacentor* and *Hyalomma.* Ticks are considered the reservoir of infection. [4]

B. equi is transmitted by eleven species within the genera *Rhipicephalus, Dermacentor, Hyalomma* and *Boophilus.* Transmission only occurs trans-stadially, and so horses are considered the reservoir of infection. [4]

Infection is followed by parasitaemia. Parasitaemia in horses infected with *B. caballi* rarely exceeds an erythrocyte parasitism rate of 1% (and is often much lower). In *B. equi* infections the erythrocyte parasitism rate ranges from 1-7%, but may reach 80%. [2] Horses usually remain carriers of *B. caballi* for 1-3 years, but may remain carriers of *B. equi* for life. [4]

Piroplasmosis is also transmitted iatrogenically. When introduced into Australia, spread among horses occurred as a result of the use of non-sterile equipment for intravenous procedures. At the time, blood-letting was a popular practice. It appears that spread by ticks was not a feature of the disease in Australia, and so the infection eventually died out. [6] Iatrogenic transmission of *B. equi* has been described in a herd of horses and donkeys in the UK. The infection was introduced into the herd in a seropositive mare, and spread to 92% of all horses on the property over the subsequent 6 years. Transmission was suspected to have occurred through the use of the same syringe for collection of blood samples (even though new needles were used on each occasion). No health effects had been noticed during the spread of the infection. [7]

There are no reports of transmission of equine piroplasmosis through semen. [4]

28.6 Diagnosis

Direct microscopic examination of blood smears made from peripheral blood in the acute phase of infection may enable a diagnosis by experienced workers. The method has poor sensitivity because the parasitaemia is often at very low levels, particularly with *B. caballi* infections. [2] Diagnosis, particularly of the latent carrier state, requires serological detection of antibodies or antigen by a variety of immunoassays.

The CFT is widely accepted as the official test and is one of the tests prescribed for international trade by the OIE, the other being the IFAT. [1] The CFT detects antibodies from 8 days post-infection, with titres declining from 2-3 months during the chronic phase of infection. Problems with

the CFT include the anticomplementary action of sera from some horses and a proportion of false negatives because of fluctuations in the titres of complement fixing antibody. [2] The CFT titre of a single experimentally *B. equi*-infected horse was plotted for 40 weeks, and was shown to cycle between negative and positive readings. [8]

Seropositive animals will become negative to the CFT 3-15 months after the elimination of *B. caballi* and 24 months after the elimination of *B. equi*. [4]

The IFAT is used in cases where CFT is inconclusive, as it is more sensitive. The test requires considerable experience to interpret, and reading results is time-consuming. There are also practical problems with the production and storage of reagents for both the CFT and IFAT, which has lead to research efforts focussing on development of ELISAs. [1, 2, 4]

The monoclonal antibody EMA-1 was used to develop a competitive inhibition ELISA for *B. equi*. The c-ELISA was found to be sensitive and specific for antibodies to a range of isolates, and showed good agreement (90-94%) with the CFT. [1, 8]

An ELISA using recombinant *B. caballi* antigen for serodiagnosis of the *B. caballi* latent carrier state has also been developed. The recombinant ELISA, CFT and IFAT were tested on known-positive (n= 100) horses at day 10 following experimental infection and known-negative (n= 82) horses. The sensitivity and specificity of the recombinant ELISA were found to both be 100%. The sensitivity of the CFT was 47% and the IFAT 98% in this experiment. [9]

28.7 Immunity

No vaccines are available. [1] Maternal antibodies persist for 1-4 months after birth. [4]

28.8 Treatment

While a number of drugs are available to treat disease, none are satisfactory for the eliminating *B. equi* infections. *B. caballi* is more susceptible and can be eliminated. The most favoured regimes use multiple doses of imidocarb diproprionate at 5mg/kg at 72 hour intervals. [2]

28.9 Risk assessment

28.9.1 Release assessment

Horses imported from endemic areas could be infected with piroplasmosis. Imports from countries outside the endemic areas also present a risk, unless those countries have policies that exclude imports of seropositive horses. The prevalence of infection in endemic areas is unknown. Infection may be subclinical, or the clinical signs mild. Chronic infection is very common, and so any previously infected horse (as reflected by serology) should be considered to potentially be a carrier of piroplasmosis.

Imports of semen do not present a risk.

28.9.2 Exposure assessment

The only tick species in New Zealand that commonly occurs on livestock is *Haemaphysalis longicornis*. No *Haemaphysalis* sp. is known to act as a vector of equine piroplasmosis. Infection is unlikely to establish and spread naturally under New Zealand conditions.

If an infectious horse were imported iatrogenic spread would be a possibility. There is a greater awareness of and compliance with hygienic requirements for procedures that carry a risk of disease transmission amongst veterinarians today, so iatrogenic routes may be less important than in the past. However, as horses probably remain infected for life, the potential for iatrogenic transmission at some stage in the horse's life must be considered.

28.9.3 Consequence assessment

A single imported case of equine piroplasmosis would have minimal direct consequences. A limited investigation to demonstrate that transmission had not occurred would probably be required. If absence of transmission were demonstrated, control measures would concern only the infected horse.

New Zealand's importation policy with respect to piroplasmosis could affect the conditions imposed by other countries on our equine exports. In particular, Australia excludes imports of piroplasmosis seropositive horses. Australia has endemic tick species, such as *Boophilus microplus* and *Rhipicephalus sanguineus*, which are potentially capable of transmitting piroplasmosis. [(6)] Allowing seropositive horses to be imported into New Zealand would probably result in measures being imposed on horses exported to Australia, and perhaps other countries also. There is also the potential for the presence of seropositive horses in New Zealand to adversely affect the export trade in equine serum products.

28.9.4 Risk estimate

Imported horses could be infected with equine piroplasmosis, and the risk is not necessarily confined to areas where the disease is endemic. The disease would be unlikely to spread in New Zealand, limiting the likely direct consequences. However, indirect consequences for the equine industries would include measures during exports of live horses, including exports to Australia. Considering the importance of this trade, measures are warranted to ensure horses are not infected when imported. Imports of semen do not present a risk.

28.10 Risk management

28.10.1 Risk management objectives

All horses imported from countries where the disease is endemic or that permit the importation of seropositive horses should be serologically tested prior to importation, and permanent entry

restricted to seronegative horses. Testing and pre-export preparation should occur within facilities that preclude contact with ticks.

The CFT is the test prescribed by the OIE for international trade, despite poor sensitivity. The IFAT, while more sensitive than the CFT, has practical problems that have prevented its application as a general screening test. The c-ELISA for *B. equi* and the recombinant ELISA for *B. caballi* are promising tests with acceptable sensitivity, but may not be widely available. Each of the available tests have potential disadvantages, so it would seem reasonable to provide exporting countries a degree of flexibility to select which will be applied based on their own routine diagnostic practice. The tests should be applied after a 10 day period of isolation to allow any recently infected horses time to seroconvert.

Insect-vector proof isolation facilities are not considered necessary to preclude contact with ticks. In the absence of livestock movements, ticks are unlikely to move into a fenced facility. Exclusion of ticks could be achieved through prophylactic treatment during entry of animals to the facility, and isolated horses checked for the presence of ticks during veterinary inspections.

Infections in other horses on the premises of origin may indicate the presence of infected ticks, and may increase the risk of iatrogenic infection. A 3 month period of disease freedom provides an assurance in both respects.

28.10.2 Risk management measures

Determining whether measures for piroplasmosis are required for imports from any particular country will require examination of that country's health status and importation policies. Measures should be applied if disease is known to occur or if unrestricted importation of seropositive horses is permitted.

The temporary importation of seropositive competition horses has been considered by authorities in some countries/regions free of the disease, such as prior to the Atlanta Georgia Summer Olympics in 1996 and the Sydney Summer Olympics in 2000. As there is probably no risk of natural transmission under New Zealand conditions, the risk associated with seropositive horses imported for competition purposes may be managed by reducing the risk of iatrogenic transmission, and by ensuring such horses do not receive biosecurity clearance and are eventually re-exported. No special precautions to exclude such horses from contact with ticks in New Zealand are necessary, because the ticks in this country are not capable of transmitting the disease. These measures are further discussed in Section 8 *Temporary importation of competition horses*.

Live horses

Either:

1. Immediately prior to export the horses were resident in a country where equine piroplasmosis does not occur and that does not permit the importation of seropositive horses.

Or:

1. The horses were kept for the 3 months prior to export on premises where equine piroplasmosis has not occurred during that period; *and*

2. The horses have undergone a minimum 10 day period of pre-export isolation. Ticks have been excluded from the isolation facility through prophylactic treatment of all horses upon entry, and absence of ticks has been monitored through regular inspections of isolated horses; *and*

3. Not less than 10 days after entering pre-export isolation the horses have been tested for equine piroplasmosis with a negative result for both *B. equi* and *B. caballi* using the CFT (positive is less than 25% lysis at dilution of 1:5), IFAT, or an approved ELISA ELISA (with the exception of competition horses temporarily imported under special conditions); *and*

4. The horses were showing no clinical signs of equine piroplasmosis on the day of export.

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29 Dourine (*Trypanosoma equiperdum*)

29.1 Aetiology

Dourine is an OIE List B venereal disease of Equidae caused by the protozoan tissue parasite *Trypanosoma equiperdum*. *T. equiperdum* differs from other trypanosomes in that it rarely invades the blood. *T. equiperdum* is closely related to *T. evansi*, the cause of surra. [1, 2, 3, 4]

29.2 Susceptibility

Dourine is a disease of horses, donkeys and mules. There is no known reservoir of infection other than equids. Subclinical infections occur commonly in donkeys and mules, whereas horses are more susceptible to clinical disease. [1, 2, 3, 4]

29.3 Distribution

Dourine spread around the world with the movement of horses for breeding but has since been eliminated from many countries and now has a shrinking distribution. [3, 4] Dourine occurs in Africa, Russia and Asia. [3] Italy reports infection, [5] but most other European countries have eradicated the disease. [3] The USA eradicated dourine in 1934, [5] but the disease still occurs in parts of South America. [2]

Dourine has never been recorded in New Zealand.

29.4 Clinical signs

The clinical manifestation varies considerably. The incubation period may be from one week to a few months. Parasitaemia occurs 21-23 days after infection and may persist for 2-4 months. [1]

The disease assumes one of three forms: asymptomatic (or latent), interstitial (or oedematous), or nervous. The asymptomatic form occurs commonly in donkeys. Animals remain subclinical but seropositive and able to transmit infection for several years. The interstitial form is the manifestation of chronic disease. There is chronic weight loss, general debilitation and susceptibility to secondary infections. Early signs may include mucopurulent discharges from the urethra in stallions and vagina in mares, followed by gross oedema of the genitalia. Nervous disorders occur in some cases after the onset of emaciation or oedema. The typical manifestation is hyperaesthesia of the skin followed by a decrease in sensory and motor nerve activity, leading to stiffness, weakness and/or lameness. [1]

Pregnant mares occasionally abort.[1]

Dourine is a fatal disease, with an average mortality of 50%. The mortality rate in stallions is higher than mares. Spontaneous recovery may occur. The disease is marked by stages of exacerbation, tolerance and relapse that vary in duration and may occur once or several times before death or recovery. [1]

29.5 Transmission

Dourine is unlike all the other trypanosomoses in that it is not transmitted by arthropod vectors. Dourine is a true venereal disease involving both stallion-to-mare and mare-to-stallion transmission. [3]

As trypanosomes are not continuously present in the genital tract throughout the course of the disease, transmission does not occur at every copulation involving an infected animal. The parasite is present in the seminal fluid and mucous exudate of the penis and sheath of infected males, and in the vaginal mucus of the infected female. Following transmission at coitus, parasites invade the tissues of the genital tract. They may then pass into the blood and be carried to other parts of the body. [1]

Mare to foal transmission occurs, either at parturition or through ingesting the milk of an infected dam. Infected foals remain serologically positive and may transmit infection once sexually mature. [3]

29.6 Diagnosis

Diagnosis is based upon recognition of the clinical signs, identification of the parasite, or serology.

Parasite identification may be difficult. The trypanosomes are present in the blood for a short period only, and then may be present in tissues at low numbers. Fresh aspirates from lesions and vaginal or preputial washings or scrapings are examined to reveal the motile trypanosomes. In areas where infection with other trypanosomes occurs, *T. equiperdum* must be distinguished in blood smears on the basis of morphology and motility. [1]

Serum antibodies are present in infected animals whether they show clinical signs or not. The CFT is the OIE's prescribed test for international trade of horses. [1] Infected animals become positive to the CFT 20-30 days after infection, and remain positive with fluctuating titres for up to 10 years. Anticomplementary activity of donkey and mule sera is a problem,[3] as are cross reactions with other trypanosomes. [1, 3]

IFAT and ELISA are also available for serodiagnosis. [1, 3] A c-ELISA has been compared to the CFT using 910 known negative, 35 known positive, and 153 sequentially obtained sera samples from experimentally infected horses. The sensitivity of the two tests was virtually identical. The c-ELISA is simple and convenient and is a useful alternative to the CFT. [6]

29.7 Immunity

There are no vaccines available. [1, 3]

29.8 Treatment

There is no effective treatment for elimination of the parasites. Some chemotherapeutants may benefit clinically-affected animals, however treatment is not recommended as animals will remain

carriers and able to infect other animals. [3]

29.9 Risk assessment

29.9.1 Release assessment

Horses and horse semen imported from endemic areas could be infected with *T. equiperdum*. The geographical distribution of dourine is apparently diminishing, but there is no information regarding the likely prevalence in endemic areas.

Once infected, horses may be considered carriers for life. Clinical signs vary, and may be mild. There is a reasonable probability that apparently healthy horses or semen collected from healthy stallions would introduce disease if imported from endemic areas.

29.9.2 Exposure assessment

Dourine is spread by sexual contact and so horses imported for breeding present a risk. Although dourine is not transmitted at every coitus, transmission would eventually occur and the disease could establish in horse populations here. Imports of infected semen probably have an even greater potential for widespread exposure.

Horses temporarily imported for competition purposes and not used for breeding do not present a risk of introducing dourine.

29.9.3 Consequence assessment

The consequences of introducing dourine would include significant direct and indirect effects. There would be clinical disease in infected horses, and any infected horse would be considered a lifelong carrier. Experience in other countries indicates control and eradication would be possible given the appropriate resources. Other countries would impose trade measures on exports of live horses and semen. As dourine only affects equids, the consequences would be confined to the equine industries.

29.9.4 Risk estimate

Imports of horses and semen from endemic areas could lead to introduction and establishment of dourine, with associated adverse consequences for the equine industries. Measures against the disease are warranted.

29.10 Risk management

29.10.1 Risk management objective

Horses and donor stallions of semen imported from endemic countries should be free from clinical signs of disease, tested negative for dourine, and protected from infection during the period from

prior to testing until export/collection.

Pre-export isolation is probably not required because the disease is not transmitted by direct contact. However, sexual contact with other horses not of the same tested health status should not occur during the period 30 days prior to testing until export/collection. Infected horses are likely to seroconvert during this 30 day period.

The CFT and the c-ELISA should be considered acceptable tests.

Direct examination of semen for motile trypanosomes is easily performed, and provides a further assurance. This is probably appropriate considering the increased exposure potential associated with imported semen.

29.10.2 Risk management measures

The OIE Code [7] establishes conditions for a country to be recognised as free of dourine. When importing from other countries, the Code recommends freedom from clinical signs on the day of export or collection, 6 months establishment of origin freedom from cases, and a diagnostic test with negative results prior to export or collection.

Measures to prevent sexual contact are an appropriate complement to the measures recommended by the OIE, and provide further assurance regarding protecting horses from infection.

Live horses

Either:

1. The horses were kept since birth, or for the 6 months prior to export, in a country that has been free from dourine for the past 6 months according to the criteria within Article 3.4.2.2. of the OIE Code.

Or:

1. The horses were kept for the 6 months prior to export on premises where dourine has not occurred during that period; *and*

2. The horses have not been naturally mated with horses not of equivalent health status during the period from 30 days prior to pre-export testing until the time of export; *and*

3. The horses were subjected to the CFT or c-ELISA for dourine with negative results prior to export; *and*

4. The horses were showing no clinical sign of dourine on the day of export.

Horse semen

Either:

1. The donor stallions were kept since birth, or for the 6 months prior to collection, in a country that has been free from dourine for the past 6 months according to the criteria within Article 3.4.2.2. of the OIE Code.

Or:

1. The donor stallions were kept for the 6 months prior to collection on premises where dourine has not occurred during that period; *and*

2. The donor stallions were subjected to the CFT or c-ELISA for dourine with negative results:
Either: i) prior to collection, and from the period 30 days prior to testing until collection has not naturally mated any mares not of the equivalent health status;
Or: ii) not less than 30 days following collection of semen for export; *and*

3. The donor stallions were showing no clinical sign of dourine on the day of collection; *and*

4. Semen for export has been examined microscopically prior to freezing and no trypanosomes were detected.

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30 Surra (*Trypanosoma evansi*)

30.1 Aetiology

Surra is an OIE List B disease caused by the protozoan parasite *Trypanosoma evansi*. *T. evansi* is closely related to and difficult to distinguish morphologically from *T. equiperdum* and the tsetse-transmitted trypanosomes [1, 2]

30.2 Susceptibility

T. evansi has a wide host range. It principally infects horses and camels, but donkeys, mules, deer, llamas, dogs, cats, cattle and buffalo may all be infected and develop severe disease. Occasional infections leading to mild or chronic disease occur in sheep, goats, pigs and elephants. [2]

30.3 Distribution

T. evansi is present in North Africa, the Middle East, countries of the former Soviet Union, India, China, South East Asia and South America. [2, 3] Surra has never been reported in South Africa, but has a restricted distribution in Angola and Zambia. [4] Surra has never occurred in New Zealand.

30.4 Clinical signs

Surra in horses, mules and donkeys is usually fatal. The incubation period is 5-60 days, and the disease may follow an acute, subacute or chronic course. Death occurs within 2 weeks in acute cases or up to 4 months in chronic cases. [2] An intermittent fever is directly associated with parasitaemia. There may be weakness, lethargy and oedema, petechial haemorrhages in the mucous membranes, or extravasation of blood in the mucocutaneous junctions at the eyelids, nostrils and anus. There may be alopecia or urticarial skin eruptions. The clinical signs progress, with loss of weight, anaemia, jaundice and eventual death. [1, 2]

The fatality rate in cattle is much lower than in equids. Death may occur up to six months after onset of signs, but most animals recover to become carriers. Deer also suffer chronic disease. [2, 3]

30.5 Transmission

T. evansi has a direct life-cycle with no intermediate host. Transmission is mechanical by biting insects of the genera *Tabanus*, *Stomoxys*, *Atylotus* and *Lyperosia*. There is no biological cycle within any of the known insect vectors, unlike the tsetse-transmitted trypanosomes. Surra is typically spread into previously free areas by movement of live animals. [2, 3, 5]

No information has been found indicating that transmission via artificial insemination occurs.

30.6 Diagnosis

The diagnostic method prescribed by the OIE for international trade is direct identification of the agent using Giemsa-stained thick and thin blood smears, or wet blood films that allow the motile organisms to be visualised. Sampling from deep vessels will increase the chances of visualising *T. evansi* during periods of low parasitaemia, but even so direct identification has poor sensitivity in chronic cases. Concentrating the organisms in the examined sample may increase the sensitivity, and can be achieved by centrifugation of blood and examination of the buffy coat layer where trypanosomes will be most numerous, if present. Haematological or biochemical tests demonstrating anaemia or flocculation are also described by the OIE. [1]

Inoculation of blood into mice (mouse sub-inoculation, or MSI) probably provides the most reliable method of diagnosis. The procedure involves intraperitoneal inoculation of 0.5 ml of blood into two mice, followed by tail-bleeding three times a week for 28 days and examination of smears or films to detect parasitaemia. [1, 2]

An antigen ELISA (ag-ELISA) and latex agglutination antigen tests are recent developments. Test systems developed for *T. brucei* can be expected to work equally well for *T. evansi*. [1]

Serological tests are available, including CFT, indirect haemagglutination, IFAT, ELISA or card agglutination tests (CATT). The OIE describes procedures for the ELISA and IFAT, but notes that all such systems require further evaluation and standardisation. [1]

Experimentally infected donkeys (n= 10) were tested using wet blood films, MSI, the ag-ELISA and two antibody detection methods, a CATT and a double immunodiffusion test (DID). Infection was consistently detected using the MSI as early as 48 hours after infection. Wet blood films also detected infection early, between 3-6 days post-inoculation. The ag-ELISA became positive between 7 and 9 days, and antibody tests between 10 and 14 days. The diagnostic efficacy of the MSI was described as 100%, followed by the ag-ELISA, then antibody detection and wet blood films. [6]

30.7 Immunity

No vaccines are available against surra. [1]

30.8 Treatment

While attempts have been made to treat surra using chemotherapeutants effective against other trypanosomes, they have proven toxic for horses and unable to completely clear infection. [3]

30.9 Risk assessment

30.9.1 Release assessment

Horses imported from endemic areas could be infected with surra. The prevalence in infected

countries is unknown. Although infection is usually fatal, there is a long incubation period and the disease may run a chronic course. Horses that are incubating or in the early stages of the disease may appear healthy. The likelihood of a horse imported from an endemic area being infected with *T. evansi* would be most dependent on the prevalence of infection in the exporting region.

Importation of semen does not present a risk of introducing surra.

30.9.2 Exposure assessment

The geographic distribution of surra indicates that, while tropical and sub-tropical climates are more favourable, infection may also establish and persist in temperate climates such as New Zealand's.

Stomoxys calcitrans, a competent mechanical vector of surra, is widely distributed in New Zealand. Uncovered ensilage stacks and a variety of other composting organic materials (including horse manure and straw) provide favourable breeding sites. The flies are most dense around such sites, and probably disperse only as far as required to obtain blood meals. Up to three meals may be required for the production of eggs. (7)

Susceptible host species, particularly horses, cattle and deer, are widely distributed here.

These factors combine to suggest that transmission of *T. evansi* could occur in New Zealand. The possibility that endemic infection may establish here cannot be excluded.

30.9.3 Consequence assessment

Introduction and establishment of surra would lead to significant direct and indirect consequences for the equine, cattle and deer industries. Direct consequences would result from disease effects and control measures. Control would be complicated by the potential for cross-species transmission, and eradication would incur significant costs. Indirect consequences resulting from trading partners imposing measures during exports of live animals are also likely.

30.9.4 Risk estimate

Imports of infected horses from endemic areas could lead to introduction and establishment of surra with significant adverse consequences potentially affecting the equine, cattle and deer industries. Measures are warranted to ensure infected horses are not imported. Imports of semen do not present a risk.

30.10 Risk management

30.10 1 Risk management objective

Horses imported from endemic areas should be tested negative for surra and protected from infection during the period prior to testing until the time of export. Horses should remain free from clinical signs of disease throughout a period of veterinary supervision equivalent to the incubation

period of surra.

Mechanical transmission by insects suggests proximity to infected animals is a risk factor. Premises of origin freedom from cases will reduce the likelihood that horses have been exposed to infection.

A period of insect-proof pre-export isolation is appropriate, during which time diagnostic screening should occur. The MSI is the most sensitive diagnostic procedure and will detect infection in the very early stages. A minimum 30 day period will allow blood samples to be taken not less than 48 hours after entry into isolation, followed by 28 days observation of the inoculated mice.

A period of post-arrival quarantine in insect-proof facilities would provide further assurance that horses cleared for entry are not infected with *T. evansi*. The incubation period of surra may be up to 60 days. A period of 30 days post-arrival quarantine will increase the likelihood of clinical disease manifesting at some point during the period of pre-export and post-arrival veterinary supervision, and provide an opportunity for direct examination of blood smears as a further assurance.

30.10.2 Risk management measures

Although surra is an OIE List B disease, the OIE Code does not make recommendations for safeguards during the importation of horses.

Live horses

Either:

1. The horses were resident since birth, or at least the previous 2 months, in a country which is free of surra.

Or:

1. The horses were kept during the 3 months prior to export on premises where surra has not occurred during that period; *and*

2. The horses have been subjected to a minimum 30 day period of pre-export isolation, and protected from insect vectors during this time and during transport to the port of departure; *and*

3. Not less than 48 hours after entering pre-export isolation the horses have been bled, and a 0.5ml sample of blood inoculated intraperitoneally into two mice per tested blood sample. The mice have been bled three times a week for 28 days and wet blood films examined for the presence of trypanosomes, with negative results; *and*

4. The horses were showing no clinical signs of surra on the day of export; *and*

5. The horses have been subjected to 30 days of post-arrival quarantine in an insect proof facility, during which time they have been bled on two occasions not less than 14 days apart

and wet blood films and thick and thin blood smears made and examined for trypanosomes, with negative results.

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31 Equine protozoal myeloencephalitis

31.1 Aetiology

Equine protozoal myeloencephalitis (EPM) is caused by a protozoan that normally cycles between opossums (*Didelphis virginiana*) and birds. The organism was named *Sarcocystis neurona* prior to its epidemiology being fully understood. Since the opossum has been identified as the definitive host, DNA testing and infection studies have shown that the organism is probably the parasite *Sarcocystis falcatula*. [(1)]

31.2 Susceptibility

Clinical disease occurs in horses when they become infected as an aberrant host. Standardbred and young horses may be more susceptible to disease. A wide variety of species are probably also susceptible to infection. [(1)] Seroprevalence studies indicate that donkeys and mules can be infected, although they are not known to be affected by EPM. [(2)]

The definitive host in the USA is the opossum *Didelphis virginiana*. [(1)] (Note that the possum which occurs in New Zealand, *Trichosurus vulpecula*, is a different species.)

31.3 Distribution

EPM has been identified in the USA, Canada, Panama, Brazil and Mexico.[(1)] Various surveys have indicated that approximately 45-53% of horses in some areas of the USA are seropositive. [(1, 2, 3, 4, 5)]

Horses imported into Hong Kong from the USA were demonstrated to have serological responses to EPM, but none showed clinical signs. [(6)]

31.4 Clinical signs

High seroprevalence probably indicates that most infections are subclinical. Horses may manifest a wide variety of CNS-related symptoms as a result of migration of the parasites through the CNS. The neurologic signs are most commonly asymmetric ataxia, paresis and spasticity, but may include lameness, airway abnormalities (resulting from paralysis of the nerves innervating the airways), muscle atrophy, back soreness or upward fixation of the patella. The time for development of clinical signs varies from a minimum of 2 weeks to up to 2 years. [(1)]

31.5 Transmission

Horses are infected by ingesting sporocysts while eating feed contaminated by opossum faeces. Horses represent an aberrant dead-end host and do not transmit *S. neurona* to other animals. [(1)]

31.6 Diagnosis

Ante-mortem diagnosis is difficult, and relies on interpretation of clinical signs, serology, and CSF antibody tests, such as the western blot analysis. [1, 7]

31.7 Immunity

The cellular immune response in the CNS is probably important in the pathogenesis of CNS disease in horses. The ability of the humoral response to clear organisms is unknown. [1]

31.8 Treatment

Treatment involves combinations of antiprotozoal and anti-inflammatory drugs for long periods. [1]

31.9 Risk assessment

31.9.1 Release assessment

Considering the high seroprevalence amongst horses in endemic areas, there is a strong possibility that an imported horse would be serologically positive or actively infected with *S. neurona*. An unknown proportion of these horses will eventually develop clinical signs of EPM. The long incubation period indicates clinical cases may occur up to 2 years after importation.

31.9.2 Exposure assessment

The horse is an aberrant dead end host for *S. neurona*. The definitive host, the opossum *Didelphis virginiana*, does not occur here. The risk of transmission and establishment in New Zealand is essentially zero.

31.9.3 Consequence assessment

Clinical cases in imported animals may lead to an exotic disease investigation. However, once a diagnosis was established no further action would be required and trading partners would be unlikely to impose additional measures on exports of horses.

31.9.4 Risk estimate

There is a relatively high probability that seropositive horses and occasional clinical cases of EPM could be imported from the Americas, but this would not lead to transmission or establishment of *S. neurona* here nor significant adverse consequences. EPM should be included in the list of differential diagnosis when neurological disease is suspected in horses imported from North America, but specific measures during imports of horses and semen are not warranted.

References

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6.5 METAZOAN DISEASES

32 Screwworm

32.1 Aetiology

Screwworm infestation is caused by larvae of two distinct species of flies: *Cochliomyia hominivorax* (New World screwworm) and *Chrysomya bezziana* (Old World screwworm). [1, 2, 3] Both are OIE List B diseases. [4]

32.2 Susceptibility

The screwworm is an obligatory parasite of all domestic and wild warm-blooded animals, including humans and birds. [1, 2, 3]

32.3 Distribution

The disease is of importance in the tropical and sub-tropical areas of Africa, the Middle East, Asia, North and South America. Pupal stages are unable to survive freezing, and ground temperatures below 10-15°C over consecutive months inhibit development and suppress overwintering. The optimal temperature range for adult flies is 20-30°C. [1, 2, 3, 4]

A major sterile insect release method campaign (SIRM) has eliminated *C. hominivorax* from the southern USA. [1, 2, 3, 4] However, proximity to infested areas in Central America and the Caribbean means intensive surveillance continues in those areas of the USA at risk of incursions. In October 1998 a *C. hominivorax* larva was removed from a wound on a goat in southwest Texas, prompting investigations which included checking 40,000 head of livestock. No further larvae were found. [5] SIRM has also successfully eradicated *C. hominivorax* in Mexico and Central America as far south as Panama. [4, 5]

The occurrence in Libya of previously exotic *C. hominivorax* was confirmed in January 1989. A combination of measures including movement controls, surveillance, prophylaxis and SIRM were eventually successful in eradication. [4]

In Asia, the range of *C. bezziana* includes the Indian subcontinent, South-East Asia and Papua New Guinea. [1, 2, 3] Unlike *C. hominivorax*, there is no large-scale sterile insect production facility for *C. bezziana,* although there is a pilot research facility in Malaysia.[4] Authorities in Australia consider *C. bezziana* a major threat because of its proximity, although the fly has never been recorded there. [3]

The range and prevalence of *C. bezziana* has been increasing in Persian Gulf countries in recent years. A large outbreak occurred in Iraq in 1996 and is still on-going. Iran and Kuwait have also reported outbreaks. [4]

32.4 Clinical signs

Strikes occur at any site of the body, but commonly the navel of the newborn, head, brisket, genital area, escutcheon and udder. Wounds from castration, de-horning and branding are prone, but minor lesions such as those resulting from a tick bite may also be struck. Strikes are deep seated and cause considerable tissue destruction. [1, 2, 3]

32.5 Transmission

The adult flies lay their eggs in shingle like clusters at the edges of fresh wounds. Larvae hatch in 12 hours, and penetrate the tissues surrounding the wound. They mature in 5-7 days, reaching a length of about 2 cm, and then leave the wound by falling to the ground. A cavity is created in the wound by larval feeding. Pupation on the ground is between 3 days and 2 months, depending on temperature. [1, 2, 3] The entire life-cycle can be completed in approximately 3 weeks in optimum conditions, or in cooler weather it can extend beyond 2 months. Aerial dispersal of adult flies may exceed 100 km. [4]

32.6 Diagnosis

Diagnosis is suspected on clinical signs in endemic or fringe areas. Larval specimens are required for laboratory diagnosis. [1, 2, 3]

32.7 Immunity

There is no immunity to disease, and untreated lesions may continue to be re-infested.[1, 2, 3]

32.8 Treatment

Treatment of wounds is by dressing with larvicide and antiseptic. Ivermectin 0.2 mg/kg subcutaneously kills all *C. bezziana* larvae up to 2 days old, and many older larvae. There is a residual protection of 16-20 days. [2]

32.9 Risk assessment

32.9.1 Release assessment

Importation of live horses from screwworm endemic and fringe areas presents a risk of introducing larvae in infested wounds. The incidence of infestation in at-risk areas is probably most dependent upon the control measures in place. Effective control can reduce the incidence of strikes to very low levels and eventually eradicate the flies, although re-infestation from neighbouring areas may occur. In areas where there is poor control, the incidence may be high. For instance, between August 1996 and July 1998 over 58,000 cases of *C. bezziana* infestation were reported in livestock in the endemic area of Iraq. [4] Intensive surveillance occurs in many at-risk areas, particularly where large-scale resources and effort have been invested in eradication programmes. Surveillance will provide a good indication of the level of risk.

The clinical signs of infestation rapidly become obvious. Horses with open wounds are particularly susceptible, but even very minor wounds may be infested.

Screwworm is not transmitted in semen.

32.9.2 Exposure assessment

New Zealand's temperate climate would limit transmission and distribution of introduced screwworm. It is very unlikely that any locality in New Zealand offers a climate which would allow screwworm pupal stages to survive over winter. Although readings of the soil temperature (10 cm depth at 9 am) are not collected at all climatological stations, they are available for Kaitaia aerodrome (latitude 35°C 04', at 80m above sea level). Even at such a northern location, mean soil temperatures are around or slightly below 10°C during the months June through August and occasional ground frosts are recorded (average of 1 or less days per month during winter). Further south, temperatures are lower and/or ground frosts more frequent.[6] However, many areas of New Zealand have climates that would allow the screwworm life-cycle to be completed during the summer months. The time of year and location of an introduction would have a large bearing on subsequent spread.

An Australian study investigated the probability of *C. bezziana* establishment throughout the year for several locations around Australia's coastline. The effects of time of year, climate, vegetation and the number of incoming flies or larvae were investigated for four locations: the tip of Cape York, Brisbane, Wyndham and Fremantle. The study concluded that in southern areas the cold winters would limit survival, and populations would not survive over winter. Favourable conditions for year round survival and breeding exist in northern areas of Australia. [7]

32.9.3 Consequence assessment

In the event of an introduction of screwworm leading to an outbreak during the summer months, significant adverse direct and indirect impacts could affect many livestock industries. The outbreak would adversely affect health and productivity in the important cattle and sheep-based agricultural sectors, and restrictions on exports of live animals would be likely. There would also be public health issues to consider. Establishment here is very unlikely because ground temperatures are too cold to allow screwworm pupae to survive over winter.

32.9.4 Risk estimate

Importation of live horses from areas where screwworm occurs could result in introduction. Horses with open wounds present the greatest risk. The risk will vary considerably according to the level of surveillance and control being applied in the exporting area, and the time of year of the export. The time of year and location of the introduction would determine whether transmission occurred, but establishment (overwintering) is very unlikely. Screwworm represents a significant threat with regard to the consequences to New Zealand of an outbreak. Measures during importation of live horses are warranted.

32.10 Risk management

32.10.1 Risk management objective

Horses imported from areas at risk of screwworm should be examined for infestation, be free from clinical signs, and be protected from infestation during the pre-export period.

The occurrence of screwworm in domestic or wild animals or humans at any time during the previous 12 months should be considered to indicate a risk in the exporting region. The ability for wide dispersal of the flies indicates appropriate caution should be exercised in determining the boundaries of at-risk areas.

A minimum 7 day period of pre-export isolation when importing from at-risk areas will allow clinical manifestation of any prior infestation. All horses moving into isolation should be inspected, with particular attention paid to any open wounds, and they should receive prophylactic treatment using an insecticide. Prophylactic insecticide treatment will provide appropriate protection from infestation without requiring isolation facilities to be insect-proof.

32.10.2 Risk management measures

The OIE Code [8] recommends measures for screwworm that include inspection of open wounds for infestation, prophylactic treatment of open wounds with oily larvicides, and prophylactic treatment of all live animals. These measures are repeated at entry into pre-export isolation and immediately prior to export. On arrival, horses should be inspected for infested wounds, and transport and quarantine bedding gathered and burned.

The OIE recommended measures are appropriate to meet the objectives discussed above.

Live horses

Either:

1. The horses were resident for at least the 21 days prior to export in a country or region that has not reported cases of screwworm during the previous year.

Or:

1. The horses were examined, found to be free of screwworm infested wounds, and treated with a prophylactic insecticide during the 48 hours prior to entering pre-export isolation; *and*

2. The horses were subjected to a minimum 7 day period of pre-export isolation, during which time any horses with wounds were monitored for signs of screwworm infestation; *and*

3. The horses were examined, found to be free of screwworm infested wounds, and treated with a prophylactic insecticide during the 48 hours prior to export.

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33 Warble fly myiasis

33.1 Aetiology

Warble fly infestation is caused by larvae of two species of flies, *Hypoderma bovis* and *H. lineatum.* [1, 2] There are other related species, but these have not been recorded on horses. [1]

33.2 Susceptibility

H. bovis and *H. lineatum* infect cattle, deer and occasionally horses. [1, 2]

33.3 Distribution

H. bovis and *H. lineatum* occur in the USA, Canada, Europe and Asia between 25° and 60° longitude. Infestations south of the equator are rare, although warble flies are known to occur in Chile. [1]

Warble flies have been eradicated from Norway, Sweden, Denmark and Malta, and their prevalence is very low in Ireland and Cyprus. [1]

An eradication campaign has been operating in Great Britain. The disease is notifiable, and surveillance using the ELISA to detect antibodies and random checks on imported cattle have played an important role. During the winter of 1996/97, a total of 200,153 sera from 5906 herds were tested, with no positives detected. These results suggest that warble fly is absent from Great Britain. [3]

33.4 Clinical signs

The larvae develop in tissues and cause obvious, painful soft swellings about 3 cm in diameter. Heavily parasitised animals may show poor growth, condition and production. Involvement with the spinal cord may cause sudden onset of posterior paralysis. [1, 2]

33.5 Transmission

Adult flies are active in spring to late summer. The eggs are laid on the legs, underbelly or rump of the animal. The eggs hatch and larvae penetrate the skin where they undergo development through two larval instars in subcutaneous tissues, finally emerging 2-3 months later and falling to the ground. In the ground they pupate and emerge as adult flies in 3-5 weeks. The timing of the fly season varies with location and climatic conditions. [1, 2]

33.6 Diagnosis

Diagnosis can be made on clinical signs or by identification of larvae.[1, 2] An ELISA to detect antibodies has been developed to screen cattle. [3]

33.7 Immunity

A vaccine using crude larval extracts has been developed for cattle, and may reduce the number of warbles and their development in tissues. (1)

33.8 Treatment

Organophospate insecticides destroy migrating larvae. Ivermectin kills all larvae and has 4 weeks residual effect. (1)

33.9 Risk assessment

33.9.1 Release assessment

Importation of live horses from endemic regions could lead to introduction of warble flies. The prevalence of infestation in the exporting area would have a large bearing on the risk, but is probably unknown in most areas. The distinctive clinical signs would probably be detected during pre-export preparation of horses, with the exception of early-stage infestation.

Semen does not present a risk.

33.9.2 Exposure assessment

Establishment and spread of warble fly in temperate regions such as Chile, Great Britain and Norway suggest that this would be possible under New Zealand conditions if infested animals were released.

33.9.3 Consequence assessment

Significant direct and indirect impacts would be likely for livestock industries, in particular the cattle industries. Productivity would be affected, and the value of hides and skins from infested animals would be significantly reduced. Measures during live cattle exports would probably be imposed by trading partners.

33.9.4 Risk estimate

Live horses imported from endemic areas could be infested with warble flies and this could lead to establishment and spread here with significant adverse consequences. Measures during imports of live horses are warranted. Semen does not present a risk.

33.10 Risk management

33.10.1 Risk management objective

Horses showing clinical signs of infestation should not be exported. All horses should be protected

from infestation during the pre-export period.

Mandatory prophylactic treatments during pre-export preparation will provide appropriate protection by ensuring imported horses are not carrying viable larvae.

33.10.2 Risk management measures

Live horses

Either:

1. The horses were resident since birth, or at least the previous 3 months, in a country or region that has not reported cases of warble fly during the previous year.

Or:

1. The horses were treated with an ectoparasiticide capable of killing warble fly larvae during the 48 hours prior to export; *and*

2. The horses were showing no clinical signs of warble fly infestation on the day of export.

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1 Radostits OM, Blood DC, Gay CC. Veterinary Medicine. Eighth edition. Bailliére Tindall, London, England. 1282-1284. 1994.

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7 POST-ARRIVAL QUARANTINE

7.1 PAQ risk management

Post-arrival quarantine (PAQ) can be used following importation of animals to extend the isolation period, thereby reducing the likelihood that they will be infectious at the time of release, and to provide an opportunity for further testing. PAQ reduces the likelihood of dissemination of diseases introduced with imported animals into the general population. An outbreak of exotic disease in PAQ is more easily contained, and trading partners are less likely to impose additional measures on exports if cases occur in imported animals while in PAQ, rather than following release into the general population.

MAF does not currently require imported horses to undergo PAQ. This risk analysis has recommended PAQ as a risk management measure for a number of diseases for which a high level of protection is considered appropriate. In particular, PAQ is recommended whenever measures prior to export can not be relied upon to detect every infected horse; where rapid spread of infection could occur, leading to establishment here; and/or where significant adverse consequences could follow disease introduction and establishment.

7.2 MAF PAQ standards

MAF import health standards are able to specify the level of security of PAQ facilities, the duration of PAQ, and the procedures (such as tests and treatments) applied.

MAF does not provide facilities for PAQ. Any individual or company may apply to have a facility registered under the Biosecurity Act 1993, section 39. In order to become registered, the facility must be built, operated and supervised according to technical standards issued by MAF. There are three MAF standards for livestock PAQ facilities, providing high, medium and low levels of security.

7.2.1 *MAF Standard 154.02.15 Standard for high security farm animal quarantine facility.*

- outlines quarantine security measures to contain diseases spread by aerosols, arthropod vectors, and direct routes.

7.2.2 *MAF Standard 154.02.14 Standard for medium security farm animal quarantine facility.*

- outlines quarantine security measures to contain diseases spread by arthropod vectors (flies, mosquitoes, ticks), and by direct routes.

7.2.3 *MAF Standard 154.02.13 Standard for low security farm animal quarantine facility.*

- outlines quarantine security measures to contain diseases spread by direct routes such as faecal contamination, direct animal to animal contact, and on fomites.

7.3 Disease recommendations

This section summarises the risk assessment considerations for specific diseases with regard to

PAQ, and notes recommendations for the level of security, duration, and any procedures.

7.3.1 African horse sickness

Pre-export measures reduce the likelihood of imported cases of AHS, and the absence of *Culicoides* spp. here makes spread or establishment very unlikely. However, AHS is an OIE List A disease, and an imported case could lead to New Zealand losing the status of an AHS-free country. PAQ is warranted because of the potential indirect consequences for exports of horses and semen. Notwithstanding recommended pre-export measures, a 14 day period would provide further assurance that horses infected prior to export were no longer infectious at the time of clearance. In the absence of competent vectors here, low security PAQ would provide appropriate security, and no testing or treatment procedures other than clinical examination prior to release are considered necessary.

7.3.2 Vesicular stomatitis

There is significant uncertainty regarding the epidemiology of VS. The likelihood of the disease becoming endemic here is considered remote, although the possibility for limited direct spread or mechanical transmission by mosquitoes and biting flies must be considered. VS is an OIE List A disease, and trade consequences from an imported case would affect many agricultural exports, including cattle and cattle products. A 21 day period of medium security PAQ is appropriate, although no testing or treatment procedures other than clinical examination prior to release are considered necessary. This measure is only recommended when horses are imported from at-risk countries where health surveillance systems able to provide rapid diagnosis and monitoring of outbreaks are not in place.

7.3.3 Venezuelan equine encephalomyelitis

Horses are amplifying hosts for VEE, and the virus may be transmitted by a wide variety of mosquitoes. The establishment of endemic cycles in New Zealand is considered very unlikely, but there would be significant public health concerns and trade impacts associated with an imported case. The viraemia in horses is of short duration, so a 7 day period of medium security PAQ would ensure horses are not infectious at the time of release.

7.3.4 Equine infectious anaemia

Infected horses may remain seronegative to EIA for several weeks. To alleviate the higher risk of undetected infection in the pre-export period, repeating the serological test during a 7 day period of PAQ is appropriate when importing horses from medium to high prevalence countries. A 100 m spatial barrier between quarantined and other horses would reduce the chance of transmission by flies, and should be a specific fencing requirement for low security PAQ facilities for horses.

7.3.5 Equine influenza

EI is a highly contagious disease that would spread rapidly through New Zealand's equine

populations causing significant adverse consequences in associated industries. A 14 day period of PAQ would reduce the likelihood of infectious horses being released, and would probably provide the best opportunity for containment in the event of an outbreak in imported horses. Although EI is spread by aerosol over short distances, low security PAQ is appropriate so long as a 100 m spatial barrier is maintained between quarantined and other horses. Any horse exhibiting clinical signs of respiratory disease should be stabled in-doors and subjected to an antigen ELISA test for EI virus.

7.3.6 Surra

Surra has a long incubation period, up to 60 days, and is difficult to diagnose in the early stages of infection. Cattle, deer and horses may become infected, and an imported case could affect exports of livestock. Species of biting flies capable of spreading the disease are present here, and so medium security PAQ is appropriate. A 30 day isolation period prior to export is recommended, and a similar duration PAQ would provide a total isolation equivalent to the maximum incubation period. Examination of blood smears during PAQ would increase the likelihood of detecting subclinical infections.

7.4 Summary of PAQ recommendations

7.4.1 High security PAQ

No equine diseases of concern warrant high security post-arrival quarantine.

7.4.2 Medium security PAQ

Importation of horses from areas where the following diseases occur should require a period of medium security PAQ (minimum duration indicated in brackets):

- vesicular stomatitis (21 days);
- Venezuelan equine encephalomyelitis (7 days);
- surra (30 days).

This measure will be likely to affect imports of horses from the following areas, based on current health status (Appendix 1): Central and South America, Africa (except South Africa), the Middle East and Asia (excluding Japan).

7.4.3 Low security PAQ

Low security PAQ is recommended for imports from areas where the following diseases occur:

- African horse sickness (14 days);
- equine infectious anaemia, medium to high prevalence countries (7 days);
- equine influenza (14 days).

This measure will affect imports of horses from the following areas, based on current health status

(Appendix 1): North America, Europe, South Africa and Japan.

8 COMPETITION HORSES

8.1 Background

Horses are moved internationally to compete in racing and sporting events, and these movements have increased substantially in the last 5-10 years. Competition horses are unlikely to accept invitations to travel if health restrictions, in particular isolation requirements, mean they are unable to arrive at their destination in full fitness. Pre-export isolation and post-arrival quarantine premises rarely provide appropriate facilities to maintain a high level of fitness. For this reason, state veterinary authority inflexibility with quarantine requirements is a major disincentive to travel for competition horses. (1)

Competition horses are typically high performing animals that are subject to continuous veterinary supervision and often kept isolated from the general population of horses. The OIE Code has recommended a model passport for international movement of competition horses.(2) The model passport provides for full identification, movement records, vaccination records, and laboratory health tests. These factors may reduce the health risk posed by competition horses by providing a higher level of confidence in assurances verified by reference to animal health records.

If imported on a temporary basis for the sole purpose of competition or performance, horses present a lower risk of introducing venereal diseases than horses imported for breeding. Rather than ensuring freedom from such diseases at the time of importation, there may be alternative approaches to risk management such as restricting post-arrival contact, in particular sexual contact, with other horses. Similarly, allowing the temporary importation of horses that would otherwise be ruled ineligible may be acceptable for diseases such as equine piroplasmosis, which are spread by arthropod vectors that do not occur in New Zealand.

In the examples discussed, horses represent the only susceptible host. Other livestock industries and public health would not be put at risk by allowing untested or known-positive horses to be imported for competition purposes.

8.2 Diseases spread by venereal routes

8.2.1 Equine viral arteritis

While geldings and mares may be acutely infected, persistence of EVA occurs only in stallions. All horses may transmit the virus in the acute phase so measures to manage this risk should be applied for all horses. However, transmission from persistently infected stallions is by the venereal route only, and so transmission could be avoided if breeding (both natural service and collection of semen for artificial insemination) did not occur.

All horses are tested for negative, stable or declining antibody titres to EVA prior to export to ensure that respiratory shedding is not occurring at the time of import. The same measure is appropriate for competition horses. Seropositive stallions are further tested by collection of semen for virus isolation. This procedure should not be required when stallions are imported for

competition purposes, if the importation is temporary and post-arrival restrictions on use for breeding are imposed.

8.2.2 Contagious equine metritis

All horses may be persistently infected with CEM, including foals. However, transmission is by the venereal route only and so geldings are not considered to present a risk. Neither would mares and stallions present a risk if breeding did not occur and basic hygienic precautions were observed during any procedure which could potentially contaminate fomites with secretions from the reproductive tract e.g. tail bandaging, veterinary examination.

Testing mares and stallions for CEM prior to export requires a series of swabs and culture to isolate the causative organism, *Taylorella equigenitalis.* These procedures should not be required when mares and stallions are imported for competition purposes, if importation is temporary and post-arrival restrictions on use for breeding are imposed.

8.2.3 Dourine

All horses may be persistently infected with dourine. There is no treatment, although spontaneous recovery may occur. Transmission occurs at coitus, or from mares to foals, and so preventing breeding will effectively prevent the spread of the disease.

Testing horses for *Trypanosoma equiperdum* prior to export is performed by serology using CFT or c-ELISA. These tests should not be required when mares and stallions are imported for competition purposes, if importation is temporary and post-arrival restrictions on use for breeding are imposed.

8.3 Diseases spread by exotic arthropods

8.3.1 Equine piroplasmosis

Equine piroplasmosis is transmitted by ticks, or iatrogenically through needles and syringes. Infected horses remain long-term, often life-long, carriers.

Importation of horses positive for piroplasmosis was considered by the organizers of equestrian events for the Summer Olympic Games in Atlanta, USA, in 1996 [(3)] and in Sydney, Australia, in 2000.[(4)] In both cases, analysis concluded the risk of piroplasmosis introduction through seropositive horses could be managed through measures including site surveillance for ticks, isolation of positive horses, and prophylactic insecticide treatments.

Haemaphysalis longicornis, the only tick species that occurs on livestock in New Zealand, is not considered to be a competent vector for either *Babesia equi* or *B. caballi.* The only realistic risk of transmission here would be through iatrogenic means. Serological testing of competition horses for *Babesia equi* or *B. caballi* prior to export is not considered necessary, so long as the importation is temporary. Post-arrival restrictions should ensure that all persons performing veterinary procedures

such as venepuncture take appropriate hygienic precautions. Measures to prevent contact with ticks are also not considered necessary in the New Zealand context.

8.4 Isolation requirements

Recommendations for pre-export and post arrival isolation have been made for specific diseases. Extended periods of isolation create a disincentive for visits by competition horses, because such horses must maintain a level of fitness throughout the import procedure. Providing exercise facilities within isolation facilities to accommodate the needs of all competition horses (equestrian and racing) would be difficult and expensive, and is probably unrealistic.

The continuous veterinary supervision, good veterinary records and degree of isolation from the general horse population, which are the norm with international-standard competition horses, suggest that the health risks posed by these may be lower than with other horses. However, the real effect of these factors is difficult to assess. There is probably insufficient technical justification for reducing the period of isolation for competition horses, because the recommendations have been based upon epidemiological considerations such as incubation and infective periods for diseases spread by means other than venereal transmission. Flexibility in the practical application of isolation presents an alternative approach, and may be justified if facilitating imports of competition horses is considered a worthwhile objective by a consensus of the equine industry sectors.

Towards this end, competition horses could be allowed to train throughout the isolation periods in training facilities outside of isolation facilities, so long as the principles of isolation are maintained. For instance, isolation recommendations have been made in respect of equine influenza. During training (and transport to and from training facilities) a 100 m separation should be maintained between isolated and other horses. When isolation requirements have been made in respect of arthropod vector borne diseases, training times should be outside expected vector feeding times and horses should be treated with prophylactic insecticides prior to leaving insect-proof facilities.

With respect to the disease risks posed by competition horses, it is also worth noting that the assurance provided by premises of origin health status declarations will differ for different classes of horse. Horses in active work or participating in competition events in the pre-export period may be exposed to horses of unknown health status, unless this is specifically excluded by isolation requirements such as discussed above. Under such circumstances, premises of origin health status declarations cannot be considered to provide an absolute assurance of isolation from animals not of equivalent health status. This is a particularly important consideration for horses being imported from Australia, which will not be subject to an isolation requirement under the recommendations of this risk analysis.

The isolation requirements for competition horses are schematically represented in figure 1 (page 213).

8.5 Legal considerations

Imported horses may only receive clearance under the Biosecurity Act 1993 once it has been established they are not infected with unwanted organisms. Uncleared horses must remain confined

within a transitional facility, unless being moved to another transitional facility under the direction of an Inspector. Import health standards prescribe the measures to be completed so goods can be determined not to be harbouring unwanted organisms, allowing clearance to be issued. Transitional facility standards prescribe the requirements for containment of uncleared goods in order to prevent the spread of any organisms that may be associated with the uncleared goods.

If competition horses were to remain untested for specific diseases they could not legally receive clearance. In that case, they would have to remain within a registered transitional facility, except when being moved between two such facilities. A transitional facility standard would need to describe measures appropriate to contain the unwanted organisms for which testing had not been completed.

In summary, the legal implications of the Biosecurity Act 1993 in respect of competition horses are that:

- The disease risks posed by particular organisms could be managed by allowing competition horses to be imported without meeting all the pre-export biosecurity requirements. The alternative regime of risk management for competition horses would be described within an import health standard;

- The import health standard could not allow competition horses that had not completed all biosecurity requirements to receive biosecurity clearance;

- Competition horses that were not eligible for biosecurity clearance would have to remain in registered transitional facilities, except when being moved between such facilities under the direction of an Inspector;

- The transitional facilities would have to be approved and registered according to a standard. The standard would describe measures appropriate to manage the risks of diseases for which the horses had not completed all the pre-export biosecurity requirements.

8.6 Recommended measures for competition horses

The measures for competition horses that import health standards and transitional facility standards must address are as follows:

1 Pre-export isolation must be completed in approved facilities. If horses have specific training requirements that pre-export isolation facilities cannot meet, training at another facility can be undertaken under the following conditions:

1.1 The training facility is approved for the purpose by an Official Veterinarian;

1.2 The training facility should be situated reasonably close to the isolation facility, and training and transport conducted under the supervision of an Official Veterinarian and in accordance with an approved schedule;

1.3 When isolation requirements have been made in respect of equine influenza, a 100 m separation between isolated and other horses is maintained at all times during training and transport;

1.4 When isolation requirements have been made in respect of arthropod vector borne diseases, training times should be outside expected vector feeding times and horses should be treated with prophylactic insecticides prior to leaving insect-proof facilities;

1.5 A document describing how the above requirements are intended to be complied with, endorsed by an Official Veterinarian, must be submitted to MAF for approval.

2 The administrative body organising the event must endorse the application to MAF for a permit to temporarily import a competition horse, if there is no intention to seek biosecurity clearance for that horse. The national body must be aware of the transitional facility requirements under which the horses will be allowed to participate and indicate how these requirements will be accommodated within the plans for staging the competition event.

3 A copy of a passport that conforms with OIE Code Model Passport No. 6.1 must accompany the application for a permit to temporarily import a competition horse. The passport must provide:

3.1 Details of ownership of the horse;

3.2 Identification of the horse;

3.3 A record of movements of the horse. As a minimum, all international movements within the previous 12 months must be recorded;

3.4 A record of all vaccinations against equine influenza and any other diseases;

3.5 The result of every test undertaken for a transmissible disease by a veterinarian or laboratory authorised by a Government Veterinary Service.

4 The original of the passport must accompany the horse to New Zealand.

5 Health certification completed by an Official Veterinarian of the exporting country providing the assurances detailed within the import health standard for importation of horses from the country of origin must accompany the horse when imported. However, pre-export testing requirements for EVA (semen testing in seropositive stallions only), CEM, dourine and piroplasmosis (or any specific combination of these tests) will be exempt within the permit issued for a competition horse if there is no intention to seek biosecurity clearance.

6 Temporarily imported competition horses must complete post-arrival quarantine

requirements in an appropriately registered transitional facility, in accordance with the requirements detailed in the import health standard for horses from the country of origin.

7 Following completion of post-arrival quarantine (or for specific training purposes during post-arrival quarantine), competition horses could be moved to premises approved and registered according to a specific MAF transitional facility standard. The importer/national body could nominate a resident/stabling premise, a training premise, and the competition premise for the duration of the horse's visit. The horse would not be allowed to leave the transitional facilities except to proceed under the authority of an Inspector to another transitional facility or to be exported.

8 A MAF standard for transitional facilities for competition horses will detail:

8.1 The process by which such facilities are registered;

8.2 The restrictions horses will be subject to in order to ensure that transmission of disease does not occur, including;

8.2.1 In the case of horses undergoing post-arrival quarantine:

- When quarantine requirements have been made in respect of equine influenza, a 100 m separation between isolated and other horses is maintained at all times during training and transport;
- When quarantine requirements have been made in respect of arthropod vector borne diseases (vesicular stomatitis, VEE, surra), training times should be outside expected vector feeding times and horses should be treated with prophylactic insecticides prior to leaving insect-proof facilities;

8.2.2 In the case of horses which have not completed testing for EVA, CEM or dourine, there must be no sexual contact with other horses;

8.2.3 In the case of all uncleared horses, any equipment used on the horse (riding, grooming and veterinary equipment) must be disinfected prior to use on any other horse.

8.3 The requirements for supervision of the horse in order to ensure that the above provisions are adhered to.

9 The horse's movements should be kept to the minimum required to train and compete, and the Inspector must authorise all such movements.

10 If there is no intention to seek biosecurity clearance, the permit shall be for a temporary importation and will prescribe a date for re-export that will be as soon as practicable after participation in the event. For the purposes of export from New Zealand, the health status of the horse shall not be considered equivalent to that of a domestically resident horse. Specific approval from the government veterinary authorities of the next destination country following the horse's visit to New Zealand must be received by MAF and approve re-export from New Zealand before a permit to import will be issued.

References

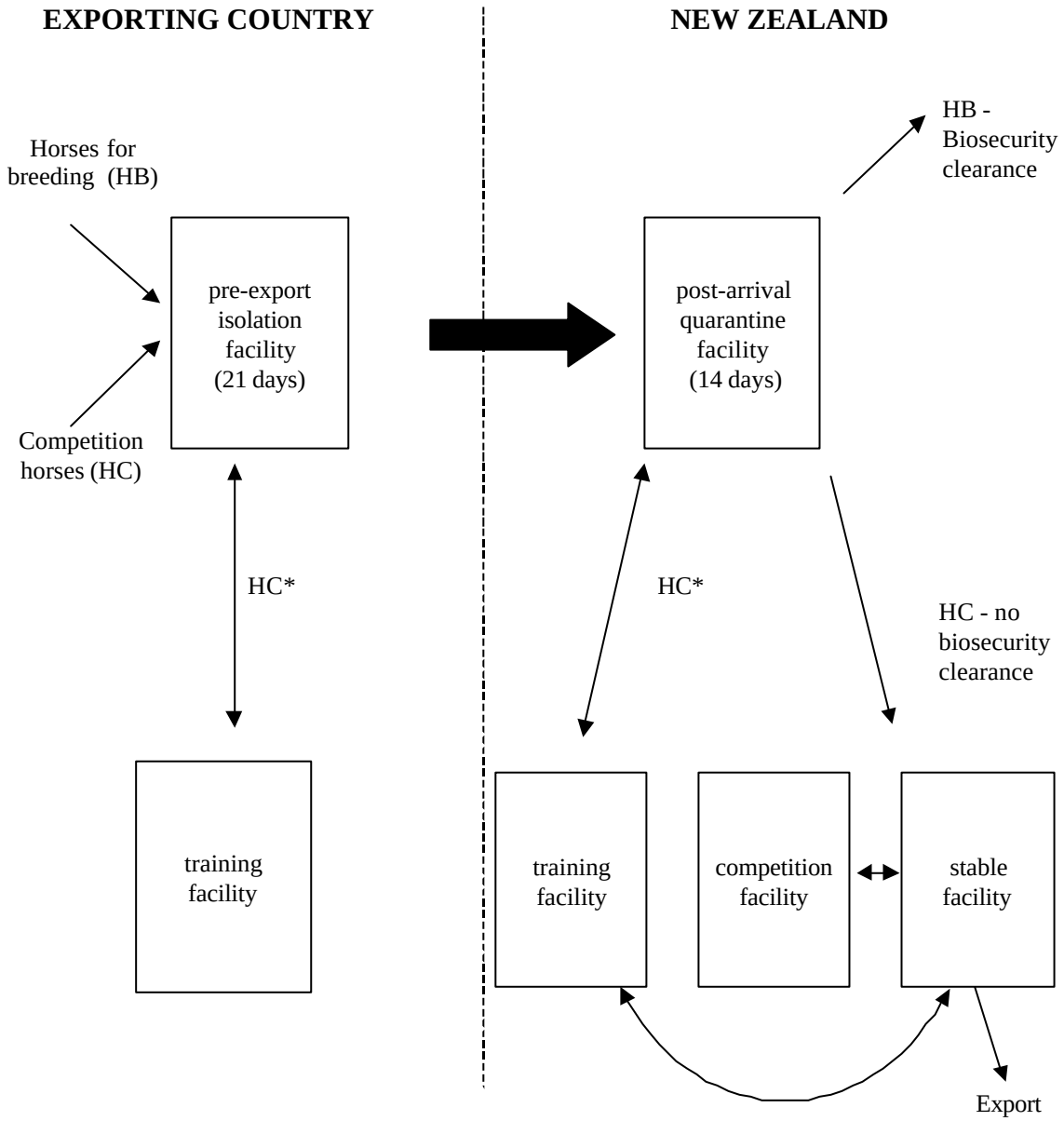
1 Sluyter F. (1998). International Horse Movement: Health requirements versus impact on equestrian sports development. Equine Infectious Diseases VIII. Proceedings of the Eighth International Conference on Equine Infectious Diseases, Dubai, March 1998. R&W Publications (Newmarket) Ltd. 334-335. 1999.

2 OIE. (1997). International Animal Health Code. Model Passport 6.1: Model Passport for International Movement of Competition Horses. 605-620.

3 Brooks LM. The equine piroplasmosis control programme at the 1996 Summer Olympic Games. Equine Infectious Diseases VIII. Proceedings of the Eighth International Conference on Equine Infectious Diseases, Dubai, March 1998. R&W Publications (Newmarket) Ltd. 371-375. 1999.

4 AQIS. Draft import risk analysis report of the temporary importation of horses that are serologically positive for equine piroplasmosis for competition of racing purposes. Australian Quarantine and Inspection Service. 6 January 1999. 30 pages.

Figure 1: Schematic representation of isolation requirements for competition horses and horses for breeding



** Competition horses will be allowed to move to and from training facilities during pre-export isolation and post-arrival quarantine under the following conditions:*

- *Approved training facility*
- *Approved transport methods*
- *Approved training schedule*
- *Supervision by official veterinarion*
- *Isolation from other horses maintained*

9 VETERINARY ESCORT

Import health standards for livestock allow the Director of Animal Biosecurity to appoint a veterinary escort to accompany consignments, at the importer's expense. The escort undertakes duties described in *MAF Standard 154.01.04 Standard for the Escort of Animal Shipments into New Zealand*. These include audit of pre-export preparations and supervision during transit. The objective is to ensure animal health status at the time of export is as required by the import health standard, and is maintained throughout the journey to New Zealand. The escort also safeguards animal welfare during transport.

To date, all consignments of horses imported by air from countries other than Australia have been escorted. The absence of post-arrival quarantine was considered to warrant a conservative approach to pre-export and in-transit risk management.

Importers and animal transporters have noted that the requirement for veterinary escort places a financial burden on this trade, resulting from the escort's airline tickets, daily charge-out rate and expenses.

Trained grooms accompany all flights, and the importers and animal transporters contend that animal welfare requirements are satisfied through the care extended by these grooms, as well as the use of private veterinarians at their own discretion. Further, air transport must comply with the International Air Transporters Association (IATA) *Live Animals Regulations*,[(1)] obligating airlines to ensure animal welfare requirements are met during transport.

Authorities in the exporting country oversee MAF's zoosanitary requirements and certify accordingly. There are guidelines governing the competence of veterinary authorities [(2)] and the principles of veterinary certification [(3)] in the OIE Code. An import health standard allowing the import of livestock will only be issued if the government veterinary authority in the exporting country has been favorably assessed. Considering this competency assessment prior to establishing trade, requiring audit of pre-export preparations for every consignment is probably not justified. Periodic audit presents a more cost-effective approach to compliance, and should target those export sources and trade routes where problems have been encountered during previous consignments of horses or for other commodities.

Operational issues may arise that require a MAF representative's input. Issues identified during routine pre-export preparation can usually be resolved through direct communication between MAF and the competent authority in the exporting country. There are rare occasions when the physical presence of a MAF representative may assist in determining the appropriateness of variations from agreed standards, such as during the approval of pre-export isolation facilities.

MAF has produced special requirements for live animal imports via air transport routes that have an associated animal health risk, for instance transit of airports in countries where there are diseases spread by arthropod vectors. A MAF representative should escort every such importation to ensure the specific requirements during transport are adhered to.

MAF no longer considers pre-export audit and escort by a MAF-representative to be mandatory during transport of horses. However, MAF retains the authority to require audit and escort at the importer's expense, and will exercise discretion based on assessment of the risks posed by specific export sources and transport routes.

References

1 International Air Transport Association. Live Animal Regulations. IATA, Montreal-Geneva. 24th Edition, 1997.

2 OIE. International Animal Health Code: mammals, birds and bees. Section 1.4. Import risk analysis. Office International des Epizooties, Paris. 16-29. 1999.

3 OIE. International Animal Health Code: mammals, birds and bees. Chapter 1.3.2. Principles of certification. Office International des Epizooties, Paris. 13-15. 1999.

10 SEMEN COLLECTION, PROCESSING AND STORAGE

Importation of equine semen presents a risk of introducing diseases able to be spread by artificial insemination. Specific recommendations have been made in the disease monographs to account for these risks. The risks include the potential for widespread dissemination of any contaminated consignment, potentially leading to simultaneous infection of horses in different locations. Industry requirements for health measures to allow for trade in fresh/chilled as well as frozen semen must also be considered.

A *MAF standard for semen collection centres for the collection of horse semen for export to New Zealand* (Appendix 1) describes the operational and technical requirements for facilities collecting, processing and storing equine semen. The measures provide a level of biosecurity within the facility considered appropriate to manage the risks of diseases potentially able to be transmitted by equine semen, when they occur in the exporting country. This document will be appended to all import health standards for horse semen, and will be the standard by which the government veterinary authorities of exporting countries shall approve horse semen collection centres for export to New Zealand. If the health status of the exporting country is such that particular disease of concern do not occur, it may be appropriate to authorise dispensations from the semen collection centre standard. These will be determined by bilateral negotiation between MAF and the veterinary authorities of the exporting country.

The basic principle is that while resident on the semen collection centre the donor stallion should not have contact with (including natural mating) horses not of an equivalent isolation and tested health status as an eligible donor. On the dates of collection of semen, the Supervising Veterinarian should examine all the animals on the semen collection centre and no animals should show any clinical evidence of infectious or contagious disease.

New, disposable artificial vagina (AV) liners and collection vessels should be used for each collection, or re-usable AV liners should only be used by a single stallion and must be thoroughly cleaned between uses.

All products and instruments used in the collection, processing and storage of the semen, particularly semen extenders and agents used in the cryo-preservation process, should be free from contamination by pathogenic microorganisms. All products and instruments should be handled in a manner that ensures they do not become contaminated during collection, processing and storage of semen.

The European Union requires that antibiotics be added to all imported horse semen, through Commission Decision 95/176/EC of 6 April 1995 amending Council Directive 92/65/EEC. This minimises the risk of contaminating bacteria being present in the semen, and transmission of specific pathogens with which the donor may have been infected. [(2)]

In accordance with the EU recommendations, antibiotics should be added to the semen to achieve an effect at least equivalent to the following dilutions:

not less than 500 IU per ml streptomycin,
500 IU per ml penicillin,
150 µg per ml lincomycin,
300 µg per ml spectinomycin.

Immediately after the addition of the antibiotics the diluted semen should be kept at a temperature of at least 15°C for a period of not less than 45 minutes.

After processing, the semen should be stored in new or previously sterilised containers (most commonly straws) which are individually identified with information that includes the date and place of collection and identity of the stallion (if a code is used, the decipher should accompany the semen). In the case of frozen semen, the containers will then be placed in liquid nitrogen within a cryo-preservation tank. The nitrogen should be fresh, and certainly should not have been used previously to store biological materials that may have been of differing health status to horse semen for export to New Zealand. If stored for any period prior to export, storage should be at a facility under the supervision of an official veterinarian. All servicing of the tank, such as topping up nitrogen or removal of semen straws, should be performed by approved veterinarians, in a manner that prevents contamination of the tank or its contents.

Importers have noted that the short-shelf life of fresh/chilled semen can often require multiple import consignments over a relatively short period, and that the previous MAF requirement for an original permit to accompany each consignment creates difficulty and unnecessary expense. In future, MAF's policy of only requiring permits when there are post-arrival requirements (the permit acts as a means of ensuring suitable arrangements to meet post-arrival requirements are in place) will mean that equine semen imports will not be subject to permits. Clearance at the New Zealand border will be solely dependent upon the consignment meeting eligibility and zoosanitary certification requirements described in the import health standard.

Imports of equine embryos or ova are a future possibility, however no conditions are proposed for this trade at present. Further risk analysis would be required to determine specific zoosanitary recommendations. Those within the OIE Code will provide a useful reference upon which future trade conditions could be based. [(3)]

References

1 IETS. Manual of the International Embryo Transfer Society. Second Edition. *Eds:* DA Stringfellow and SM Siedel. International Embryo Transfer Society, USA. 79 pages. 1990.

2 OIE. International Animal Health Code. Appendix 4.2.3.7. Equine embryos/ova. Office International des Epizooties. 338-342. 1999.

11 ACKNOWLEDGEMENTS

Dr Stuart MacDiarmid, National Manager Risk Management, MAF Biosecurity Authority edited the text and advised on technical aspects of the risk analysis.

Drs. Barry O'Neil, Group Director of Biosecurity; Wayne Ricketts, National Adviser Import Management; Noel Murray, National Adviser Risk Management; Howard Pharo, National Adviser Risk Management; Jim Edwards, National Manager International Trade; Mirzet Sabirovic, National Manager Emergency Response; Kevin Corrin, National Manager Import Management; and Kerry Mulqueen, National Adviser Import Management, MAF Biosecurity Authority reviewed the draft text and/or provided advice and feedback on various matters considered within the risk analysis.

Dr. Paul Fraser, Cambridge Veterinary Services reviewed the draft text on behalf of New Zealand Thoroughbred Breeders Association.

Drs. Murray Brightwell, Cambridge Veterinary Services, and John O'Flaherty reviewed the draft text on behalf of New Zealand Veterinary Association Equine Branch Infectious and Exotic Diseases Subcommittee.

Dr. Gary Horner, MAF National Centre for Disease Investigation, provided advice on aspects of particular diagnostic procedures.

Sarah Peters, Technical Advisory Officer International Trade, MAF Biosecurity Authority assisted compilation of the tables on country health status, preparation for publicaiton and distribution of the risk analysis.

Dr. Vaughan Seed, Veterinary Consultant, Waikanae, is primarily responsible for the equine import health policy which up until this review has served New Zealand well.

Dr. Lisa Ferguson, Animal and Plant Health Inspection Service, United States Department of Agriculture, provided information on USA equine health status and import health policy.

Drs. Robyn Martin, Australian Quarantine and Inspection Service, Animal Quarantine Policy Branch; Mike Nunn, Bureau of Resource Sciences, Department of Primary Industries and Energy; and Patricia Ellis, Horse Industry Programs, Natural Resources and Environment, State Government of Victoria, provided information on Australian equine health status and import health policy.

Prof. William A. Ellis, Department of Agriculture for Northern Ireland; Dr. Carole Bolin, Zoonotic Diseases Research Unit, United States Department of Agriculture; Dr. Roger Marshall, Info-Brok Technical Consultancy; Dr. David Millar, National Veterinary Services Laboratories, United States Department of Agriculture; and Dr. Steve Hathaway, MAF Food Assurance Authority, provided expert reviews of the chapter on equine leptospirosis.

Bruce Graham, NZ Equine Health Association Inc; Eildert Kingma, International Horse Breeders; Dr Andrew Higgins, Scientific Director and Chief Executive, Animal Health Trust; Michael

Cameron, Science & Research Division, Department of Conservation; Dr Henry Dowler, Deputy Chief Technical Officer (Health), Ministry of Health; Dr Murray Brightwell, Cambridge Veterinarian Services; Graeme Henley, Prestige Bloodstock Ltd; Quentin Wallace, International Racehorse Transport; Dr Sarah Kahn, Assistant Director, Animal Policy Quarantine Branch, Australian Quarantine and Inspection Service; Dr Peter Timoney, Chairman & Director, Gluck Equine Institute, University of Kentucky; Dr Bart R Thompson, Vet First; Dr Gabrielle Deuss, on behalf of NZ Arab Horse Breeders Society (Inc); Mr Rod Hoare, State Equine Veterinary Officer, Elizabeth Macarthur Agricultural Institute, NSW; Dr P Dollinger, Head, Permits and Inspections, Switzerland; Professor Allan Guthrie, Equine Research Centre, University of Pretoria; Dr Robin A Bell, Head - Veterinary International Trade, Ministry of Agriculture, Fisheries and Food, UK; Sharon van Gulik, NZ Equestrian Federation Inc; Dr Thomas E. Walton, Acting Deputy Administrator, Veterinary Services, United States Department of Agriculture; Dr Robert ter Horst, Director of Veterinary Service, Swedish Board of Agriculture; Dr Monique Eloit, Service de la Qualité Alimentaire et des Actions Vétérinaires et Phytosanitaires, France; Dr Ritta Heinonen, Senior Veterinary Officer, Ministry of Agriculture and Forestry, Finland; Owen Jacobson, New Zealand Deer Farmers Association; Associate Prof. Dave West, Institute of Veterinary, Animal and Biomedical Sciences, Massey University; Dr Alison Roberts, Senior Advisor Public Health Medicine, Ministry of Health; and Associate Prof. Peter Wilson, Institute of Veterinary, Animal and Biomedical Sciences, Massey University, made written submissions during the risk analysis process.

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.

Appendix 1.

Health status of various countries for the OIE List A and B diseases of concern

Source: OIE. World Animal Health in 1998. Part 2. Tables on the animal health status and disease control methods. Office International des Epizooties. Paris, France. 1999.

Legend for disease occurrence reports

0000	Disease never reported
-	Disease not reported (date of last outbreak not known)
(month/year)	date of last reported occurrence of the disease in previous years
?	Disease suspected but presence not confirmed
+	Reported present or known to be present
+?	Serological evidence and/or isolation of the causal agent, but no clinical signs of disease
( )	Disease limited to specific zones
...	No information available

Legend for disease control reports

*	Notifiable disease
Cn	Control of arthropods
Cr	Control of wildlife reservoirs
M	Monitoring
Qf	Precautions at the border
Qi	Movement control inside the country
S	Stamping out
Sp	Stamping out (Modified ~)
Su	Surveillance
Te	Screening
V	Vaccination
Vp	Vaccination prohibited
Z	Zoning

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.

OIE	DISEASE NAME	FRANCE		GERMANY		IRELAND		PORTUGAL		SPAIN	
	OIE List A diseases										
A020	vesicular stomatitis	oooo	* Qf S	oooo	* Qf Qi S	oooo	* Qf S Vp	oooo		oooo	*
A110	African horse sickness	oooo	* Qf S	oooo	* Qf Qi S	oooo	* Qf S Vp	1989	* Qf Vp	1990	*Vp
	OIE List B diseases										
B051	anthrax	+	* Qi V	1994	* Qf Qi S Vp	1970	* Qf Qi Vp	-	* Qf V	+	*V
B056	leptospirosis	+		+		+	Qf V	+	V	+	V
B058	rabies	+ ()	* V Z	+	* Qf Qi Sp V	1903	* Qf Qi S	1984	* Cr Qf S V	+ ( )	* M Qf V
B060	screwworm	oooo		oooo		oooo		oooo		oooo	
B201	contagious equine metritis	+	* Qf Su	+	Qf Qi	1982	* Qi	-		-	
B202	dourine	1958	* Qf	1953	* Qf Qi	-	*	1925		1955	*
B203	epizootic lymphangitis	oooo	* Qf S	oooo		1906	*	-		-	
B204	EEE & WEE	-	* Qf S	oooo	* Qf Qi S	oooo	*	-		oooo	
B205	equine infectious anaemia	+	* Qf Sp	1993	* Qf Qi Sp	oooo	*	-		1983	
B206	equine influenza (virus type A)	+	V	+	V	+	V	1996		+	V
B207	equine piroplasmosis (babesiosis)	()		-		-		1996		+	
B208	equine viral rhinopneumonitis	+		+	V	+	V	-		1989	
B209	glanders	1965	* Qf S	1955	* Qf Qi S	1920	*	1952		1956	*
B210	horse pox	-		-		-		-		oooo	
B211	equine viral arteritis	+		+	Qf	-	* Qi	-		-	
B212	Japanese encephalitis	oooo	* Qf S	oooo	* Qf Qi S	oooo	*	-		oooo	
B215	surra (*Trypanosoma evansi*)	oooo	*	oooo		oooo		-		oooo	
B216	Venezuelan equine encephalomyelitis	oooo	* Qf S	oooo	* Qf Qi S	oooo	*	oooo		oooo	

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.

OIE	DISEASE NAME	SWEDEN		UNITED KINGDOM		ICELAND		SOUTH AFRICA		UAE	
	OIE List A diseases										
A020	vesicular stomatitis	oooo	* S Vp	oooo	* Qf S Vp	oooo		oooo	Qf S Su	oooo	
A110	African horse sickness	oooo	Qf S Vp	oooo	* Qf S Vp	oooo		+	* M Qi Su V Z	oooo	
	OIE List B diseases										
B051	anthrax	1981	*	07/1997	* Qf Sp V	1965	S	+	* M Qi V	-	*
B056	leptospirosis	+?	*	-	Qf	1995		+			
B058	rabies	1886	*	1970	* Qf	oooo	Vp	+	* V	1994	* V
B060	screwworm	oooo	*S	0		oooo		oooo	Qf	oooo	
B201	contagious equine metritis	1996	* Te	1997	* Qi	oooo		oooo	Qf S Su	oooo	* Qi
B202	dourine	oooo	*	oooo	* Qf	oooo		+	* Qf Qi Sp	oooo	* S
B203	epizootic lymphangitis	oooo	*	1906	* Qf	oooo		+		oooo	S
B204	EEE & WEE	oooo	*	oooo	* Qf	oooo		oooo	Qf S Su	oooo	* S
B205	equine infectious anaemia	1989	*	1976	* Qf	oooo		1955	Qf S Su	oooo	* S
B206	equine influenza (virus type A)	+	*V	+		oooo		1996	V	1996	V
B207	equine piroplasmosis (babesiosis)	+?	*	oooo		oooo		+		+	
B208	equine viral rhinopneumonitis		*V	+		oooo		+		-	
B209	glanders	1943	*	1928	* Qf	oooo		1945	Qf S Su	oooo	* S
B210	horse pox	oooo	*	oooo	* Qf	oooo		-		oooo	
B211	equine viral arteritis	+	*	(+?)	* Qf Su	oooo		1996		oooo	*
B212	Japanese encephalitis	oooo	*	oooo	* Qf	oooo		oooo	Qf S Su	oooo	
B215	surra (*Trypanosoma evansi*)	oooo	*	oooo		oooo		oooo	Qf S Su	+	
B216	Venezuelan equine encephalomyelitis	oooo	*	oooo	* Qf	oooo		oooo	Qf S Su		

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.

OIE	DISEASE NAME	JAPAN		HONG KONG		SINGAPORE		MALAYSIA	
	OIE List A diseases								
A020	vesicular stomatitis	oooo	* Qf S	oooo		oooo	* Qf S Vp	oooo	
A110	African horse sickness	oooo	* Qf	oooo		oooo	* Qf S Vp	oooo	
	OIE List B diseases								
B051	anthrax	1991	* Qf S	1982	*	oooo	*Qf	-	
B056	leptospirosis	+	Qf	?		1987		…	
B058	rabies	1956	* Qf S	1987	*	1935	* Qf S Vp	+	Q S *
B060	screwworm	oooo	Qf			oooo	Qf	oooo	
B201	contagious equine metritis	+	Qf	-		oooo	*Qf	oooo	
B202	dourine	-	Qf	oooo	*	oooo	*Qf	oooo	
B203	epizootic lymphangitis	-	* Qf	oooo	*	oooo	*Qf	oooo	
B204	EEE & WEE	-	Qf	1971	*	oooo	*Qf	oooo	
B205	equine infectious anaemia	1993	* Qf S Su	1976	*	oooo	*Qf	oooo	
B206	equine influenza (virus type A)	1972	* Qf V	1992	*	1977	* V	1977	V
B207	equine piroplasmosis (babesiosis)	oooo	* Qf S	-		oooo	*Qf	oooo	
B208	equine viral rhinopneumonitis	1996	Qf V	+		oooo	*Qf	oooo	
B209	glanders	1935	* Qf S	oooo	*	oooo	*Qf	1907	
B210	horse pox	oooo	Qf	...		oooo	*Qf	oooo	
B211	equine viral arteritis	oooo	Qf	oooo		oooo	*Qf	oooo	
B212	Japanese encephalitis	1985	* Qf Su	1981	*	1988	* V	+	V
B215	surra (*Trypanosoma evansi*)	oooo	* Qf	-		oooo	*	(+)	Cn
B216	Venezuelan equine encephalomyelitis	oooo	Qf	oooo		oooo	* Qf	oooo	

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.

OIE	DISEASE NAME	USA		CANADA		AUSTRALIA		NEW ZEALAND	
	OIE List A diseases								
A020	vesicular stomatitis	+ ()	* Qf Qi Su	1949	* Qf	oooo	Qf	oooo	*Qf
A110	African horse sickness	oooo	* Qf Su	oooo	Qf	oooo	Qf	oooo	*Qf
	OIE List B diseases								
B051	anthrax	+ ()	V	(+)	*	+ ()	* Qi Su V	1954	* Qf
B056	leptospirosis	+	V	+	V	+	Qi V	+	Qf V
B058	rabies	+	* V	+	* Qi V	1867	* Qf	oooo	* Qf
B060	screwworm	+	* Cn Qf	oooo	Qf	oooo	* Qf	oooo	Qf
B201	contagious equine metritis	+?	* M Qf Su	oooo	* Qf	1980	* Qf	oooo	Qf
B202	dourine	1934		1921	* Qf	oooo	* Qf	oooo	Qf
B203	epizootic lymphangitis	oooo		oooo	Qf	oooo	Qf*	oooo	Qf
B204	EEE & WEE	+ ()	* Cn M V	+	V	oooo	Qf*	oooo	* Qf
B205	equine infectious anaemia	+	* Cn M Qf Qi Su	+	* Qf Qi Sp Te	+ ? ()	Qf*	oooo	* Qf
B206	equine influenza (virus type A)	+	V	+	V	oooo	Qf*	oooo	* Qf
B207	equine piroplasmosis (babesiosis)	+ ()	* Qf	1987	* Qf	1976	Qf*	oooo	* Qf
B208	equine viral rhinopneumonitis	+	V	+	V	+	Qf	+	V
B209	glanders	1942	* Qf	1938	* Qf	1891	P *	oooo	* Qf
B210	horse pox	oooo		oooo	Qf	oooo		oooo	Qf
B211	equine viral arteritis	+	* V	+	V	+?	Qf	+?	* Qf Qi V
B212	Japanese encephalitis	oooo		oooo	Qf	+ ? ()	* M Qf	oooo	Qf
B215	surra (*Trypanosoma evansi*)	oooo	*	oooo	Qf	oooo	* Qf	oooo	* Qf
B216	Venezuelan equine encephalomyelitis	1971	* Qf V	oooo	Qf	oooo	* Qf	oooo	* Qf

Appendix 2.

MAF STANDARD FOR EQUINE SEMEN COLLECTION CENTRES COLLECTING SEMEN FOR EXPORT TO NEW ZEALAND

1 HEALTH STATUS

1.1 The centre must have remained free from the following diseases for the indicated calendar period prior to collection of semen for export to New Zealand, and following removal of any earlier case and subsequent testing of all horses to re-establish freedom:

African horse sickness (2 months)
vesicular stomatitis (21 days)
equine infectious anaemia (3 months)
equine viral arteritis (3 months)
glanders (6 months)
contagious equine metritis (3 months)
dourine (6 months)
equine salmonellosis (*Salmonella abortus-equi*) (3 months)

1.2 All horses on the centre during the period of semen collection for export to New Zealand must be of an equivalent health status as eligible donor stallions.

2 LOCATION

2.1 The centre may be located on an established equine enterprise. In that case, the entire premises should meet the health status requirements noted at 1.1 above. For the duration of the period of collection of semen for export to New Zealand, contact between horses on the centre and other equines must be prevented.

2.2 The centre must be conveniently located for supervision by a Government Veterinary Officer or Government approved Veterinarian (an *Official Veterinarian*).

3 FACILITIES

3.1 The centre must be surrounded by two secure stock-proof fences at least 5 metres apart except where the wall of a building forms part of the perimeter. [Exceptions may be approved by MAF if they are considered to provide equivalent quarantine security.]

3.2 Stables on the centre should offer protection against sun, wind, rain and extremes of temperature, and must be so constructed that they can be readily cleaned and disinfected. Stables, yards, fences, and feeding and watering arrangements should be so constructed that the horses are protected from injury, and other welfare needs are met.

3.3 The centre shall have facilities for veterinary examination of animals and the collection of

Figure 1 – Health status of various countries for the OIE List A and B diseases of concern.
samples, and facilities for the segregation and isolation of sick animals.

3.4 Semen must be processed in a room or building or mobile laboratory set aside for that purpose, separate from areas where animals are housed and where semen is collected. All working surfaces in this facility must be cleaned and disinfected before use.

4 OPERATION

4.1 The centre must be approved by an *Official Veterinarian* prior to each period of collection of semen for export to New Zealand.

4.2 Before approval, an *Official Veterinarian* must be satisfied that all equipment and working surfaces likely to come into contact with semen for export or personnel handling semen has been appropriately cleaned and disinfected.

4.3 All measures described in the zoosanitary certification, including identification of donor stallion and semen, disease testing, semen collection, processing and storage must be supervised by an *Official Veterinarian* .

4.4 Liners used in artificial vaginas during the collection process should be:
Either: new disposal liners on each occasion;
Or: re-usable rubber liners dedicated to individual stallions, which have been thoroughly cleaned and dried between each use.

4.5 Personnel collecting and processing semen must be trained in, and practice, proper disinfection procedures and hygiene techniques.

4.6 Semen must be stored in a secure area.

4.7 Any health problems affecting horses or other stock on the centre during the collection period must be promptly reported to the *Official Veterinarian*, who shall investigate in order to rule out infectious diseases of concern during trade in equine semen.

4.8 Records detailing identification of all equines on the centre, their origins, dates of entry, dates and results of disease tests or investigations, treatments either therapeutic or prophylactic, any departures from good health and condition, inspection visits by the *Official Veterinarian,* and any other information relevant to each animal's health status while it resident on the centre must be kept by the operator and/or the export agent.

4.9 Unauthorised access to the centre should be prevented. All visitor entries must be recorded.