AUSTRALIAN VETERINARY EMERGENCY PLAN

AUSVETPLAN

1996

Disease Strategy

African horse sickness

AUSVETPLAN is a series of technical response plans that describe the proposed Australian approach to an exotic animal disease incursion. The documents provide guidance based on sound analysis, linking policy, strategies, implementation, coordination and
emergency-management plans.

Agriculture and Resource Management Council of Australia and New Zealand

This Disease Strategy forms part of:

AUSVETPLAN Edition 2.0, 1996

[AUSVETPLAN Edition 1.0, was published in 1991]

This strategy will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to the AUSVETPLAN Coordinator (see Preface).

Record of amendments to this manual:

[Insert record of amendments as necessary]

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PREFACE

This **Disease Strategy** for the control and eradication of **African horse sickness** (AHS) is an integral part of the **Australian Veterinary Emergency Plan,** or AUSVETPLAN Edition 2.0. AUSVETPLAN structures and functions are described in the **Summary Document.**

This strategy sets out the disease control principles that were approved by the Agriculture and Resource Management Council of Australia and New Zealand (ARMCANZ) out-of-session in January 1996, for use in a veterinary emergency caused by the introduction of AHS to Australia.

AHS is designated as a List A disease by the Office International des Epizooties (OIE). List A diseases are, 'Communicable diseases which have the potential for serious and rapid spread, irrespective of national borders; which are of serious socioeconomic or public health importance and which are of major importance in the international trade of animals and animal products'. The principles contained in this document for the diagnosis and management of an outbreak of African horse sickness conform with the **OIE International Animal Health Code 1992** (OIE Code; Appendix 3).

AHS is not included in the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases.

Detailed instructions for field implementation of the strategies are contained in the AUSVETPLAN **Operational Procedures Manuals** and **Management Manuals.** Cross-references to strategies, manuals and other AUSVETPLAN documents are expressed in the form:

> Document Name, Section no.
> For example, **Decontamination Manual, Section 3**.

In addition, *Exotic Diseases of Animals: A Field Guide for Australian Veterinarians* by W.A. Geering, A.J. Forman and M.J. Nunn, Australian Government Publishing Service, Canberra, 1995 (**Exotic Diseases Field Guide**) is a source for some of the information about the aetiology, diagnosis and epidemiology of the disease and should be read in conjunction with this strategy.

This strategy will be reviewed regularly. Suggestions and recommendations for amendments should be forwarded to:

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The writing group was responsible for drafting this strategy. However, the text may have been amended at various stages of the consultation/approval process and the policies expressed in this version do not necessarily represent the views of all members of the writing group. Contributions may also have been made by other people not listed above and the assistance of all involved is gratefully acknowledged.

CONTENTS

1 NATURE OF THE DISEASE

African horse sickness (AHS) is an acute or subacute insect-borne viral (arboviral) disease affecting mainly the Equidae (horses, donkeys and relatives). It is frequently fatal in susceptible horses, producing clinical signs associated with impairment of respiratory and circulatory function. Mild forms of the disease occur particularly in endemic areas.

1.1 Aetiology

AHS is caused by a virus in the *Orbivirus* genus of the family Reoviridae, which also contains bluetongue and epizootic haemorrhagic disease (EHD) viruses. There are nine known serotypes of AHS virus. Serotypes 1–8 are all highly pathogenic for horses and cause 90–95% of mortality but serotype 9 is slightly less pathogenic resulting in mortality rates of about 70% (Coetzer and Erasmus 1994).

1.2 Susceptible species

All members of the horse family (Equidae: horses, mules, donkeys, zebras) are susceptible, with horses generally experiencing severest disease and highest mortality rates. Zebras become infected but generally have mild or subclinical disease. Dogs are also susceptible. The disease does not affect humans.

The domestic horse should be considered an accidental or indicator host and does not remain a long-term carrier of the virus. In Africa indigenous wild Equidae populations used to be plentiful enough to maintain the virus. Zebra in Kruger National park maintain the virus through the year-round presence of the vector and susceptible zebra foals (Barnard 1994).

Little evidence of antibody to AHS virus has been detected in ruminants, with the possible exception of camels. Serological surveys in Africa apparently showed AHS antibodies in elephant sera, but this is now thought to have been due to the elephant sera reacting non-specifically in the complement fixation test used.

1.3 World distribution and occurrence in Australia

AHS occurs endemically in all parts of Africa south of the Sahara, with periodic spread further north. It has occurred in Egypt and the Middle East, extending to Pakistan and India in the early 1960s. Spread also occasionally occurs from north Africa to the Iberian peninsula. This distribution is primarily dictated by the presence of the principal insect vector, *Culicoides imicola*. The most recent outbreaks were in Spain and Portugal (1987–90), Algeria (1989) and Morocco (1989–91).

There has been no occurrence of AHS in Australia.

1.4 Diagnostic criteria

[See Glossary for any terms not defined in the text]

AHS can be suspected on the basis of clinical history but laboratory tests are required to confirm diagnosis and identify the virus serotype.

1.4.1 Clinical signs and lesions

There are three classical clinical disease syndromes of AHS:

- pulmonary;
- cardiac; and
- mild.

However, there is enough overlap to make these rigid distinctions into distinct AHS syndromes difficult to justify. Most cases are, to a greater or lesser degree, mixed in type.

Acute or 'pulmonary' form:

- characterised by a short clinical course, fever, severe respiratory distress and high mortality (up to 95%);
- the incubation period is 3–5 days and onset of the disease is marked by depression and a high temperature (up to 42°C);
- progressive, severe respiratory distress involving increased breathing rate, with the animal adopting a wide based stance, neck extended, nostrils dilated;
- paroxysmal coughing develops, which becomes more frequent and severe as the disease progresses;
- commonly, there are bouts of inability or unwillingness to stand, rolling and colic-like symptoms as the animal becomes increasingly distressed until death occurs from suffocation and drowning in the animal's pulmonary exudates;
- frothy white, sometimes blood-tinged, foam may flow from the nostrils of the moribund animal for several hours before death;
- death usually occurs 4–5 days after the onset of depression;
- recovery is rare with a mortality rate of up to 95%.

This form of the disease occurs in highly susceptible horses and is the most likely form to be encountered in Australia unless a less than fully virulent strain of AHS virus enters Australia. The fatal disease in dogs is usually of this form.

Subacute or 'cardiac, oedematous' form:

- somewhat longer clinical course marked by fever and oedematous swellings with a mortality rate from 50–70%;
- the incubation period can be from 7–21 days. The first clinical sign is fever of 39–41°C persisting for 3–4 days;
- as the fever subsides, generalised oedematous swellings develop along the fascial planes of muscles, particularly of the head and neck. Swelling (oedema) of the area above the eye, the eyelids, lips and tongue and even of the brisket, thorax and ventral abdomen is also seen, but generally not of the legs (important in differential diagnosis);
- bouts of abdominal discomfort, colic and rolling occur because of compromised blood supply and oxygen deficiency of the digestive tract;
- stethoscope examination may reveal fluid exudate in the pleural cavity (hydrothorax) and the pericardial sac (hydropericardium);

- terminally, breathing becomes more difficult, paralysis of the oesophagus with inhalation of stomach contents can occur and oedema of the legs results from cardiac insufficiency;
- death usually occurs within 4–8 days after onset of fever. In animals that recover, oedema usually subsides in 3–8 days.

This form of AHS is caused by less virulent virus strains or where there is already some degree of immunity in the population. Mules can develop this form of AHS.

Mild or 'horse sickness fever' form:

- mildest form of AHS, frequently subclinical and therefore easily overlooked;
- clinical signs consist of influenza-like symptoms and a transient fever up to 40°C can occur for 2–3 days with fever typically more pronounced in the afternoon;
- variable incubation period of 4–14 days.

This form develops in partially immune horses, or horses infected with low virulence strains of virus. It is the most common form of the disease manifested in mules, donkeys, elephants and zebras.

1.4.2 Pathology

AHS resembles a septicaemic disease where circulating virus and possibly toxic factors increase the permeability of the endothelial lining of capillaries. This results in the transudation of plasma into the tissues and body cavities. Lesions can be explained by the pathogenesis of the disease, which resembles that associated with an immune-complex reaction.

Gross lesions

In field outbreaks of AHS, postmortem findings are usually combinations of pulmonary and cardiac forms, with secondary digestive vascular lesions. The gross lesions are variable and in peracute cases there may be few obvious lesions.

If AHS is suspected, the postmortem technique should be varied slightly, to ensure that the skin of the horse over the head, neck, chest and back is first reflected and the musculature carefully examined. A yellow, gelatinous oedema is frequently seen in the intermandibular space, frontal area, fascial planes of the muscles adjacent to the ligamentum nuchae, along the jugular groove and the intercostal muscles.

On opening the abdomen, ascites may be seen, together with areas of hyperaemia, petechial haemorrhages and cyanosis of the serosal surfaces of the large and small intestines. Hyperaemia of the glandular fundus of the stomach, enlarged, oedematous mesenteric lymph nodes, subcapsular petechia of the kidney and spleen and ecchymotic or petechial haemorrhages on the diaphragm are often seen.

Marked hydrothorax with 3–5 litres of fluid in the chest and severe pulmonary oedema with prominent interlobular septa, wet, heavy lungs and froth-filled trachea are the most striking features in peracute cases of AHS. Periaortic and peritracheal oedematous infiltration and oedema of the mediastinal nodes are often present. Petechial haemorrhages are present in the pericardium and there may be a slight increase in pericardial fluid.

Hydropericardium is the most prominent feature of the subacute form. The pericardium can contain up to 2 litres of straw-coloured fluid. Usually there are petechial and

ecchymotic haemorrhages on the epicardium and endocardium, often most prominent along the course of the coronary vessels and the heart valves. The lungs may be only slightly oedematous and hydrothorax is not prominent.

Microscopic lesions (histopathology)

There are no specific microscopic features for AHS. Pulmonary oedema, early cardiac necrosis and congestion, haemorrhage and oedema in many body tissues are usually seen, but are not unique to this disease.

1.4.3 Laboratory tests

Animal specimens should initially be sent to the State or Territory diagnostic laboratory from where they will be forwarded to the Australian Animal Health Laboratory (AAHL), Geelong for exotic disease testing after obtaining the necessary clearance from the chief veterinary officer (CVO) of the State or Territory of the disease outbreak and informing the CVO of Victoria (for transport of the specimens to Geelong).

Specimens required

Specimens for virus isolation are best taken from animals in the early febrile stages of the disease. Whole blood samples (at least 10 mL) from up to 5 affected horses, should be collected in EDTA (anticoagulant).

From dead animals, a portion of spleen, taken as cleanly as possible, should be put into glycerol buffer, pH 7.4. To assist diagnosis, specimens of spleen, heart, brain, liver and kidney should be collected into neutral buffered formalin for histopathology.

For horses in the convalescent stages, blood samples for antibody detection must be collected. These samples should be at least 20 mL for the collection of serum.

Transport of specimens

Blood and unpreserved tissue samples for virus isolation should be forwarded to the laboratory on water ice or with frozen gel packs. For further information see the **Laboratory Preparedness Manual, Section 6 and Appendix 3**.

Laboratory diagnosis

Confirmation of a diagnosis of AHS and determination of the serotype involved can be made at AAHL.

The diagnostic tests currently available at AAHL are shown in Table 1.

1.4.4 Differential diagnosis

AHS must be differentiated from other diseases causing sudden death, respiratory distress or oedema.

The diseases to be included in the differential diagnosis relevant to the primary clinical signs include:

Sudden death:

- adverse drug reaction
- acute fulminating colitis
- snake bite
- pneumothorax
- toxic plants and chemicals
- endotoxaemia
- monensin toxicity

- anthrax
- equine encephalosis (also exotic)

Peripheral oedema:
- lymphatic obstruction
- trauma
- cellulitis
- parasitism
- hypersensitivity with urticaria, eg drug reaction
- vasculitis
- purpura haemorrhagica
- protein losing enteropathy
- phenylbutazone (PBZ) toxicity
- renal failure
- heart failure
- equine viral arteritis
- equine infectious anaemia
- equine babesiosis

Respiratory distress:
- anaphylaxis
- pneumonia/pleuropneumonia
- choke
- tumour of respiratory tract

Table 1 Diagnostic tests currently available at AAHL for African horse sickness.

Test	Specimen required	Test detects	Time taken to obtain result
Virus isolation in tissue cultures, eggs and animals, and serological identification, and EM	whole EDTA blood/ spleen	virus	up to 2 weeks
Competitive and indirect ELISA	serum	group-specific antibodies	1 day
Polymerase chain reaction	whole EDTA blood/ spleen	viral RNA	2 days
Indirect sandwich ELISA	spleen	viral antigen	1 day
Serum neutralisation	serum	serotype-specific antibodies	5 days
Transmission test in susceptible horses	whole EDTA blood	virus	up to 2 weeks

Source: Information provided by AAHL, 1995 [refer to AAHL for the most up-to-date information].

1.5 Resistance and immunity

1.5.1 Innate and passive immunity

All equines are susceptible to AHS virus, however manifestation is most severe, and mortality highest, in horses, less in mules and lowest in donkeys and zebras. Zebras have a higher degree of innate immunity.

In the endemic regions of Africa, donkeys are very resistant and experience a subclinical infection. European and Asian donkeys are moderately susceptible and a mortality rate of 10% may be observed. Zebras are also markedly resistant and, apart from a mild fever, show no clinical signs of infection. Subclinical infection of elephants in Kenya has been demonstrated through serological surveys.

Passive protection against AHS virus has been demonstrated. There is a good correlation between the level of AHS virus antibody in the sera of mares, in their colostrum, and in sera of their foals. The rate of decline of this passively acquired antibody is proportional to the initial titre. Hence, if antibody titres to individual serotypes of AHS virus are initially low, these titres may decline to undetectable levels by 2–4 months after birth. Although vaccination of foals at this age results in a weak antibody response due to interference from maternal antibody, it does boost pre-existing antibody levels. Hence, early vaccination may be recommended in the face of an AHS outbreak to ensure adequate protection of young foals.

1.5.2 Active immunity

Lifelong, protective immunity against the infecting AHS virus serotype is observed in equines recovered from AHS. However, there are no definitive studies that specify the source of this protective immunity (humoral immunity, cellular immunity or a mixture of both).

There are nine different serotypes of AHS virus and immunity is serotype specific, but there is some degree of cross-protection between serotypes. Antibodies are generated to a number of different proteins induced by AHS virus. These include VP2 and VP5, which are the major outer viral capsid proteins and are responsible for inducing virus neutralising antibody. These proteins are not conserved among the nine different AHS virus serotypes and this accounts for the lack of cross-protection.

Other types of antibody demonstrated in animals exposed to AHS virus include complement fixing antibody (CF), precipitating antibody and haemagglutination inhibiting antibody. Studies have shown an early appearance of neutralising antibodies combined with persistence of high titres of neutralising versus CF or other antibodies. Neutralising antibodies first appear in infected animals 15–18 days post-infection. There is a strong association between production of neutralising antibody and protection of horses against AHS (Burrage and Laegreid 1994).

Cell-mediated immunity

The natural role of cell-mediated immunity in AHS is unclear and has not been investigated. For bluetongue virus (another orbivirus) cellular immune responses have been demonstrated and have been shown to be broadly reactive, but shortlived. However, as sheep can resist challenge with active virus in the absence of neutralising antibody, it is thought that cell-mediated immunity probably also plays an important role in protection against the orbiviruses.

Interferons

Bluetongue virus has been shown by some to be a potent stimulator of interferons though similar experiments have not been performed for the other orbiviruses. It is thought that lymphokines may cause sheep to be temporarily refractory to infection with a second virus type during early infection with an initial serotype and may explain why dual infections are uncommon in individual animals when multiple serotypes are active in a livestock population. Similar responses may occur with AHS virus.

1.5.3 Vaccination

Important attributes of an effective AHS vaccine include the induction of a high level of protection against death and clinical disease and the ability to prevent viraemia or minimise the titre and duration of viraemia to avoid infection of insect vectors. Vaccines can be used to induce immunity in susceptible animals or to produce a barrier of resistant animals.

Blanket vaccination in a restricted area (RA) (see Appendix 1) would be a valuable strategy in the control of AHS to produce a barrier of resistant animals between infected and free zones and to prevent clinical disease. The aim must be to achieve and maintain a high level of population immunity. Three types of vaccines can be considered: inactivated ('killed'), attenuated ('live') and recombinant virus vaccines, each of which is discussed below.

It should be noted that vaccination prevents disease but not viraemia in vaccinated horses. However, the level of viraemia is considerably lower than in non-vaccinated horses and in most instances is below the level thought to be necessary to transmit to the insect vectors (10^4 $TCID_{50}$/mL) (Dubourget et al 1992).

Inactivated vaccines

Inactivated virus vaccines for AHS do not revert to virulence, do not cause a significant viraemia in inoculated animals, and do not reassort with wild-type orbivirus strains in the field. With inactivated vaccines, it may be possible to differentiate antibody elicited by the vaccine from that resulting from infection with an active virus, which would allow free international movement of equines.

The first inactivated AHS virus vaccines were prepared by adding formalin to infected horse tissue emulsions and have been used experimentally since 1929. More recently, this technique has been superseded by production of inactivated AHS virus vaccines using purified formalin-treated virus prepared in cell culture on an industrial scale.

This vaccine is commercially available as *Equipest* (a formalin-inactivated, aluminium hydroxide adjuvant AHS-4 virus vaccine) from Rhone Merieux Laboratories, Lyon, France. Under field conditions this vaccine is safe and potent, including on its use in mares.

Attenuated vaccines

Vaccination with attenuated *monovalent* serotype AHS vaccine produces a solid immunity that probably lasts indefinitely. In Spain about 10% of animals immunised for the first time with an attenuated monovalent AHS virus serotype 4 failed to seroconvert. However, at least some animals that failed to respond serologically were resistant to challenge infection.

Vaccination with an attenuated *multivalent* vaccine (containing AHS serotypes 1–6) produced a neutralising antibody response to all 6 serotypes and demonstrated that a

higher overall titre could be induced in most horses by multiple immunisations. Furthermore, some horses failed to respond to one or more of these serotypes despite the numerous immunisations and no cross protection against serotypes 7–9 was observed. The inability to respond to all virus strains in a polyvalent vaccine is thought to be due to 'antigenic competition' or over-attenuation of vaccine strains. For these reasons annual vaccination of horses in endemic AHS areas is advocated. In South Africa two quadrivalent vaccines are produced; one containing serotypes 1, 3, 4, and 5; the other serotypes 2, 6, 7, and 8. Serotype 9, which is rare in South Africa, is covered by cross protection from serotype 6.

Most recently, polyvalent and later, monovalent (AHS-4) attenuated virus vaccines from South Africa have been used during the 1987 through 1990 AHS outbreaks in Spain. While the disease has recurred — although it now appears due to overwintering of the vector (Rawlings and Mellor 1994) — during three consecutive years in the province of Andalucia, Spain, it appears that the vaccine has been safe and effective when properly applied in vaccinating equines (Walton 1992). Vaccine-related deaths of small numbers of horses due to AHS have occurred in Qatar.

However, the use of attenuated vaccines in areas where the disease is considered exotic has several disadvantages including:

- vaccination with live virus may give rise to difficulties in the export/import of horses;
- the risk of recombination of the vaccinal strain with field virus that could give rise to a new strain of virus of high virulence;
- the potential problem of reversion to virulence;
- pregnant animals vaccinated with attenuated vaccines may abort or produce foals with congenital abnormalities;
- the vaccine must be made from the serotype(s) responsible for the outbreak of clinical disease;
- substantial logistical problems are associated with maintaining stocks of live attenuated virus of all required serotypes 'on the shelf' and would be a substantial recurring cost;
- some vaccine-related deaths have occurred in Qatar (see above).

For these reasons the use of attenuated vaccines is not recommended in Australia (see Section 2.2.9)

Recombinant vaccines

Work on vaccines incorporating the virus proteins VP2, VP3, VP5, and VP7 is being done. VP7 is thought to be a group-specific antigen.

1.6 Epidemiology

AHS is a non-contagious viral disease of equines which is transmitted by blood-sucking arthropods. Dogs can be infected by the ingestion of fresh infected horsemeat.

1.6.1 Incubation period

The incubation period may be as short as 3–5 days for the acute or pulmonary form but is usually from 5–9 days. Incubation periods of up to 21 days have been recorded. The OIE

maximum incubation period for AHS, for regulatory purposes, is 40 days (see Appendix 3).

1.6.2 Persistence of virus

General properties/environment

The AHS virus is very stable outside the host. However, this is of little epidemiological significance (see Section 1.6.3). The virus has the following properties (Coetzer and Erasmus 1994):

- an optimal pH for survival of 7.0–8.5; the virus is sensitive to acid pH values but is relatively resistant to alkaline pH conditions;
- resistant to ether and other lipid solvents;
- relatively heat stable; the infectivity of citrated plasma containing AHS virus is not inactivated by heating at 55–75°C for 10 minutes;
- can be stored for at least six months at 4°C in saline containing 10% serum; and
- not destroyed by putrefaction and may retain infectivity in putrid blood for more than two years (Coetzer and Erasmus 1994). Virus can be recovered for 12 months from washed erythrocytes stored at 4°C.

AHS virus does not contain lipid and is resistant to detergents. Acid disinfectants are most suitable for decontamination procedures (see Section 2.2.8).

Live animals

Generally, virus can be detected in blood for 4 days before and 2 days after clinical signs are first observed. Persistence of the viraemic state for up to 49 days has been observed in vaccinated horses. Viraemias of 90 days were reported by early researchers before the multiplicity of virus serotypes was realised but lengthy viraemias in convalescent horses have not been confirmed by later work. In the more resistant hosts (donkeys and zebras) viraemia can persist for as long as 4 weeks.

Animal products and by-products

It can survive in frozen but not salted meat. At pH value below 6.0, that is, at the sort of pH usually found in meat which has undergone *rigor mortis*, the virus of AHS is inactivated quickly. It is also inactivated by temperatures greater than 60°C (MacDiarmid 1991).

Fomites

As AHS is a non-contagious disease, fomites generally do not present a risk. However, mechanical transmission could possibly occur via veterinary equipment, multiple dose vials and hypodermic needles contaminated by AHS infected blood.

Vectors

It is generally accepted that vectors that become infected with an arbovirus remain so for life (the life of an adult is about 20 days).

Newly-emerged adult culicoids (midges) take a blood meal within 1 day of emergence from the larval stage and then may take a blood meal every 3–4 days. AHS virus multiplies and reaches a high titre in *C. imicola* on the fifth day after ingestion of infected blood. This midge has been able to transmit infection to other horses 7–13 days after feeding on an infected horse. The shortest period between a midge biting an infected horse and the disease being seen later in another horse bitten by that midge could be from 12 to 16 days (mean 14 days).

Transmission of virus to embryonating chick eggs has been shown 7 days after feeding by colonised *C. variipennis.*

Experimentally-infected mosquitoes transmitted AHS virus to horses 15–18 days post-infection. In some mosquitoes, virus was still present 35 days after infection. However, field observations indicate that mosquitoes are unlikely to play a natural role in the ecology of the virus.

1.6.3 Modes of transmission

Live animals

AHS is not a contagious disease. It is transmitted between susceptible animals by blood-sucking insects. It is not spread by aerosol or direct contact between infected and non-infected animals.

Virus replication occurs mainly in the lungs, spleen and lymph nodes. Viraemia generally parallels the fever in horses and mules. The average duration of viraemia after natural infection in these species is about 4–8 days and rarely as long as 18 days.

Although virus is present in urine, milk and other body secretions of infected animals, no transmission of disease by contact, inhalation or ingestion of these materials is known.

Artificial breeding

The virus is present in semen and embryos and might therefore be transferred this way, although there is no documentation to support transmission in either (see the **Artificial Breeding Centres Enterprise Manual**).

Animal products and by-products

AHS is an insect-borne disease and cannot be transmitted between horses by contact. However, if fresh uncooked meat from clinically or subclinically-infected horses is fed to dogs, they may become infected and serve to infect vectors before themselves dying from the disease (MacDiarmid 1991).

Fomites

Mechanical spread of the virus could occur by the use of contaminated needles, multiple-dose vials and veterinary equipment.

Biological transmission by vectors

Insect vectors provide the natural means of transmission of AHS. Biting midges of the family *Culicoides* are recognised as the most important vectors. In South Africa and Spain, *C. imicola* has been incriminated as the species most likely to be involved. At least two other species have also been implicated as vectors in Spain. *C. brevitarsis,* a close relative of *C. imicola,* is widespread in Australia where it will feed on horses in large numbers, often causing an allergic dermatitis known as Queensland itch. The competence of Australian species of *Culicoides* for AHS is unknown but it must be assumed that *C. brevitarsis* is competent.

The principal North American bluetongue vector, *C. variipennis* has also been shown experimentally to be a competent vector of AHS.

AHS has been *experimentally* transmitted to horses by three species of mosquito, *Aedes aegypti, Culex pipiens* and *Anopheles stephensi,* the former two of which occur in Australia. AHS virus has also been isolated from the brown dog tick, *Rhipicephalus sanguineus,* after it fed on experimentally-infected dogs, and the camel tick, *Hyalomma dromedarii,* collected in the field. Both these species have transmitted the virus

experimentally, the former (which occurs in Australia) to dogs and horses and the latter to camels.

Mechanical transmission by vectors

There is no evidence of mechanical transmission by vectors. However, this means of spread, particularly by stable flies, buffalo flies and March flies, should not be ruled out.

Vertical transmission in vectors

Transmission of AHS virus from adult insect vectors into eggs (transovarial transmission) has not been demonstrated in any insect or tick. It has been suggested, but not proven, as an overwintering mechanism. However, field evidence strongly suggests that vertical transmission, and hence overwintering in temperate regions, does not occur (Rawlings and Mellor 1994). In the camel tick *H. dromedarii*, AHS virus can be transmitted after passing from larva to nymph and from nymph to adult (transtadial transmission).

1.6.4 Factors influencing transmission

Environment/climate

As the main means of spread of AHS virus is by biting midges, conditions that favour the presence of large populations of these insects are required for epidemics of the disease to occur. Favourable conditions are high temperatures and humidity after widespread rains. In South Africa, AHS seldom occurs at altitudes over 500 metres although there have been exceptions under unusual conditions of temperature and rainfall. In Spain, Portugal and Morocco, outbreaks have occurred in low-lying, moist warm areas where vectors are prevalent. In temperate areas, the peak incidence of the disease occurs in late summer and early autumn and it invariably disappears after the first heavy frosts (see Appendix 3, Article 2.1.11.2.).

Vector activity

In Australia, the principal potential vector of AHS is *C. brevitarsis*, because of its close relationship with *C. imicola*, the main vector in South Africa and Spain. Where it is abundant, *C. brevitarsis* will feed on horses in very high numbers. As many as 2000 have been collected off one horse around sunset. 'Queensland itch' is an allergic reaction to the bite of this species. In eastern Australia, *C. brevitarsis* numbers reach a peak in summer and autumn, with the peak occurring later in the season at the southern end of the distribution. In northern Australia, *C. brevitarsis* is present throughout the year, and numbers may be higher in the dry season than the wet season. Figure 1 shows the maximum known range of *C. brevitarsis* based on insect collections over many years to June 1990.

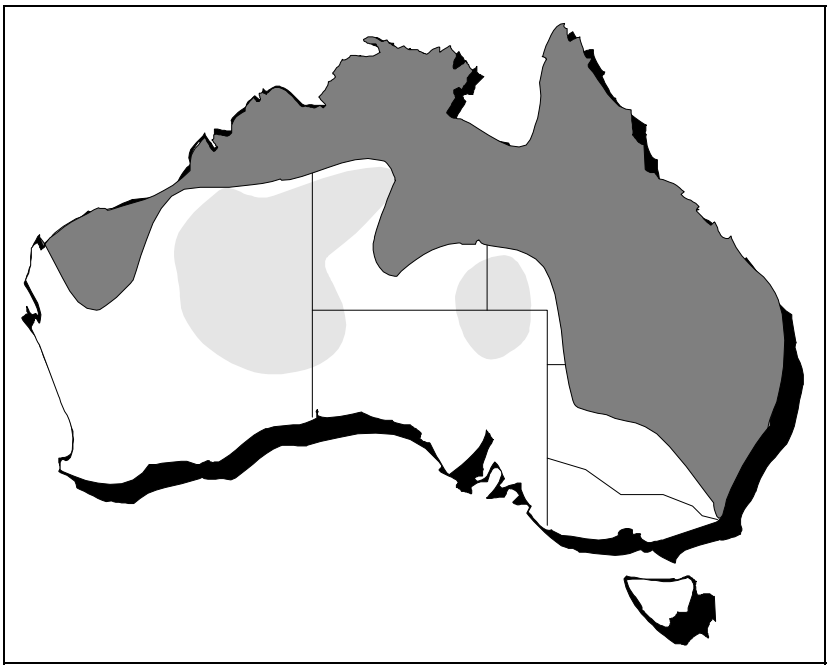
Culicoid midges generally feed in the twilight periods after sunset and before sunrise on fine clear nights. In overcast and cloudy conditions or in cooler weather, biting activity will occur in late afternoon, before sunset, and in the mornings after sunrise. *C. brevitarsis* prefers to bite along the back of horses, whereas mosquitoes are more likely to bite around the legs. Biting activity is reduced by windy conditions or at temperatures lower than about 16–18°C. *C. imicola* breeds in shallow water and mud and is associated with watercourses, dams and low-lying ground. *C. brevitarsis* breeds in cattle dung, so the ecologies of the two midges vary considerably.

No specific attempts have been made to determine the range of biting insects that feed on horses, but it can be assumed horses are attacked by a broad spectrum of insects that feed on mammals. Overnight housing of horses provides a means for controlling vector access to horses (see Section 2.2.11).

Windborne spread of vectors

Analyses of meteorological conditions suggest that windborne spread of infected vectors may have been responsible for a number of outbreaks of AHS in Spain, around the Mediterranean, and in the Middle East and India. Distances involved varied from 40–700 km.

In Australia there is strong evidence that bovine ephemeral fever and Akabane viruses have been carried over long distances by windborne dispersal of vectors. Given favourable meteorological conditions, spread of AHS by wind could be expected to occur in Australia. However, the likelihood of this occurring is probably minimal given horse population and density numbers are much lower than those of cattle in Southeast Asia, Indonesia and Northern Australia.



Prepared by CSIRO Division of Tropical Animal Production 1994

Figure 1 Maximum known distribution of *C. brevitarsis*

1.7 Manner and risk of introduction

AHS has a demonstrated ability to spread regionally and internationally. Potentially, AHS could be introduced into Australia by infected vectors, hosts, vaccines or genetic material (in the order of most likely possibility).

Introduction by vectors

Routine quarantine procedures for the disinsection of all inbound international aircraft reduce the risk of introduction of infected vectors by this means.

AHS is spread between areas by the movement of host or vector. Most outbreaks have occurred following the movement of infectious animals into areas where favourable vectors and a susceptible horse population exist. In some outbreaks, the transport of infected vectors by wind has been implicated.

Wind can play an important role in dispersing infected insects and has been implicated in the spread of previous AHS epidemics, which, in 1960, extended as far as northeastern

India. If a future AHS epidemic extended further eastward to countries to the north of Australia, infected vectors could be carried by wind into areas of northern Australia where potentially competent vectors and large numbers of feral horses and donkeys exist. Such extension of an epidemic is considered unlikely because it is thought there are insufficient equines and no reservoir hosts in Asia to sustain an epidemic. However, the insidious windborne spread of serotypes of the closely-related virus, bluetongue, from Asian countries to northern Australia suggests that, as the horse industry in Asia expands, the health status of the region should be closely monitored for any future AHS epidemic spreads into the Indian subcontinent.

Introduction by hosts

AHS has also been spread into areas free of the disease by the movement of inapparently infected animals. The 1987 outbreak in Spain is thought to have been associated with the importation of zebras from South West Africa. AHS is highly unlikely to be introduced to Australia in this manner due to stringent quarantine controls over the importation of equine species (including zoo animals), and because Australia does not import equines directly from countries or zones affected by AHS.

Introduction by vaccines

Import and use of any AHS vaccine in Australia is prohibited and any equine vaccines approved for import must originate from an approved source and undergo testing for the presence of adventitious viruses.

Introduction by genetic material

Spread of AHS by genetic material has never been documented but interest in the international transport of horse semen is increasing. As advances in equine reproductive technology occur, future quarantine protocols should address the risks of introduction of AHS by genetic material.

2 PRINCIPLES OF CONTROL AND ERADICATION

2.1 Introduction

Before embarking on any control strategy, the initial outbreak must be investigated thoroughly. All environmental factors and recent weather conditions should be recorded. The presence, numbers and movements of equine species onto and off the property and adjacent properties should also be recorded.

Control of AHS relies on four basic principles:

- preventing contact between susceptible animals and AHS virus;
- stopping the production of virus by infected animals;
- stopping the production of virus by insect vectors; and
- increasing the resistance of susceptible animals.

These principles can be applied by:

- quarantine and movement controls to stop the spread of infection (see Section 2.2.1)
- animal management to limit vector exposure (see Section 2.2.11);
- eliminating sources of infection by slaughtering infected and exposed animals, sanitary disposal and disinfection (see Sections 2.2.5, 2.2.7 and 2.2.8);
- initiating vector control (see Section 2.2.11);
- establishing immunity by vaccination (see Section 2.2.9).

2.2 Methods to prevent spread and eliminate pathogens

2.2.1 Quarantine and movement controls

Effective quarantine and movement controls are essential to prevent the spread of virus by animals. Even if the virus has become established in an insect vector population, it will still be necessary to reduce the spread by animal movements. Initially, stringent controls on the movement and congregation of susceptible livestock should be imposed. These may be reduced once the situation has been fully investigated.

Quarantine and movement controls should be imposed at several levels. Infected premises (IPs) and dangerous contact premises (DCPs) will be identified and a *restricted area* (RA) will be drawn around all IPs and DCPs. The distance in any one direction is determined by factors such as livestock concentrations, weather conditions and prevailing winds, the distribution and movements of susceptible wild animals and the presence of possible vectors. A high level of movement control and surveillance will apply.

A *control area* (CA) will be imposed around the RA. The CA must include all premises adjacent to known IPs (in settled areas adjacent premises are likely to be part of the RA). The purpose of the CA is to control movement of susceptible livestock for as long as is necessary to complete trace-back and epidemiological studies. Less stringent movement control and surveillance will apply. Once the limits of the disease have been confidently defined, the CA boundaries and movement restrictions should be relaxed or removed. However, if the disease becomes widespread in an insect vector population, the CA may need to be expanded to include that vector's known geographical range.

Movement controls should be maintained to some degree until the disease is either eradicated or declared endemic. If a vaccination campaign is carried out, the restrictions on vaccinated animals (once their immunity is established) will be far fewer than on non-vaccinated animals.

While movement restrictions remain in place, the value of artificial insemination as a strategy to permit mating to minimise industry disruption needs to be considered.

For further information on declared areas and movement controls see Appendixes 1 and 2.

Zoning

Zoning, in accordance with the requirements of the OIE Code (Appendix 3), is an important strategy to reduce the economic and social impact of AHS by freeing up export markets and by allowing continuation of local, national and international competition and movement of horses originating from outside the infected zone.

The area at risk to AHS may be determined by the geographical range of competent insect vectors and, in particular, the range of competent *Culicoides* spp. If it can be established that the disease has been introduced to, and vectors are limited to, a particular geographical region of Australia, then control procedures can be principally confined to that region or zone (see Appendix 2).

2.2.2 Tracing

Urgent and meticulous trace-back and trace-forward of all contacts with infected animals and premises is vital if the disease is to be effectively contained. Tracing should include:

- all horses, donkeys, mules, zebras, etc;
- equine products where virus is likely to persist and be potentially infective (eg blood, semen, embryos); and
- collection of insects for identification and virus isolation.

It is possible that the first reported animal case will not be the index case, and trace-back will identify other cases. Surveillance of both livestock and vectors is necessary to assess the extent and likely spread of the disease.

2.2.3 Surveillance

Livestock surveillance

Equines in the RA should be observed daily for clinical signs of disease and blood taken at weekly intervals from a statistically valid sample of animals and tested for antibody to AHS virus. This testing should commence following diagnosis of the index case and continue for as long as necessary following the last confirmed case (see Appendix 4).

Serological monitoring should then be continued at monthly intervals for the next 12 months, and then quarterly for a further two years (see Section 1.4 for information on specimen collection and laboratory testing).

Vector surveillance

In the event of an outbreak, surveillance for vectors can be for virus isolation and/or to record the current population of biting insects. Collection for virus isolation is both labour and expertise intensive. Currently, it is limited by the personnel available with the taxonomic capacity to accurately identify potential vectors collected as live insects as these should be identified immediately. Laboratory capacity is limited and only relevant species need to be examined.

Collections made purely for population analysis can be made by non-expert personnel after brief instruction. An adequate number of carbon dioxide baited light traps should be available at short notice. A number of local councils and State/Territory departments of health currently use such traps for arbovirus surveillance. Collections should be stored in suitable condition for later identification. It may also be possible to process these insects for virus isolation with techniques currently being developed. Available expertise will be a limiting factor for the sorting of these collections, which may be a lengthy process.

A range of collection techniques, including carbon dioxide light traps, truck traps, animal baits and larval sampling, would be necessary (see Appendix 5).

2.2.4 Treatment of infected animals

Young horses less than 6 months old seldom respond to treatment and mortality rates approach 100%. However, in several hundred cases of AHS in Zimbabwe, older horses treated symptomatically with diuretics, cortisone or other palliative treatments showed around a 50% recovery rate. The severity of the clinical symptoms and recovery were not related and whereas some cases with severe signs of AHS recovered, some apparently mild cases did not. However, once white froth appears out of the nostrils the animal usually dies, whereas the less acute cases (with swollen heads) are probably worth treating (pers. comm. Alastair 'Tink' Robey, WA veterinarian, formerly of Zimbabwe).

Any treatment of infected horses must take account of the possible spread of virus in the vector population and must be associated with control of insects in and around the area as outlined in Section 2.2.11.

2.2.5 Destruction of animals

The selected strategy for AHS of stamping out would involve destruction of all susceptible animals on an IP. Its implementation would be highly dependent on whether compensation was available (see Section 3.5) and is complicated by the high monetary, genetic and sentimental value of many horses. This strategy would be of value for an index case in an area of low epidemic potential, ie where it was believed it was unlikely that AHS would become established in a competent insect vector or uncontrolled feral horse or donkey population, or would not become established due to geographic or climatic factors.

Clinically-affected horses may die rapidly or are likely to have to be destroyed for welfare reasons. On an IP, there may be circumstances when it may not be appropriate to destroy individual horses with suspicious clinical signs (eg fever) until a laboratory diagnosis has been established or the disease has progressed to demonstrate unequivocal clinical signs (see the **Destruction of Animals Manual).**

2.2.6 Treatment of animal products

Horsemeat should be cooked before feeding to dogs.

2.2.7 Disposal

Burial or rendering rather than cremation are the preferred methods for disposal of carcases. Factors such as topography, soil type and water table depth must be considered when selecting a burial site. Carcases should not be sent to knackeries for petfood because of the risk to dogs from AHS carcases (see **Disposal Procedures Manual, Sections 3.1 and 3.5**).

2.2.8 Decontamination

Fomites do not normally present a risk. Disposable needles should be used for collection of blood samples and precautions taken to avoid mechanical spread. Because AHS is not contagious, decontamination of inanimate objects is not important for control of the disease. If it is considered necessary to decontaminate blood-contaminated areas or equipment, acidic disinfectants such as 2% acetic or citric acid are recommended for the inactivation of AHS virus. Both these substances are mildly corrosive for metal objects and may leave a sticky residue on rubber objects unless well washed with water. Acetic acid can be purchased as vinegar (4% acetic acid) or as 99.5% glacial acetic acid. Dilute these substances with water for application as a 2% solution.

For further information see the **Decontamination Manual, Tables 2.1, 3.1 and 4.**

2.2.9 Vaccination

One of the main measures used to control the 1987–90 AHS outbreaks in Spain was a vaccination program for all susceptible equines in the outbreak area, using an attenuated monovalent serotype AHS-4 vaccine.

AHS virus vaccines presently used in the world (Africa mainly, and Portugal and Spain recently) are prepared with attenuated AHS virus strains cultivated in mammalian cell lines. These vaccines are usually safe and effective, and, in newly-infected countries, their widespread use seems to impair the development of further outbreaks.

However, due to the disadvantages associated with the use of attenuated vaccines (see Section 1.5.3), it may be preferable to recommend the storage and use of inactivated virus vaccines for protection against AHS in Australia.

It should be noted that vaccination prevents disease but not viraemia in vaccinated horses. However, the level of viraemia is considerably lower than in non-vaccinated horses (see Section 1.5.3).

Vaccine stocks

At present there are no stocks of either attenuated or inactivated AHS vaccine available in Australia. Thus, after the initial diagnosis of an outbreak there would be a delay in the availability of vaccine. The length of time before vaccines were available would depend upon the availability of vaccine worldwide.

Vaccination schedules

It is recommended that horses should be vaccinated twice in their second year of life because repeated immunisation boosts antibody levels. However, there appears to be no benefit in vaccinating more than once annually thereafter since too frequent administration of vaccine can apparently lead to unresponsiveness or even hypersensitivity.

Identification of vaccinates

To comply with the OIE Code (eg Article 2.1.11.6; see Appendix 3) and to allow future interpretation of serology results, all vaccinated animals need to be permanently identified by means of branding, passports with individual identification graphics and vaccination record pages or by being implanted with a centrally registered microchip. Freeze branding is the preferred system of identifying a vaccinated horse as it is a simple method for large numbers of horses and, at subsequent inspections, proof of vaccination is readily apparent.

2.2.10 Wild animal control

Feral horses and donkeys in Australia pose a considerable threat to AHS control as they are present in large numbers in inaccessible areas where *Culicoides* are active all year round. While feral horses in Australia would be totally susceptible and suffer high mortality rates, donkeys are said to be more resistant to AHS than horses, have lower mortality rates and longer viraemia. Donkeys could provide a mobile reservoir of virus and source of infection for vectors. The susceptibility of Australian feral donkeys and the role that feral equines would play in an outbreak, is unknown.

Tracing and surveillance of feral animals

The actual or potential role of feral animals must be assessed early in the outbreak. Initially, the distribution and abundance of feral animals should be surveyed, especially on and near IPs, to determine which feral animals, if any, are likely to have come into contact with infected vectors. The presence or absence of AHS in feral horse and donkey and camel populations should be determined by mounting trapping and shooting operations. If serological or virological evidence of AHS is found, then more extensive and systematic epidemiological studies should be undertaken to monitor the extent and spread of the disease in feral populations. If a large feral population is found to be infected in areas where *Culicoides* activity occurs all year round, eradication from that zone may be very difficult. Primary efforts should be then directed at establishing a buffer zone to protect free areas.

Depopulation of feral animals

Depending on the circumstances, in some areas depopulation of feral horses and donkeys may be an option. Initial efforts should be directed toward feral donkeys. Depending on the terrain, trapping or shooting using trained marksmen in helicopters are the recommended methods of control (for further detail see the **Wild Animal Control Manual, in press**).

2.2.11 Vector control

In the event of an outbreak, the decision to conduct vector control will depend on the particular circumstances. For a single case in or near a large metropolitan area there may be no need for vector control. For an isolated case in a rural area, it is possible that reduction of potential insect vector populations would be attempted as rapidly as possible if the incident remained localised. Aerial spraying and ground application of insecticide as ultra low-volume (ULV) fogs would be considered initially. In addition, attempts should be made to modify the environment to reduce breeding sites for vectors, eg clean up manure, urine and surface water from around stables.

The most readily accessible source of vector control expertise and equipment is with State health departments, local government authorities or the Australian Plague Locust Commission. The chemical used should be suitable for vector control and readily available in large quantities.

Application of insecticide to the environment

In the event of spraying being undertaken, care should be taken to advise all the appropriate persons/groups, including the local council, local landholders, police and apiarists operating in the area. Appropriate protective equipment must be provided and its use made compulsory for staff involved in insecticide applications. Staff must follow

recommended safety guidelines. First aid measures and material safety data sheets for the chemicals being used must be on hand.

Efficiency of vector control may be limited by lack of knowledge of the most important target species (ie the vectors of most concern) and thus an inability to focus control actions on the relevant species.

Ivermectin treatment of livestock

Treatment of livestock (including cattle and sheep) in the restricted area or even on the IP and neighbouring premises with either a systemic insecticide such as ivermectin or a topical insecticide will also reduce the population of some of the potential vector species particularly *C. brevitarsis*. However, as these chemicals have withholding periods that need to be observed, eg for ivermectin administered subcutaneously, the withholding period for meat for human consumption is 42 days and 28 days for milk and milk products, it is unlikely that this action would be taken to reduce culicoides populations across a large area.

Housing

Where animals are held in stables, the interior should be treated with an appropriate insecticide. If possible, animals on open pasture should be moved to stables or some form of covered shed, which should also be treated with insecticide. Sheds fitted with insect gauze would be ideal, but even open-sided sheds may help to limit insect exposure, especially to biting midges. Where animals remain on open pasture, they should be rugged and/or treated with a suitable insecticide to reduce exposure to biting insects.

In fully enclosed sheds, ultraviolet light insectocutors may help to control vectors. These insectocutors should only be used in sheds where the light is not visible to the outside. They should **NOT** be used outside sheds, as they will attract insects from the open to the vicinity of the shed, and do not reduce biting rates under these circumstances. Other lights in sheds should be kept off as much as possible.

All vehicles leaving the site of the outbreak should have the interior treated with an aerosol insecticide to prevent spread of potential vectors. For further details of vector monitoring and control, see Appendix 5.

2.2.12 Sentinel and restocking measures

Restocking can be considered when the risk of spread from infected insects has diminished and/or a vaccine is available to protect animals prior to introduction. For further information on the use of sentinel animals see Appendix 4.

2.2.13 Public awareness

On account of the emotive aspects of a large-scale epidemic of acute horse deaths, which could involve domestic pets, it will be necessary to ensure that the public is kept fully and accurately informed.

The pastoral industry, horse industry and zoo personnel must also be advised of the symptoms of AHS and the action they should take if they suspect the disease. Details should be given of the movement restrictions imposed. Media information should stress that cooperation by the horse industry is critical to control the disease. Extension efforts should also be directed at knackery owners who process horsemeat for consumption by domestic pets.

A media information kit similar to those recommended in the **Public Relations Manual, Appendix 1** should be available as soon as a positive diagnosis of AHS is confirmed. Industry contact lists for the equine industry can be obtained from the Australian Horse Council and State departments with responsibility for horse racing.

Special kits should be available to circulate to veterinarians via the Australian Veterinary Association and Australian Equine Veterinary Association. Early contact should also be made with, and detailed information distributed to, horse industry bodies such as the State Horse Council (if one exists in the affected State) or Australian Horse Council, the Conference of Principal Racing Clubs, the Australian Harness Racing Council and the Equestrian Federation of Australia. The Australian Horse Council should be able to advise contact points for other national horse industry organisations.

2.3 Feasibility of control in Australia

Feasibility of control depends on the location of the index case. If the index case occurs in an area with low epidemic potential, is detected rapidly and transmission to insect vectors and hence other equines is prevented, AHS could be eradicated from Australia.

However, as the most likely means of introduction of AHS is by windborne spread from Asia, the index case is likely to occur in northern Australia, which is an area with a high potential for an epidemic. Unless the outbreak runs its natural course and 'burns out' due to the lack of a suitable reservoir and/or a sufficient number of susceptible animals, AHS may be very difficult to eradicate from this region.

3 POLICY AND RATIONALE

3.1 Overall policy for African horse sickness

African horse sickness (AHS) is an OIE List A disease that has the potential for serious and rapid spread and is important in the international trade of horses.

The policy is to eradicate the disease if circumstances permit. Eradication would be feasible if there is early detection of the disease in isolated animals and there is an absence of infected vectors, or if the disease occurs in a vector-free area (or if frosts were imminent in vector areas). If the disease occurs in a competent vector-inhabited area early in the vector season, then eradication will be difficult.

A combination of strategies will be used in the eradication or control of the disease including:

- ***judicious slaughter of clinically affected animals;***
- ***quarantine and movement controls* on animals in declared areas to contain infection;**
- ***treatment and husbandry procedures* to control vector attack on susceptible animals in declared areas;**
- ***vector control* over an area may be considered;**
- ***vaccination*, which will be the main disease control strategy if infection establishes in insect vectors;**
- ***zoning* to define infected and disease-free areas;**
- ***a public awareness campaign* to facilitate cooperation from industry and the community.**

An outbreak of African horse sickness could have a major economic impact on individuals, the horse industry and governments by disrupting horse racing and other equestrian activities. Even if it was confined to a small area, there would be severe disruption to horse exports until free zones were established.

AHS is not in the Commonwealth/States cost-sharing agreement.

The CVO(s) in the State(s)/Territory(s) in which the outbreak(s) occurs will be responsible for implementing disease control measures (in accordance with relevant legislation), and will make ongoing decisions on follow-up disease control measures in consultation with the Consultative Committee on Exotic Animal Diseases (CCEAD), the State/Territory and Commonwealth governments, and representatives of the affected industries. The detailed control measures adopted will be determined using the principles of control and eradication (Section 2) along with epidemiological information about the outbreak. For further information on the responsibilities of the State/Territory disease control headquarters and local disease control centre(s), see the **Control Centres Management Manual**.

3.2 Strategy for control and eradication

Unless the disease can be found before the virus has become widespread in an insect vector population or the index case or cases occur in an area subject to seasonal vector kill, the eradication strategy would be based on vaccination and a level of quarantine and movement control during the outbreak and the following period. Other strategies which will be used include slaughter of infected animals, vector control and zoning to help reduce the amount of virus and spread of infection.

Because of the individual value of some horses and the emotional attachments involved with horse ownership, it will be necessary to ensure close liaison with industry, horse owners and the media.

3.2.1 Stamping out

Stamping out may be used where the disease is confined to a small number of animals or to a limited area, where virulent virus has been identified or strongly suspected, where there is a strong possibility of spread to a competent vector population and the chances of restricting spread of virus is good, ie a best case scenario with low epidemic potential.

Clinically-affected animals are likely to die or may have to be destroyed for animal welfare reasons. Any decision to stamp out clinically-affected animals or infected animals is warranted and justified on the basis of the devastating effects the clinical disease would have (see Section 3.2.4, below).

3.2.2 Quarantine and movement controls

Quarantine and movement controls will be imposed on the infected premises at the time of confirmation or suspicion of AHS. In more intensive animal areas the IP would include neighbouring horse properties in a declared ‘infected area’, or as a cluster of infected properties. Similar restrictions will apply to DCPs and suspect premises (SPs).

A restricted area and a control area will be declared immediately the disease is suspected. The early declaration of premises and areas permits the rapid implementation of movement controls for susceptible animals and their products to confine the virus within specified limits and to prevent the inadvertent spread of virus and infected vectors to free areas where further spread could occur. The confinement of infected or suspect animals allows time for an epidemiological investigation to be undertaken so that more informed control measures can be implemented.

The initial RA boundary should be at least 10 km from the infected animals or IPs/DCPs in the first instance on the assumption that infected vectors are probably present in the area. This can be altered as epidemiological information becomes available.

The initial CA may correspond to the State borders in the early part of the outbreak or may even extend to the limits of the range of the vector if this is known and documented. The CA boundaries can be modified as more information becomes available from the investigations.

Movement controls will be strictly enforced until such time as the status of declared areas can be redefined or until other strategies can be implemented (eg vaccination). Movement of animals out of the infected area will be strictly controlled.

For further information on declared areas and movement controls see Appendixes 1 and 2.

Zoning

Once the epidemiological information has been collated and assessed and the disease distribution has been defined, it will be advantageous to establish a zoning strategy so the international trade in equine species and their products may recommence at the earliest possible time. This should reduce the social and economic effects for a major part of the country which will remain free of the disease. The criteria to be applied should be those recommended by the OIE Code (see Appendixes 1, 3 and 4).

The infected zone should have a radius of at least 100 km in which vaccination may occur. This zone will be surrounded by a surveillance zone of at least 50 km in which no vaccination may be carried out. The actual size of the infected zone will depend on the period of time vectors are normally present and other climatic factors that may have a bearing on vector activity and distribution.

The free movement of susceptible animals out of the infected zone will be prohibited and animals will only be moved to the free zone under strict conditions.

Surveillance of the infected zone must be continued for a minimum period of two years and there must be no vaccination performed in the infected zone during the period of 12 months before declaration of freedom.

3.2.3 Treatment of infected animals

Treatment is generally ineffective but may be attempted if stamping out is not practised and there is little risk of virus spreading from viraemic animals (see Section 2.2.4).

3.2.4 Treatment of animal products and by-products

The major animal products affected by AHS that are of concern, are meat, semen and embryos. Meat from an RA should be destroyed in the early part of the outbreak. As the situation becomes clearer, meat from susceptible animals from the RA may be used if subjected to correct heat treatment.

The risk from semen and embryos has not been determined and these materials should not be collected from infected animals. Products in storage may be assessed on their merits, taking into consideration the time of collection and other factors that might cause contamination of samples.

3.2.5 Vaccination

Vaccination will play an important role in the control and eradication of AHS if the virus is present in the vector population and the disease becomes widespread. The use of vaccine will provide assurances against the spread of disease for the movement of animals both into and out of the disease control areas. All vaccinated animals must be permanently identified.

An inactivated vaccine will be the vaccine of choice and will be used in the RA and possibly the CA. Early and widespread vaccination will only be able to take place if there is a current vaccine (incorporating the right serotype) available overseas and which can satisfy Australian import requirements.

If an outbreak of AHS occurs, one of the first tasks will be to initiate action to ensure an effective vaccine is available. Sufficient quantities of homologous AHS vaccines could be available if the outbreak is associated with the right virus serotype. CCEAD would need to determine whether vaccination should be mandatory or voluntary, and hence at public or private expense. An imported vaccine would need to meet Australian quarantine standards.

3.2.6 Tracing and surveillance

Tracing and surveillance will need to be undertaken quickly to determine the source of the infection, to identify the risk premises and to define as closely as possible the extent of the infection. A full epidemiological investigation will be undertaken involving both vector and virus surveys, detailing environmental and ecological conditions leading up to the outbreak, the stage of the vector season (if present) and an assessment of the likelihood of continuing vector activity and future seasonal outbreaks. If the outbreak occurs towards the end of the vector season in an area where vector activity is reduced or eliminated by frosts then the advice may be to 'wait and see' before a full eradication and control program is instigated.

Feral horse, donkey and camel populations would need to be included in any surveys if these are present in the area where the disease occurs.

Trace-back and trace-forward must extend over a period of 40 days from the time of the first clinical signs and up to the time that quarantine was imposed. It will involve the tracing of susceptible animals from the IPs and risk premises. Any suspect horsemeat must be traced.

A major program of surveillance for virus and vectors will need to be undertaken during the initial investigations and following the last cases in order to demonstrate proof of freedom (see Appendixes 4 and 5).

3.2.7 Vector control

Vector control can play a major role in preventing vector contact with infected and susceptible animals. Animals, if in small numbers, can be individually treated and handled in ways that can limit the spread of virus from the infected source.

The housing of animals, use of ivermectin, external application of insecticide and treatment of vector breeding areas by ground and aerial spraying are all effective measures in limiting the spread of the virus.

Insect protection measures for horses and other equine species are outlined in Section 2.2.11 and in Appendix 5.

3.2.8 Decontamination

The AHS virus can survive in some products such as blood for some time (see Section 1.6.2), so it will be necessary to ensure sanitary conditions are maintained in the environment and fresh blood removed. Veterinary equipment must be cleaned and sterilised after use on infected animals. Fomites do not play a role in the transmission of AHS but vehicles should be treated for vectors if leaving the RA.

3.2.9 Wild animal control

Wild horses and donkeys and possibly camels are likely to be infected if competent vectors are present and shown to be widespread. This would be the case if *Culicoides brevitarsis* is implicated.

The wild populations of susceptible animals in the vicinity of an outbreak will need to be surveyed for the presence of antibodies or virus. It would be necessary to undertake an intensive control program to destroy wild populations of susceptible animals if they are shown to be infected.

3.2.10 Media and public relations

The slaughter of horses and the clinical effects of AHS will be an emotive issue with the public and horse-owners in particular. The high value of some animals is likely to lead to opposition to any stamping-out strategy. To ensure cooperation and support, it will be necessary to carefully and sensitively convey to industry, horse-owners and the public, information on the disease, its ramifications for the horse industry in Australia and the eradication/control measures to be employed. Any sensationalising of the eradication program would be detrimental to the successful eradication of AHS.

3.3 Social and economic effects

The diverse nature of the horse industry makes it difficult to determine the social and economic impact that the introduction of AHS and its control would have in Australia. Such assessments are further complicated by the epidemiology of the disease and the many variable factors influencing it (outlined in Section 1.6). These factors will ultimately determine the morbidity and mortality rates in the event of outbreak.

Whether it is a child's pony worth $200, or stallions, broodmares and/or racehorses worth millions of dollars that contract the disease, the impact will vary from significant emotional loss on the one hand, to possible financial disaster for the affected owners and trainers on the other. Given that the Australian horse population has not been immunologically exposed to AHS virus, it can be reasonably assumed that the mortality rates in infected horses could be very high (up to 90% or more depending on the strain of virus introduced).

A hypothetical example of an average stud farm situated in a low to moderate vector activity area where it is the only farm infected, losing a stallion and five mares could result in estimated losses of at least $1.9 million. Should this stud be one of the major breeding properties in the Hunter Valley of NSW, for example, these estimates could probably be multiplied up to ten times.

If a major metropolitan training establishment, enjoying reasonable success with a stable of 40 horses, was quarantined for two months and suffered the slaughter/death of five infected horses, the costs (to owners, trainers, riders and staff) could easily be in excess of $430 000.

In northern Australia, where there is greater availability and activity of suitable vectors combined with favourable environmental conditions, there is likely to be more widespread disease and increased morbidity and mortality.

In 1990–91 racing contributed around $6 billion to the national income, $2.4 billion or 0.63% to the GDP and directly employed 132 000 people in full or part-time jobs (ie 40 000 full-time equivalents). An estimated 330 000 people are employed directly and indirectly in the racing industry throughout Australia (ACIL 1992a, Pilkington and Wilson 1993).

If cases of AHS occur in an infected premises located, for example, in one of the major training centres in or around Melbourne, depending on the nature of the movement controls employed in the RA and CA, racing could be virtually shut down. Considering that the majority of horses in Melbourne are trained at centres in close proximity to each other, it is quite feasible that all of the approximately 2800 horses in training might be prevented from racing for a prolonged period resulting in lost training fees of approximately $4 million in one month alone. Limited racing may be able to continue in some country areas in certain circumstances but these would have little effect in defraying the financial losses.

Assuming a total shutdown of racing in Victoria for one month only, on average 77 race meetings and $243 million in betting turnover (including $14 million in direct State government revenue from gambling alone); $6.25 million in stakemoney to owners, trainers and jockeys; and $50 million to the State GDP would be lost (ACIL 1992b). There would be additional losses through the significant percentage of the full or part-time jobs and ancillary services. These losses would be greater during a major racing carnival.

An outbreak in NSW (especially in Sydney and/or the Hunter Valley region), would result in greater losses as the racing and, particularly the thoroughbred breeding industry, are bigger than in Victoria. In NSW $926 million is contributed by the racing industry to the State GDP. It is also possible that a significant percentage of Australia's overseas trade in both live horses and horsemeat (approximately $60 million in 1990–91) could be jeopardised.

None of this accounts for the tremendous social disruption and economic impact AHS would have on the probable cancellation of a wide range of equestrian events such as gymkhanas, show jumping, hunting, three-day events, polo/polocrosse tournaments, horse shows, rodeos, pony clubs, riding schools, circuses and recreational riding.

3.4 Criteria for proof of freedom

If AHS became endemic in northern Australia, efforts to restore trade should be directed towards proving the regional freedom of other areas.

The OIE Code, Article 2.1.11.2 (see Appendix 3 and 4), lays down the conditions for proof of freedom and restoration of freedom and involves an intensive surveillance program for virus and vectors within both the infected and free zones.

International trading partners may impose additional conditions, the most likely one being a serological testing protocol with high confidence limits (at least 95%) assuming a low disease prevalence and with random sampling in the whole free zone.

3.5 Funding and compensation

As AHS is not included in the Commonwealth/States cost-sharing agreement for the eradication of certain exotic animal diseases, funds to pay the costs of eradication, including compensation, will have to be found from other sources. Possible sources are:

- State government funds;
- State disease compensation funds;
- Commonwealth government funds;
- special industry levies; and
- other agreed arrangements.

Alternatively the costs and losses might have to be borne by individual owners. Thus, owners of susceptible animals, particularly in the highly-valued racing and breeding industries, must be encouraged to insure their animals against losses due to an exotic disease.

3.6 Strategy if the disease becomes established

Vaccination and zoning in accordance with OIE guidelines will be the selected strategies if AHS becomes endemic in regions of Australia. See Appendixes 3 and 4.

APPENDIX 1 Guidelines for classifying declared areas

Infected premises (IP)

A premises classified as an IP will be a defined area (which may be all or part of a property) in which AHS disease or the virus exists, or is believed to exist. An IP will be subject to quarantine served by notice and to eradication or control procedures.

Dangerous contact premises (DCP)

Premises classified as DCPs will be:

- those that have received animals from the IP during the period of 40 days before the first clinical signs and up to the introduction of quarantine; and
- premises contiguous with an IP (because of vector transmission).

Suspect premises (SP)

This term should be used sparingly. Every effort should be made to clarify the status of a property to either free, DCP or IP as soon as possible.

Restricted Area (RA)

The RA should cover an area of at least a 10 km radius around known infected animals on the infected premises; or, in extensive farming areas, this distance should be at least 50 km. The boundary of this area should follow property boundaries and where possible use barriers such as roads and railway lines. Boundaries such as watercourses, which are potential breeding sites for insect vectors, should be avoided.

It is important to prevent the spread of the disease by animal movements although some local spread may still occur due to movement of infected vectors. A distance of 10 km should ensure that the disease is contained if there are no illegal movements of animals.

Control area (CA)

The CA may be as large as the whole State initially but may be modified on the basis of epidemiological, meteorological, geographical and ecological data as it comes to hand. Even in remote areas, the CA must include the IPs and any adjacent premises.

In establishing the boundaries of the CA, consideration should be given to the long-term strategies when zones may have to be developed in accordance with the OIE guidelines (see Appendix 3, Article 2.1.11.2).

APPENDIX 2 Recommended quarantine and movement controls

Infected and dangerous contact premises	Suspect premises
Movement out of susceptible animals: Movement of all members of the horse (equine) family is prohibited. Confine dogs and prevent their access to carcases.	As for IP/DCP.
Movement in of susceptible animals: Prohibited. No restriction of genetic material from AHS free zones.	As for IP/DCP.
Movement out of specified products: Movement of horsemeat, genetic material or biological products derived from equines prohibited.	As for IP/DCP.
Movement out of other animals: All other animals to have free movement after disinfection.	As for IP/DCP.
Movement in and out of people: No restriction.	As for IP/DCP.
Movement in and out of vehicles and equipment: Vehicles leaving an IP or DCP must be disinfected. Equipment contaminated with blood or body fluids must also be decontaminated.	As for IP/DCP.
Movement out of hay, crops, grains. wool, eggs, milk and meat (other than equine): No restriction.	As for IP/DCP.
Containment of susceptible animals: Equine species to be contained in stables/stalls/insect-proof accommodation/covered sheds that are treated with insecticide/have UV insectocutors operating during dusk, dark and daybreak hours.	As for IP/DCP.

Restricted area	Control area
Movement out of susceptible stock: No equines may leave RA.	Horses may be sent for slaughter at an approved knackery under permit. Only fully immune equines may leave the CA under permit.
Movement in of susceptible stock:	

Discouraged. Genetic material from AHS-free zones allowed under permit only.	Under permit only.
Movement within of susceptible stock: Not allowed (except within a premises) except under permit. Permits may be granted to fully vaccinated horses or healthy horses consigned directly to slaughter within the RA. No movement of other equines.	Under permit only.
Movement through of susceptible stock: Not allowed.	Under permit only.
Movement of specified products: Movement of uncooked horsemeat and equine biological products originating in RA prohibited.	As for RA.
Movement of other animals, people, equipment: Movement of non-susceptible animals and people unrestricted. Equipment contaminated with blood or body fluids must be disinfected.	As for RA.
Vehicles: Disinfect aircraft after leaving RA in areas of high epidemic potential.	As for RA.
Disinfection of other vehicles encouraged if travel is at periods of peak vector activity.	
Risk enterprises: Routine activities can continue at racecourses, studs, riding schools, zoos, circuses, knackeries, etc only under permit and only during daylight hours.	As for RA.
Races, sales, shows, rodeos, etc: Cancel or hold at sites outside the RA.	Can be held under permit.
Stock routes, rights of way: No movement of equines.	As for RA.
Containment of susceptible animals: No movement of equines	As for RA.

APPENDIX 3 OIE International Animal Health Code for African horse sickness

[NB The following text is taken directly from the OIE International Animal Health Code (1992); Chapter 2.1.11. For definitions, Appendixes etc see the original text. The OIE Codes are amended every year in May. The Code for AHS was amended in 1993 to include a new section on restoration to free status at the end of Article 2.1.11.2; the amended version is shown below. There were no further amendments in 1994 or 1995.]

Preamble: For diagnostic tests and vaccine standards, reference should be made to the *Manual* (A11) [☞see OIE publications under References].

Article 2.1.11.1.

For the purposes of this *Code*, the *infective period* for African horse sickness (AHS) shall be considered to be 40 days for domestic horses.

Article 2.1.11.2.

For the purpose of this *Code*:

AHS: *free country*

A country may be considered free from AHS when the disease is compulsorily notifiable in the country, and when no clinical, serological (in non-vaccinated animals) or epidemiological evidence of AHS has been found for the past two years nor have any domestic horses or other equines been vaccinated against the disease during the past 12 months.

AHS: *free zone*

A zone of a country may be considered free from AHS when the disease is compulsorily notifiable in the whole country and no clinical, serological (in non-vaccinated animals) or epidemiological evidence of AHS has been found in the zone during the past two years nor have any domestic horses or other equines been vaccinated against the disease during the past 12 months. The free zone must be clearly delineated by substantial geographical barriers if possible, and the animal health regulations to prevent the movement of domestic horses and other equines into the free zone from an infected country or infected zone published, with notification to the OIE in accordance with Article 1.2.0.4. of Section 1.2. of the Code, and rigorously implemented. Regular inspection and supervision of movement should be made of domestic horses and other equines in the free zone to ensure freedom from AHS.

If an AHS free country or free zone imports domestic horses or other equines from an infected country or infected zone, the *importing country* or zone will not be considered infected, provided the importation has been carried out in conformity with the provisions of Article 2.1.11.6.

AHS: *infected zone*

For the purposes of this disease, an infected zone shall comprise two areas:

1) a protection zone of radius of approximately 100 kilometres around an *outbreak*;

2) a surveillance zone of at least a further 50 kilometres around the protection zone and within which no vaccination program for AHS has been carried out.

The infected zone shall be maintained for two years after the last outbreak.

The boundary between the infected zone and a free country or free zone shall not be limited by national frontiers, must be clearly defined, and must take account of geographical and ecological factors as well as all epizootiological factors which are relative to this disease. The area of the zone should be extended or reduced if necessary to satisfy the following factors:

a) Epizootiology of the disease

AHS is a non-contagious disease. It can be readily transmitted by the parenteral injection of infective blood or organ emulsion. The main natural mode of transmission is by female midges of the genus *Culicoides* of which *C. imicola* appears to be the most significant vector. In areas with a temperate climate, the peak incidence of disease occurs in the late summer and early autumn. Its prevalence is directly influenced by climatic conditions favouring insect breeding and outbreaks are abruptly curtailed with the appearance of heavy frost.

b) Ecological factors

A heavy frost involving three periods of temperature of –3°C lasting a minimum of 2–3 hours each, during a three-week period (under study) would eliminate both adult midges and hatching larvae of *Culicoides* species in an area. During an outbreak, the percentage of infected midges is extremely low and, although an infected midge may harbour a relatively large amount of virus, the potential for spread of disease by this means over long distances is extremely low.

c) Geographical factors

The activity of the midge vectors is significantly reduced at high altitudes. The presence of mountain ranges at the boundary of an infected zone will provide a natural barrier to the movement of vectors. Extensive areas of arid terrain would also serve as a natural barrier.

d) The factors to be taken into account in delineating the extent of an infected zone should include:

i) the presence or otherwise of the insect vector throughout the year,

ii) the absence of frost conditions necessary to eliminate the vector,

iii) the presence of mountain ranges or areas of arid terrain to act as a natural barrier to the movement of insect vectors.

Within and at the border of the infected zone there must be effective veterinary control of domestic horses and other equines and their transportation. The regulations must be published and rigorously implemented.

No domestic horse or other equine may be moved out of the infected zone except in accordance with the provisions of Article 2.1.11.6.

All vaccinated domestic horses or other equines in the infected zone must be clearly identified with a permanent mark at the time of vaccination.

AHS: restoration to free status

A country or zone of a country may be restored to AHS free status if:

1) the disease has been compulsorily notifiable in the whole country for at least two years;
2) no clinical, serological (in non-vaccinated animals) and/or epidemiological evidence of AHS has been found in the country or zone during the last two years;
3) no equines have been vaccinated against the disease in the country or zone during the last 12 months;
4) no equines have been imported from infected countries or zones except in conformity with the provisions of Article 2.1.11.6.;
5) a system making compulsorily notifiable any mortality in equines has been in force for at least two years; any dead equine has been investigated so as to confirm the absence of AHS;
6) documented evidence that all the above conditions have been fulfilled should be sent to the OIE.

Article 2.1.11.3.

Veterinary Administrations may prohibit importation or transit through their territory, directly or indirectly, from countries or zones of a country considered infected with AHS of:

1) domestic horses and other equines
2) *semen* of domestic horses or other equines.

Article 2.1.11.4.

When importing from AHS *free countries* or *free zones*, *Veterinary Administrations* should require:

<u>for domestic horses</u>

the presentation of an *international animal health certificate* attesting that the animals:

1) showed no clinical signs of AHS on the day of shipment;
2) have not been vaccinated against AHS within two months of export;
3) were kept in an AHS free country or free zone since birth or for at least the past two months.

Article 2.1.11.5.

When importing from AHS *free countries* or *free zones. Veterinary Administrations* should require:

<u>for other equines</u>

the presentation of an *international animal health certificate* attesting that the animals:

1) showed no clinical signs of AHS on the day of shipment;
2) have not been vaccinated against AHS within two months of export;
3) were kept in an AHS free country or free zone since birth or for at least the past two months; and

if the animal originates from a zone or country adjacent to an *infected zone* or a country considered infected with AHS:

4) were kept in a *quarantine station* for 60 days prior to shipment and were subjected to the diagnostic test for AHS with negative results;

5) were protected from insect vectors during quarantine and transportation to the *place of shipment*.

Article 2.1.11.6.

When importing from countries or zones considered infected with AHS, *Veterinary Administrations* should require:

for domestic horses only

the presentation of an *international animal health certificate* attesting that the animals:

1) have been exported only during seasons when the insect vectors are at a low level of activity;

2) showed no clinical signs of AHS on the day of shipment;

3) were kept in a *quarantine station* for a minimum period of 40 days immediately prior to shipment;

4) have been vaccinated against AHS at least two months prior to export and have been clearly identified with a permanent mark; or

5) in non-vaccinated animals, were subjected to the diagnostic test for AHS within ten days prior to shipment with negative results; and

6) were protected from insect vectors during quarantine and transportation to the *place of shipment.*

Article 2.1.11.7.

When importing from AHS *free countries* or *free zones*, *Veterinary Administrations* should require:

for *semen* of domestic horses

the presentation of an *international animal health certificate* attesting that the donor animals:

1) showed no clinical signs of AHS on the day of collection and for the following 40 days;

2) had not been vaccinated against AHS within two months of the date of collection of the semen;

3) were kept in an AHS free country or free zone for at least 40 days prior to collection.

Article 2.1.11.8.

When importing from countries or zones considered infected with AHS, *Veterinary Administrations* should require:

for *semen* of domestic horses

the presentation of an *international animal health certificate* attesting that the donor animals:

1) were kept in a *quarantine station* for at least 40 days prior to collection;

2) were protected from insect vectors during quarantine;

3) showed no clinical signs of AHS on the day of collection and for the following 40 days;

4) have been vaccinated against AHS at least two months prior to the date of collection of the semen; or

5) in non-vaccinated animals, were subjected to the diagnostic test for AHS at least ten days after collection with negative results.

APPENDIX 4 Procedures for surveillance and proof of freedom

The OIE Code (Appendix 3, Articles 2.1.11.2 and 2.1.11.6) gives the conditions for proof/restoration of freedom and for importing from countries or zones considered to be infected.

It is possible that international trading partners vary the conditions relating to surveillance of livestock and possibly vectors before regarding Australia or a zone within Australia as free.

Surveillance

A statistically-valid sample of horses from premises within the RA must be sampled fortnightly until 60 days following the last confirmed or clinical case, or at least 30 days after the first heavy frosts. Thereafter samples may be collected bimonthly for the next twelve months and quarterly for the subsequent twelve months. Programs to collect samples should be established at knackeries in the RA and CA.

In a widespread outbreak where vaccination is used, due to sheer logistics, the amount of sampling may have to be reduced until no more clinical cases are being detected. In this case, the monthly sampling of sentinels may then commence.

Sampling rates have not been determined although a confidence limit of 95% or higher must be assumed. To some extent, it will depend on the livestock density, climate and insect populations in the RA.

Surveillance of vectors may be carried out as described in Appendix 5.

Sentinels

In an isolated outbreak in an area of low epidemic potential, serologically-negative horses could be introduced as sentinel animals to premises where AHS was previously confirmed or to comparable premises in the vicinity. Sentinels must be maintained in a manner that allows free access by insect vectors and must not be stabled during times of maximum vector activity. No vector control systems should be used on premises where sentinels are kept. The horses should be examined regularly for clinical evidence of AHS and bled at monthly intervals for serological testing. Sera should be examined for AHS group antibodies. Reactors should be examined for infecting AHS serotype.

APPENDIX 5 Procedures for vector monitoring and control

In the event of an outbreak of AHS, the main targets for monitoring and control will be *Culicoides* biting midges. However mosquitoes and ticks may also play a role in the spread of the virus.

Monitoring

The most commonly used method of collecting biting midges and mosquitoes is the light trap. CSIRO, the Queensland Department of Primary Industries, and New South Wales Agriculture should have adequate numbers of these traps available. Many local government authorities also use carbon dioxide baited light traps to collect mosquitoes. An essential supplementary collection method for biting midges is a vehicle-mounted trap, sometimes called a truck trap. This is particularly useful in areas or times where evening and night temperatures are low enough to reduce insect activity, ie below 16–18°C.

Larval sampling is considerably more time-consuming than adult sampling, and may not be as reliable an indicator of specific insect prevalence in an area.

Maps of appropriate detail will be required to plot the distribution of traps and potential breeding sites.

The limiting factor in any monitoring program will be the availability of staff with taxonomic expertise to identify the collections.

If collections are to be processed for virus isolation, insects will need to be collected live for immediate processing, or held in suitable storage such as liquid nitrogen. Collections for population analysis should be stored in 70% ethanol. The technology to allow isolation of virus from insects preserved in alcohol is currently being refined.

Collections should aim to give:

- a list of all the potential vectors present;
- the relative abundance of those species; and
- breeding sites of those species.

Control

The main aim of any vector control program must be to break the transmission cycle by rapid reduction in numbers of all insects capable of taking up virus from vertebrate hosts. The main types of insecticide application to control adult insects are:

- ultra-low volume (ULV) application from the ground;
- ULV application from the air;
- thermal fogs or mists from the ground; and
- systemic or topical treatment of livestock.

In the event of a decision to treat a broad-scale area, ground-based ULV application would be the most likely method. The insecticide to be used will be determined after consultation with appropriate environmental authorities (from whom a permit may be required), bearing in mind what products are rapidly available in sufficient quantity. Treatment will depend on prevailing weather, the terrain and its influence on machinery access, and identification of significant breeding sites of potential vectors. Ground-based spraying equipment would normally be operated from the back of a truck, utility or four-

wheeled farm bike, but back-pack machinery may be needed to treat breeding sites not accessible by vehicle.

Aerial applications of insecticide may be necessary because of access difficulties and/or the need to cover large areas rapidly. In this case, organisations such as the Australian Plague Locust Commission should be consulted. Costs are substantially higher than for ground-based application, and no assessment has been made of the effectiveness of this type of treatment against potential vectors associated with livestock. All legal requirements must be complied with.

If *C. brevitarsis* is a major target of a control program, the use of a systemic insecticide in cattle in the area should be considered. For example, a subcutaneous injection of a formulation of ivermectin will produce 99% mortality in *C .brevitarsis* feeding on treated cattle for up to 10 days after treatment. The larval stages in dung of treated animals will also be controlled for up to 4 weeks. A pour-on formulation is also available. Horses can be treated with ivermectin, but the effect of the treatment on biting flies has not been tested. The subcutaneously-delivered formulation is not repellent. Withholding periods must be observed.

Control measures will need to continue for as long as the virus continues to be found in animals at the outbreak site. In the case of a dung-breeding midge such as *C. brevitarsis,* larvae and pupae will be protected from routine control measures, other than some systemic treatments, and newly emerged adults will continue to appear for some time after initial treatments. This may also be the case where breeding sites of other midge species cannot be found or treated.

Appropriate protective equipment must be provided and its use made compulsory for staff involved in insecticide applications. These staff must follow recommended safety guidelines. Adequate first aid measures and material safety data sheets must be on hand. When systemic or topical insecticides are used on livestock, the relevant withholding periods must be observed.

APPENDIX 6 Procedures for vaccination

AHS vaccine is not currently manufactured in Australia. In the event of an outbreak of AHS, vaccine would be required quickly. and, as there would be a considerable time-lag before vaccine was produced in Australia, so it would have to be imported. Only *inactivated* AHS vaccines should be considered for use in Australia.

To enable an inactivated vaccine to be imported at short notice, it is essential for there to be approval of vaccines and manufacturing processes before an outbreak of AHS. This will require good cooperation between government authorities, vaccine manufacturers and importers.

For rapid availability of a vaccine, the following will be required:

- prior assessment of an inactivated vaccine;
- stocks of the particular AHS serotype vaccine; and
- sufficient supplies of the vaccine to enable an effective vaccinated buffer zone to be established.

Delays in the availability of an appropriate vaccine may allow the disease to spread, requiring vaccination of a larger buffer zone and consequently a larger vaccine supply.

Rhone Merieux[1] produce an inactivated vaccine approved by the European Union, which has been used in Spain to control AHS spread from Morocco. This vaccine reflects the AHS serotype occurring in northwestern Africa.

Vaccine use and distribution in Australia would be controlled by government to limit spread of the disease. Any general use of an AHS vaccine would only be permitted when there was adequate vaccine supply and the disease was spreading beyond control.

If a vaccination program is initiated, it will be essential to identify and maintain records of all vaccinated horses. Most horses in Australia do not have an adequate identification documentation process. The horse passport system provides the basis for establishing an adequate system. The horse passport system would provide the necessary assurances of vaccination to allow the movement of horses that have certification of their vaccination status.

[1]Rhone Merieux, 254 rue Marcel Merieux, Lyin Cedex 07, Lyon 69342, France

GLOSSARY

Animal by-products	Products of animal origin destined for industrial use, eg raw hides and skins, fur, wool, hair, feathers, hooves, bones, fertiliser.
Animal products	Meat products and products of animal origin (eg eggs, milk) for human consumption or for use in animal feeding.
Arbovirus	*Ar*thropod-*bo*rn-*virus*es — transmitted by ticks, insects, etc. The virus replicates in an arthropod and is transmitted by bite to a vertebrate host in which it also replicates.
AUSVETPLAN	A series of documents that describe the Australian response to exotic animal diseases, linking policy, strategies, implementation, coordination and counter-disaster plans.
Consultative Committee on Exotic Animal Diseases	A committee of State/Territory CVOs, AAHL and CSIRO, chaired by the CVO of Australia (Cwlth DPIE), to consult in emergencies due to the introduction of an exotic disease of livestock, or serious epizootics of Australian origin.
Control area	A declared area in which defined conditions apply to the movement into, out of, and within, of specified animals or things. Conditions applying in a control area are of lesser intensity than those in a restricted area (*see* Appendix 1).
Complement fixation test	Assay for complement by its ability to cause lysis of red blood cells. Fixation of complement by combination of antibody and antigen reduces its ability to lyse red blood cells.
Cyanosis (adj. cyanotic)	Blueness of the skin and/or mucous membranes due to insufficient oxygenation of the blood.
Dangerous contact animal	An animal showing no clinical signs of disease but which, by reason of its probable exposure to disease, will be subjected to disease control measures.
Dangerous contact premises	Premises containing a dangerous contact animal(s) (*see* Appendix 1).
Declared area	A defined tract of land for the time being subject to disease control restrictions under exotic disease legislation. Types of declared areas include *restricted area; control area; infected premises;* and *dangerous contact premises.*
Decontamination	Includes all stages of cleaning and disinfection.
Ecchymotic haemorrhage	Small round spots or purplish discolouration caused by bleeding or bruising in the skin or mucous membrane.
ELISA	Enzyme-linked immunosorbent assay — a serological test designed to detect and measure the presence of antibody or antigen in a sample. The test uses an enzyme reaction with a substrate to produce a colour change when antigen–antibody binding occurs.
Endocardium	Endothelial lining of heart cavities.
Equidae (equines)	Family of herbivorous mammals of the order Perissodactyl with grinding teeth, including horses, asses, donkeys and zebras.

Fomites	Inanimate objects (eg boots, clothing, equipment, vehicles, crates, packagings) that can carry the exotic agent and spread the disease through mechanical transmission.
Haemagglutination	Agglutination of red blood cells by a specific antibody or other substance.
Hydropericardium	Fluid exudate in the pericardial sac.
Hydrothorax	Fluid exudate in the pleural cavity.
Hyperaemia	Congestion or excess of blood in an organ or tissue.
Immunodiffusion	A serological test to identify antigens or antibodies by precipitation of antibody–antigen complex after diffusion through agar gel.
Incubation period	The period that elapses between the introduction of the pathogen into the animal and the occurrence of the first clinical signs of the disease.
Index case	The first or original case to be diagnosed in a disease outbreak (on the index property).
Infected premises	*see* Appendix 1.
Interferons	Proteins with antiviral activity released from cells in response to virus infection or other immunological stimuli.
Local disease control centre	An emergency operations centre responsible for the command and control of field operations in a defined area.
Lymphokine	Generic name for proteins (other than antibodies or surface receptors) released by lymphocytes stimulated by antigens or other means, that act on other cells involved in the immune response.
Movement controls	Restrictions placed on movement of animals, people and things to prevent the spread of disease.
Pericardium	Double-layered membranous sac enveloping the heart. (Epicardium — inner of the 2 layers, attached to the heart.)
Petechial haemorrhages	Tiny, flat, red or purple spots in the skin or mucous membrane caused by bleeding from small blood vessels.
Premises	A defined area or structure, which may include part or all of a farm, enterprise or other private or public land, building or property.
Prevalence	The number of cases of a specific disease (or infection or positive antibody titre) occurring in a given population at a particular time (expressed as the proportion of sampled animals with the condition of interest at a given time).
Quarantine	Legal restrictions imposed on a place, animal, vehicle or other things limiting movement.
Restricted area	A declared area in which defined rigorous conditions apply to the movement into, out of, and within, of specified animals, person or things (*see* Appendix 1).
Sentinel animals	Animals of known health status monitored for the purpose of detecting the presence of a specific exotic disease agent.
Serotype	A subgroup of a genus of microorganisms identifiable by the antigens carried by the members.

Serum neutralisation	A type of serological test designed to detect and measure the presence of antibody in a sample. The test is based on the ability of an antibody to neutralise the biological activity of an antigen.
Stamping out	Eradication procedures based on quarantine and slaughter of all infected animals and animals exposed to infection.
State/Territory disease control headquarters	The emergency operations centre that directs the disease control operations to be undertaken in the State/Territory.
Surveillance	A systematic program of inspection and examination of animals or things to determine the presence or absence of AHS.
Susceptible species	Animals that can be infected with the disease (for AHS — Equidae [ie horses, zebras, mules, donkeys] and dogs; elephants and camels have also been shown to have antibodies indicating the possibility of subclinical/inapparent infection; see Section 1.2).
Suspect animal	An animal that may have been exposed to an exotic disease such that its quarantine and intensive surveillance is warranted; OR an animal not known to have been exposed to a disease agent but showing clinical signs requiring differential diagnosis.
Suspect premises	Premises containing suspect animals (*see* Appendix 1).
$TCID_{50}$	Tissue culture infectious dose — a measure of virus concentration or dose. Serial dilutions of virus are added to susceptible cells in culture. The dilution of virus at which half of the cultures are infected is called the $TCID_{50}$.
Tracing	The process of locating animals, persons or things that may be implicated in the spread of disease, so that appropriate action can be taken.
Vaccine	
– attenuated	A vaccine prepared from infective or 'live' microbes that have lost their virulence but have retained their ability to induce protective immunity.
– inactivated	A vaccine prepared from a virus that has been inactivated ('killed') by chemical or physical treatment.
– recombinant	A vaccine produced from virus that has been genetically engineered to contain only selected genes, including those causing the immunogenic effect (subunit and construct vaccines).
Vector	A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. A *biological* vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. A *mechanical* vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.
Viraemia	The presence of viruses in the blood.
Zoning	Dividing a country into defined infected and disease free areas. A high level of movement control between zones will apply.
Zoonosis	Disease transmissible from animals to people.

Abbreviations

AAHL	CSIRO Australian Animal Health Laboratory, Geelong
AHS	African horse sickness
AUSVETPLAN	Australian Veterinary Emergency Plan
CA	Control area
CCEAD	Consultative Committee on Exotic Animal Diseases
CSIRO	Commonwealth Scientific and Industrial Research Organisation
CF	Complement fixing [antibody]
CVO	Chief veterinary officer
DCP	Dangerous contact premises
ECE	Embryonating chick eggs
EDTA	Ethylene diamine tetra-acetic acid (anticoagulant for whole blood)
ELISA	Enzyme-linked immunosorbent assay
EM	Electron microscopy
GDP	Gross domestic product
IP	Infected premises
OIE	World Organisation for Animal Health [Office International des Epizooties]
RA	Restricted area
SP	Suspect premises
TCID	Tissue culture infective dose
ULV	Ultra-low volume

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Training resources

[See the **Summary Document** for a full list of training resources.]

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