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

Primary Industries Ministerial Council
This disease strategy forms part of:

AUSVETPLAN Edition 3

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

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IMPORTANT NOTE: Important regulatory information is contained in the OIE Terrestrial Animal Health Code for surra, which is updated annually and is available on the internet at the OIE website: http://www.oie.int/eng/normes/en_mcode.htm. Further details are given in Appendix 3 of this manual.

DISEASE WATCH HOTLINE

1800 675 888

The Disease Watch Hotline is a toll-free telephone number that connects callers to the relevant state or territory officer to report concerns about any potential emergency disease situation. Anyone suspecting an emergency disease outbreak should use this number to get immediate advice and assistance.
Preface

This disease strategy for the control and eradication of surra is an integral part of the Australian Veterinary Emergency Plan, or AUSVETPLAN (Edition 3). AUSVETPLAN structures and functions are described in the AUSVETPLAN Summary Document.

This strategy sets out the disease control principles that have been approved by the Primary Industries Ministerial Council (PIMC) out-of-session on 15 June 2006 for use in an animal health emergency caused by the occurrence of surra in Australia.

Surra in horses is designated by the Office International des Epizooties (OIE) as a notifiable disease. Surra in other species is not listed by the OIE. However, the principles contained in this document for the diagnosis and management of an outbreak of surra conform with the OIE International Animal Health Code (see Appendix 3).

In Australia, surra is included as a Category 4 emergency animal disease in the Government and Livestock Industry Cost Sharing Deed In Respect of Emergency Animal Disease Responses (EAD Response Agreement).\(^1\) Category 4 diseases are diseases that could be classified as being mainly production loss diseases. While there may be international trade losses and local market disruptions, these would not be of a magnitude that would be expected to significantly affect the national economy. The main beneficiaries of a successful emergency response to an outbreak of such a disease would be the affected livestock industry(s). For this category, the costs will be shared 20% by governments and 80% by the relevant industries (refer to the Deed of Agreement for details).

Detailed instructions for the field implementation of AUSVETPLAN are contained in the disease strategies, operational procedures manuals, management manuals and wild animal manual. Industry-specific information is given in the relevant enterprise manuals. The full list of AUSVETPLAN manuals that may need to be accessed in an emergency is:

**Disease strategies**
- Individual strategy for each disease

**Operational procedures manuals**
- Decontamination
- Destruction of animals
- Disposal
- Public relations
- Valuation and compensation

**Enterprise manuals**
- Animal quarantine stations
- Artificial breeding centres
- Aviaries and pet shops
- Feedlots
- Meat processing
- Poultry industry
- Saleyards and transport
- Veterinary practices
- Zoos

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Management manuals
- Control centres management (Volumes 1 and 2)
- Animal Health Emergency Information System
- Laboratory preparedness

Wild animal manual
- Wild animal response strategy

Summary document

In addition, *Exotic Diseases of Animals: A Field Guide for Australian Veterinarians* by WA Geering, AJ Forman and MJ Nunn, Australian Government Publishing Service, Canberra, 1995 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 manual is a new manual in AUSVETPLAN Edition 3.0. It was prepared by a writing group with representatives from the Australian national and state/territory governments:
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- Ian Douglas, Department of Primary Industries, Queensland;
- Patricia Ellis, Department of Natural Resources and Environment, Victoria; and
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Scientific editing was by Dr Janet Salisbury of Biotext, Canberra.

The manual has been reviewed and approved by:

**Government**
- Commonwealth of Australia
- State of New South Wales
- State of Queensland
- State of South Australia
- State of Tasmania
- State of Victoria
- State of Western Australia
- Northern Territory
- Australian Capital Territory

**Industry**
- Australian Pork Limited
- Australian Racing Board
- Australian Harness Racing Council Inc.
- Australian Horse Industry Council

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1 Nature of the disease

Surra is a haemoparasitic disease transmitted by biting flies. It affects a wide range of host species, causing fever, weight loss, anaemia and a range of other symptoms leading to death in a large proportion of naive animals.

1.1 Aetiology

Surra is caused by Trypanosoma evansi, which is one of the salivarian group of trypanosomes. Trypanosomes are parasites occurring in the blood of a large number of wild and domestic hosts. T. evansi is a small, actively motile trypanosome, generally about 23–25 µm long. It is morphologically indistinguishable from the slender form of T. brucei, and it is thought by some to be a variant of this organism. It multiplies by binary fission. T. evansi is found in the blood during the acute stages of the disease, but disappears rapidly after the death of the host (Hoare 1972).

There are antigenic differences between isolates of T. evansi because the trypanosome shows variation in its antigen coat. There is limited, equivocal information concerning the existence of strains of T. evansi of different pathogenicity (Hoare 1972, Queiroz et al 2000). Some strains are referred to colloquially as ‘highly pathogenic’ but this may be a result of host vector factors, such as stock and insect densities and the susceptibility of host species (Hoare 1972).

1.2 Susceptible species

Surra has a wide host spectrum. The disease is most severe in horses, donkeys, mules, camels, dogs and cats. Disease also occurs in mild or subclinical forms in cattle, alpacas, llamas, and buffalo. Occasional mild, chronic or subclinical disease occurs in sheep, goats, pigs, capybaras and elephants (Hoare 1972). Deer may differ in their susceptibility to infection with T. evansi. T. evansi is highly pathogenic in Muntiacus muntjak (barking deer) (Hoare 1972) and deer in the Philippines (De Jesus 1963), and it induces a chronic disease in Axis axis (axis deer) (Hoare 1972) and Cervus timorensis (rusa deer) (Reid et al 1999).

Mortality rates are low in cattle and buffalo raised in endemic areas, but movement of animals from Australia (a non-endemic area) to Indonesia (an endemic area) has resulted in high mortality rates (Payne et al 1988, 1991a).

Two species of wallaby commonly found in northern Australia and Papua New Guinea (PNG) — Macropus agilus (agile wallaby) and Thylogale stigmatica (pademelon) — are known to be susceptible to experimental infection (Reid et al 2001a), but the susceptibility of other species of Australian native fauna to T. evansi is unknown. However, dingoes and feral pigs should be considered as potential hosts.

Human infection, despite hundreds of millions of exposures to the infective agent, is not an issue. There is anecdotal evidence of natural infection occurring in
humans. Suspected cases of human trypanosomiasis have been reported in one man from Sri Lanka (ProMED-mail 1999) and in two individuals from India (ProMED-mail 2004, ProMED-mail 2005). Confirmation that the infecting trypanosome was *T. evansi* has only been made in one of the Indian cases (WHO 2005) despite long-term exposure in endemic areas.

### 1.3 World distribution and occurrence in Australia

Surra is found over a wide range of climates, but is more common in the tropics. It occurs in a broad band encompassing northern and central Africa (although South Africa is free), the Middle East, Iran, Pakistan, India, Nepal, Southeast Asia, China, and Central and South America (Hoare 1972). In Southeast Asia, it has been reported from Indonesia, the Philippines, Thailand, Laos and Malaysia, but is probably present throughout the region (Luckins 1988, Reid 2002).

Surra is found in most parts of Indonesia, and there is serological evidence of its presence in West Papua (formerly Irian Jaya). Positive serology for *T. evansi* has also been detected in cattle and small ruminants on the border between PNG and Indonesia, but its presence in PNG has not been confirmed (Reid and Copeman 2000).

Despite buffalo, cattle, banteng and camels being imported from India and Indonesia in the early part of the last century, surra did not become established in Australia. The disease was diagnosed in nine camels imported from India into Port Hedland in 1907, but destruction of the consignment prevented the spread of the disease.

### 1.4 Diagnostic criteria

#### 1.4.1 Clinical signs

The severity and course of clinical signs of surra vary according to the virulence of the strain of *T. evansi*, the host species and other stress factors on the animal. Acute and chronic syndromes are seen but may overlap. Immune suppression following surra can lead to a higher susceptibility to intercurrent diseases, which may complicate the clinical picture. Infection can terminate in death, lead to complete recovery, or persist in reservoir hosts. Introduction of the parasite to new areas is characterised by a high prevalence of infection, with mortality reaching 30–100% (Payne et al 1990).

The acute form of the disease may last for up to three months and is characterised by irregular fever, progressive weight loss in the presence of continued good appetite, anaemia, recurrent keratoconjunctivitis and urticarial plaques on the neck and flank, and dependent oedema of the thorax, abdomen, genitalia and legs (Hoare 1972). Increases in body temperature correspond with peaks of parasitaemia.

The clinical signs in chronic cases are less distinctive. Production deficits, lethargy, rough hair coat, progressive emaciation, anaemia, weakness and recurrent fever may be observed. Terminal central nervous system involvement is common.
Horses, donkeys, mules, dogs and cats

In the absence of adequate treatment, surra is often acute and rapidly fatal in horses, donkeys, mules, dogs and cats (Hoare 1972). The course of the acute form of the disease may be as short as 2–3 weeks or as long as four months. Some breeds may also be more tolerant to the trypanosomes than others. There is evidence that donkeys have an extended form of infection and become reservoir hosts (Hoare 1972). Local breeds of horse in Indonesia thrive in endemic areas where introduced thoroughbreds rapidly succumb to infection.

In addition to the typical signs of the acute syndrome described above, petechial (pinpoint) haemorrhages in visible mucous membranes, a wide-based stance, loss of balance and hindlimb proprioceptive deficits (impaired awareness of joint position) may be seen in affected horses (Monzon et al 1990). In South America, the disease in horses is known as mal de caderas (‘swaying of the hips’) and is characterised by gradual development of central nervous system involvement with weakness, hyperexcitability and incoordination, usually progressing to terminal weakness and paralysis (Hoare 1972).

In dogs, there is marked oedema particularly of the scrotum, ears and neck (Husein et al 1994). Progressive emaciation (despite good appetite), fluctuating fever, enlarged lymph nodes, anaemia and ataxia have also been associated with infection (Husein et al 1994). Corneal opacity may be present. Dogs and cats occasionally exhibit nervous signs suggestive of rabies.

Camelids

In herds of dromedary and Bactrian camels, individual variation in response to infection occurs. Acute syndromes of 2–3 months duration can occur, but the more common chronic form may last up to three years and be associated with wasting, abortion, premature birth, inability to produce milk, and weak newborn calves (Diall et al 1994). If untreated, the disease produces 90% mortality.

Infections in South American camelids such as alpacas and llamas are usually subclinical, although clinical disease has been reported in llamas.

Cattle, buffalo, goats, sheep and deer

In these species, disease is usually less severe. Response to infection can be inapparent, mild or chronic. Appetite is often not affected. Abortion and a sudden drop in milk yield have been reported in buffalo. Death may occur up to six months after the onset of signs, but many animals recover and become reservoir hosts.

Mortality rates are low in cattle and buffalo in endemic areas. However, movement of previously unexposed Australian buffalo into an endemic area in Indonesia has resulted in high mortality in the introduced animals (Payne et al 1991a). Clinical disease has also been seen in previously unexposed cattle.

Emaciation, orchitis (inflammation of the testes) and spermatozoal abnormalities have been reported following experimental infection of goats.
Pigs
Occasional outbreaks of clinical disease have been recorded in pigs. Investigations of livestock losses in a piggery in Indonesia and death and abortion in a piggery in Thailand showed *T. evansi* to be the cause of disease (Kraneveld and Mansjoer 1947, Sirivan et al 1989). Experimental infection of domestic pigs revealed that, after a period of 24–30 days, trypanosomes were rarely detected in blood smears but the blood remained infective to rats (Reid et al 1999).

Rodents and lagomorphs
Rats, mice, guinea-pigs and rabbits are susceptible to infection in the laboratory. The significance of wild populations in the epidemiology of the disease is unknown.

Wildlife and zoo animals
Infection has been reported in a wide variety of wild animals. They include captive tigers and other large felines from India and Sumatra; wolves from Kazakhstan; wild dogs (*Canis azarac*) from South America; foxes (*Vulpes vulpes, V. bengalensis*) from Asia; a Sumatran orang-utan; deer, tapirs, pikas and capybaras (*Hydrochoerus hydrochaeris*); and vampire bats (*Desmodus rotundus*) from Central and South America.

Capybaras are considered to be important reservoirs of infection in South America, and in Indonesia wild deer and monkeys have been suggested as important reservoir hosts, but the significance of other species in the wild is unknown.

Experimental infection of agile wallabies and pademelons, found in northern Australia and PNG, has demonstrated a high susceptibility to infection. The animals maintained high parasitaemia until death occurred within 30–60 days (Reid et al 2001b). The susceptibility of other species of Australian native fauna to *T. evansi* is unknown.

1.4.2 Pathology

Gross lesions
The general pathology of infection with *T. evansi* reflects an active lymphoreticular response by the immune system throughout the body. Gross pathological changes seen at postmortem in animals infected with *T. evansi* vary both between species and between individuals of the same species. In buffalo experimentally infected with *T. evansi*, gross changes included emaciation, serous atrophy of fat, hydropericardium, splenomegaly, lymphadenopathy and active haemopoiesis in the bone marrow (Damayanti et al 1994). The bone marrow is dark red and hyperplastic in acute cases but becomes atrophied, gelatinous and yellowish in chronic cases (Damayanti et al 1994). Gross changes reported in other species include corneal opacity and petechiation of the heart in dogs, congestion of the abomasum and small intestine in camels (Raisinghani et al 1980) and vulval swelling in rabbits (Uche and Jones 1992). Gastric ulceration was reported in horses infected with *T. brucei* (McCully and Neitz 1971) but not in horses infected with *T. evansi*.
Microscopic lesions (histopathology)

The histopathological lesions of surra, especially during early stages of infection, are diagnostic for the disease. Important tissues to examine are choroid plexus, cardiac muscle including the heart valves, lung, spleen and bone marrow. At necropsy, the brain should be sectioned transversely before being placed in fixative to facilitate examination of the choroid plexus and ventricles.

Histological lesions of surra are characterised by hyperplastic changes in the lymph nodes and spleen and lymphocytic infiltration in lungs, kidneys, myocardium, meninges and choroid plexus (Losos and Ikede 1972, Damayanti et al 1994). Trypanosomes may be seen (in sections stained with haematoxylin and eosin) in the interstitium in association with these mononuclear infiltrates, and in blood vessels, appearing as aggregates of small coccoid bodies (nuclear DNA). Such lesions may occur in any organ with antigen-processing cells but are most consistently found in the choroid plexus (interstitial choroiditis), cardiac muscle (focal nonsuppurative myositis) and lung (diffuse alveolar pneumonitis).

Clinical pathology

The main clinical feature of surra is anaemia. Its pathogenesis is associated with activated macrophages in the spleen, liver, lungs, lymph nodes and bone marrow, which remove red blood cells from the circulation (Murray and Dexter 1988). In addition, macrophages in the bone marrow phagocytose precursor cells, especially those of red cells, platelets, neutrophils and eosinophils (Murray and Dexter 1988). The anaemia is initially responsive before becoming nonresponsive. Peripheral blood has decreased packed-cell volume and red blood cell count, increased lymphocytes, decreased neutrophils and eosinophils, and an initial drop in monocytes followed by normalisation in numbers.

The immune response to infection also results in progressive changes in serum protein levels and particularly increased IgM levels. Increases in the serum enzymes sorbitol dehydrogenase and glutamate pyruvate transaminase may also occur.

1.4.3 Laboratory tests

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 about the transport of the specimens to Geelong.

Specimens required

Tissues required for the diagnosis of surra include:

- whole unclotted blood (with heparin or EDTA)
- dried blood collected using Whatman FTA® cards
- serum
- tissues fixed in formalin:
  - brain (transversely sectioned — see above)
cardiac muscle
- heart valve
- lung
- spleen
- bone marrow.

Transport of specimens
Whole blood and serum samples should be kept cool and in a lightproof container (eg 4°C) but not frozen during transport. Whatman FTA cards can be stored at room temperature in a plastic bag containing a desiccant. Specimens should be shipped in leakproof containers to the AAHL at Geelong. The laboratory should be notified in advance that the samples are being dispatched.

Laboratory diagnosis
Parasitological, serological and molecular tests are available, and augment clinical signs and history in the diagnosis of surra.

Parasitological tests
Parasitological tests cannot reliably detect low numbers of trypanosomes in blood. Consequently, they are unable to confirm infection between the periodic peaks of parasitaemia that characterise the chronic phase of surra.

In animals with parasitaemia, buffy coat (white blood cell fraction) can be used in either rodent (mouse) inoculation (RI) or mini-anion exchange centrifugation tests (MAECT), which are able to detect one trypanosome in 2 mL of blood (Reid et al 2001a). These tests are recommended for diagnostic use in animals during the first few months after infection, when the probability of parasitaemia is highest. Further information on these tests is presented in Table 1 and Appendix 6.

The haematocrit centrifuge technique (HCT) is a simple, rapid test that can be performed in the field. Less sensitive than RI tests, it is able to detect approximately 100 T. evansi per ml of blood, which can be increased if multiple tubes are examined.

Serological tests
Although serological tests are more sensitive than parasitological tests, they are incompletely standardised for Australian conditions, making interpretation of test results equivocal. There is currently no serological test that can be recommended for use in individual Australian animals that will detect, with useful accuracy, infection or freedom from recent infection with T. evansi. Moreover, there is no useful information on the use of such tests in many species of interest, including dogs, pigs, deer, sheep, goats and macropods.

Although not recommended for the detection of recent infections, the card agglutination test (CATT) has some value in identifying longstanding infections, provided that results are interpreted on a herd rather than individual animal basis. After standardisation, its specificity has been determined at 99% in northern Australian cattle (Reid and Copeman 2003). However, the test is not very sensitive for identifying recent infections, as the antigen it is designed to detect may not
appear until many months after initial infection, and in some animals may not appear at all.

The antibody-ELISA (enzyme-linked immunosorbent assay) has a better sensitivity than the CATT, reportedly detecting 89% of infected buffalo, but with a slightly lower specificity (92% in cattle and buffalo) (Davison et al 1999). However, antibody-ELISA test results should be interpreted with caution, particularly on an individual animal basis, and where possible should be augmented with parasitological tests. An antibody-ELISA using a semipurified antigen was shown to have a sensitivity and specificity of 81% and 99.6% respectively when used to test serum from Indonesian cattle infected with *T. evansi* and uninfected cattle from Townsville (Reid and Copeman 2003). This test is being developed in a kit format.

**Molecular tests**

PCR has been used to detect *T. evansi* in the blood of infected animals and in the blood meal of tabanids (Wuyts et al 1994, Ijaz et al 1998, Holland et al 2001). This work has been extended as part of the ACIAR-funded project AS1/2000/009, and a PCR-based test using primers designed by Wuyts et al (1994) and able to detect 1 trypanosome per mL of blood has been optimised for use with fresh or frozen tissue samples, frozen blood and blood dried onto Whatman FTA cards. Further work is required before this test is fully validated for use in Australia.

### Table 1 Diagnostic tests for surra currently available in Australia

<table>
<thead>
<tr>
<th>Test</th>
<th>Specimen required</th>
<th>Test detects</th>
<th>Time taken to obtain result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rodent inoculation</td>
<td>EDTA blood&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Live <em>T. evansi</em></td>
<td>7–30 days</td>
</tr>
<tr>
<td>Mini-anion exchange centrifugation test</td>
<td>EDTA blood&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Live <em>T. evansi</em></td>
<td>1 hour</td>
</tr>
<tr>
<td>Haematocrit centrifugation technique</td>
<td>EDTA blood</td>
<td>Live <em>T. evansi</em></td>
<td>30 minutes</td>
</tr>
<tr>
<td>CATT</td>
<td>Serum</td>
<td>IgM and IgG</td>
<td>15 minutes</td>
</tr>
<tr>
<td>Antibody ELISA</td>
<td>Serum</td>
<td>IgG</td>
<td>4 hours</td>
</tr>
<tr>
<td>Polymerase chain reaction</td>
<td>EDTA blood, dried blood, fresh or frozen tissues</td>
<td><em>T. evansi</em> DNA</td>
<td>4 hours</td>
</tr>
</tbody>
</table>

CATT = card agglutination test; ELISA = enzyme-linked immunosorbent assay; IgM/IgG = immunoglobulins

<sup>a</sup> After collection, blood should be stored at 4°C in a lightproof container and processed within 24 hours.

The available information on sensitivity and specificity of tests for *T. evansi* is presented in Appendix 6.

Source: Information provided by Simon Reid, James Cook University, December 1998

### 1.4.4 Differential diagnosis

The following diseases should be considered in a differential diagnosis of surra:

- African horse sickness
- canine babesiosis
- chronic parasitism
- equine babesiosis
- equine infectious anaemia
- equine viral arteritis
- haemobartonella infection

*Surra* 13
• purpura haemorrhagica
• *Trypanosoma theileri* (cattle, deer, antelope)
• haemorrhagic septicaemia (cattle)
• malignant catarrhal fever (cattle)
• rabies (dogs).

*Trypanosoma theileri* is a large trypanosome up to 120 $\mu$m long, but its length varies according to the strain of the parasite. It occurs in Australian cattle but has a narrower host range than *T. evansi*, does not infect rodents and is usually nonpathogenic (Bose et al 1987). In blood films and in the HCT, it can be differentiated from *T. evansi*, which is smaller and more motile.

### 1.4.5 Treatment of infected animals

A range of drugs have been recommended for the treatment of surra, but only one — the arsenical drug melarsomine dihydrochloride (Cymelarsan™, Merial) — has been shown to have reasonable efficacy in different host species (Payne et al 1994a). Melarsomine is the only commercially available compound with high efficacy against *T. evansi*. It is currently registered by the Australian Pesticides and Veterinary Medicines Authority only for use in the treatment of canine heartworm in Australia under the trademark Immiticide™. Melarsomine is rapidly absorbed following intramuscular (IM) injection and is cleared from the circulation within six hours. It is also said to cross the blood–brain barrier and, therefore, may be effective in preventing relapses often seen after treatment with other trypanocides (Raynaud et al 1989).

Two factors affect the efficacy of chemotherapy with melarsomine. The first is variation in susceptibility of different isolates, with some isolates requiring up to four times the dose required by others to kill the parasites (Zhang et al 1991). The second is whether the infection is acute or chronic; efficacy of treatment may be reduced in chronically infected animals. Studies in rabbits have demonstrated that relapse of parasitaemia occurs when treatment is given 30 days after infection with *T. evansi* but not in animals treated 15 days after infection (Biswas and Hunter 1993), and treatment of horses experimentally infected with *T. evansi* with 1 mg/kg Cymelarsan did not prevent fatal relapses occurring in one study (Wernery et al 2001).

Melarsomine is well tolerated by domestic animals (although it causes significant pain and inflammation in dogs) and is the only available drug safe to use in horses by IM injection (Wernery et al 2001). The recommended dose of melarsomine (Cymelarsan, Merial) in camels is 0.25 mg/kg by IM injection, and a dose of 0.5–0.75 mg/kg IM was shown to be effective in cattle in Indonesia (Payne et al 1994ab). The manufacturer recommends that horses be treated with 0.25 mg/kg by IM injection in South America, but the experiences of Wernery et al (2001) suggest that this dose rate is insufficient to effect a cure and that careful monitoring is required after treatment to confirm elimination of the parasite.
1.5 Resistance and immunity

1.5.1 Innate and passive immunity

Surra is most severe in horses, donkeys, mules, camels, dogs and cats (Hoare 1972). Mortality rates may be high in these species (see Section 1.4.1). However, in Lombok (Indonesia), horses positive by HCT and others with high antibody titres have been observed not to have clinical signs and to be in good condition with a normal packed cell volume (Utami 1996). Trypanotolerance to African animal trypanosomes is recognised in some breeds of domesticated animals. The mechanism is not completely understood, though the trait is heritable (Authié 1994).

Mortality rates are low in cattle and buffalo raised in endemic areas, but movement of animals from Australia (a non-endemic area) to Indonesia (an endemic area) has resulted in high mortality rates (Payne et al 1988, 1991a). Infection has not been reported in birds or reptiles.

1.5.2 Active immunity

Trypanosomes have multiple genes that code for variable surface-coat glycoproteins (VSGs). The number of different antigenic types of VSGs is unknown for T. evansi but there are at least 15 (Jones and McKinnell 1985). Antigenic variation is observed in the host as a fluctuating parasitaemia.

As the trypanosome multiplies in the host, new antigenic variants arise as switching of genes controlling expression of the VSG occurs, giving rise to trypanosomes with a new VSG coat (also known as a variable antigenic type, or VAT) (Van Meirvenne et al 1975, Seed et al 1984). This gene switching produces a mixed population of VATs, with a major VAT and several minor VATs present in the host at the same time. As the major VAT population exceeds the host’s immune-recognition threshold, an effective immune response develops and clears those trypanosomes from the blood, allowing the next major VAT to multiply and thus the next wave of parasitaemia to occur (Seed et al 1984).

Animals infected with trypanosomes show immunosuppression, and may succumb to secondary infections (especially early in the course of infection) and fail to respond to vaccines. However, claims for immunosuppression in trypanosomal infections are equivocal, and several authors report conflicting results (Stephen 1986).

Animals treated in the early stages of an initial infection produce only a transient antibody titre. In chronically infected animals, on the other hand, antibodies have been detected for at least four months after use of a trypanocidal drug (Nantulya 1990). Seroconversion to T. evansi generally occurs within two to three weeks after infection (Luckins et al 1978).

1.5.3 Vaccination

The existence of at least 15 VATs in T. evansi has, to date, confounded efforts to produce a vaccine.
1.6 Epidemiology

1.6.1 Incubation period

The period between initial infection and the onset of clinical signs is extremely variable, but generally ranges between 5 and 60 days — although longer periods (such as 3 months) have been recorded. The interval between infection and the demonstration of parasites in the blood is usually less than 14 days.

Factors that affect the incubation period include the initial infective dose (equivalent to the number of infective insect bites), strain of parasite and stress. In nonclinical cases, the sensitivity of the method used to detect parasitaemia influences the time taken between infection and detection of the parasite.

Although laboratory infections using very large infective doses can produce infective animals after a few days, under field conditions (in which natural infections from biting insects result in much smaller infective doses) it is unlikely that animals would become infective within one week.

1.6.2 Persistence of agent

General properties/environment

*T. evansi* is a fragile organism that does not survive for long in the environment or after the death of the host (Geering et al 1995). It could persist for short periods on equipment contaminated with fresh blood, but would not be expected to survive once the blood dried. *T. evansi* is unlikely to survive outside a live host for more than one day at ambient temperatures in blood or carcases, and is likely to survive only for hours on animal handling equipment and surfaces at animal handling facilities. Exposure to direct sunlight for 30 minutes is lethal (Holland et al 2001).

Live animals

The trypanosomes persist in untreated animals for indeterminate periods, during which time multiple peaks of parasitaemia occur, coinciding with febrile periods. There is little information on the duration of long-term carrier states.

Semen and embryos

*Trypanosoma equiperdum* is found in the semen of horses, and Brun et al (1998) and Wang (1988) reported that *T. evansi* could be transmitted directly through coitus. *T. evansi* is routinely cryopreserved in laboratories, and is therefore likely to survive in frozen semen (Holland et al 2001).

The survival of the trypanosome on washed, trypsinised embryos is highly unlikely.

Animal products

All *T. evansi* organisms were still viable at four hours postmortem in inoculated, parasitaemic rats stored at room temperature; less than 1% were viable at 10 hours (although mice were infected); and by 12 hours all parasites had degenerated (Sarmah 1998). *T. evansi* is unlikely to survive in infected meat for more than 8 hours at ambient temperatures (Kraneveld and Djaenoedin 1939). There are no
data on the survival of *T. evansi* in chilled meat. However, it is reasonable to expect that survival in chilled meat will not exceed the survival of *T. evansi* in refrigerated blood, which is at least 48 hours (Reid et al 2001b).

Live organisms have been recovered from unclotted blood held at 4°C for 48 hours (Reid et al 2001b), although survival at ambient temperatures and exposed to light is only a few hours.

**Vectors**

*T. evansi* does not survive very long on the mouthparts of biting flies. The probability of survival of the agent on the mouthparts of tabanid flies decreases rapidly with time between successive feeds, from 0.5 at less than 15 minutes to 0.04 at 1 hour and 0.0003 at 24 hours (Nieschultz 1927).

**1.6.3 Modes of transmission**

**Vectors**

The only known means of transmission is mechanical, chiefly by means of biting flies. Tabanids (eg horseflies, march flies) are the most important vectors; stable flies (*Stomoxys calcitrans*) have also been implicated, but not proven. The role of other biting insects, such as mosquitoes and buffalo flies (*Haematobia irritans exigua*), is not known.

Vampire bats may act as both host and vector.

**Live animals**

Oral transmission to dogs through the ingestion of infected meat was first shown experimentally in 1907 (Cleland 1907, Raina et al 1985). Oral transmission occurs in vampire bats (Hoare 1972).

**Animal products**

The fragility of *T. evansi* outside the host means that spread by animal products or biologicals is of no significance. Cryopreserved material is a theoretical risk, but there are no reports of spread by this means.

There is anecdotal evidence of outbreaks in dogs and zoo carnivores fed infected meat (Losos 1986).

**Instruments and equipment**

As the parasites are in the blood of an infected animal, passage via needles, surgical instruments, dehorners and other such equipment contaminated with fresh infected blood is likely.

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2 http://www.promedmail.org (archive ref: 19990830.1518 and 20000707.1131
1.6.4 Factors influencing transmission

Effects of rainfall
Tabanids are widespread and common during the wet season in coastal and subcoastal regions of Australia, but numbers decrease greatly and are focally distributed around permanent water and swamps during the dry season.

There is insufficient information to determine whether transmission may be more or less likely during the wet season. Extrapolation from another parasite model (Putra 1991) would suggest that concentration of susceptible animals and tabanids at watering points may favour transmission during the drier months, except on flood plains where animals may be forced to congregate on islands created by floodwater during the wet season.

Windborne vector spread
Tabanids tend to fly upwind in response to host stimuli, reportedly only over distances less than about one kilometre (Foil 1989). Little else is known about windborne spread of tabanids, and there is no information about this characteristic in Australian species.

Other factors
Tabanids are the most efficient natural vectors of surra because of their large mouthparts and persistent feeding behaviour. These flies have a large, painful bite, and animals do not usually permit them to complete their blood meal uninterrupted. If the feeding is completed on a single animal, the chance of transmission is reduced, but it is increased if the fly, after interruption, quickly resumes feeding on a nearby uninfected animal (Foil 1989).

Experimental studies in the United States on the transmission of equine infectious anaemia, a disease that is also transmitted by tabanids, have demonstrated a linear correlation between horse-to-horse distance and the percentage of flies refeeding on the same horse (Issel et al 1988). A separation of 200 metres has been shown to effectively eliminate spread of equine infectious anaemia because of the tendency of flies to complete their blood meal on the original host (Foil 1989).

Little is known about the behaviour of tabanids in Australia, but surra is likely to have an increased prevalence in low-lying timbered areas and near rivers where environmental conditions are favourable for the breeding of insect vectors. Studies on tabanid behaviour in the United States indicate that they breed in or near water, the larvae living in mud or at the bottom of dams, swamps and creeks (Kettle 1984). The adults are active during the day, particularly during hot, sultry weather. Most species will not enter buildings in pursuit of a blood meal, and the rate of attack on horses at pasture decreases with distance from wooded areas.

The level of parasitaemia in individual hosts would also influence the likelihood of transmission.


1.7 Manner and risk of introduction to Australia

Introduction by hosts

Surra could be introduced to Australia by the import or illegal entry of an inapparently infected animal. It has been postulated that surra was originally spread from Africa to Asia by infected camels used in caravans and military campaigns. Further spread throughout Asia and to South America is thought to have followed export of livestock from India.

Provided appropriate quarantine restrictions are maintained, surra is unlikely to be introduced to Australia by legally imported animals from known endemic areas. Disease in horses, dogs and cats is usually acute and terminal, with a maximum duration of about 90 days. In epidemiological terms, these animals usually play only a short-term role in the spread of the disease. However, horses and dogs that survive a clinical disease episode may become persistently infected and, therefore, pose a quarantine risk. As a wide variety of wild animals and ruminants are effective carriers of trypanosomes, particular attention should be paid to quarantine protocols for zoo animals and camelids from endemic areas.

A possible route of introduction is by the accidental or illegal entry of an infected dog, deer or pig from countries to the north of the Australian mainland. Entry of live animals from the Torres Strait Protected Zone onto the Australian mainland is regulated under Quarantine Proclamation 166A. Ocean-going vessels are inspected by Australian Quarantine and Inspection Service officers to ensure compliance. This risk is also managed by public awareness programs targeting relevant groups in northern Australia (eg Torres Strait communities and schools, primary producer organisations) and by the inclusion of testing for surra in the Northern Australia Quarantine Strategy surveillance program.

Deer are known to swim between the PNG mainland and the northernmost islands in the Torres Strait, but the northern island group and other Torres Strait islands are separated by 35 kilometres. Further southerly spread by dogs and pigs on watercraft within the area is a threat.

Introduction by vectors

Introduction by vectors is not a significant risk because *T. evansi* does not survive very long on the mouthparts of biting flies.

Introduction by animal products, biologicals and genetic material

Introduction by frozen semen or other cryopreserved material is theoretically possible. Other products are unlikely to introduce *T. evansi* because the parasite does not survive long outside the host (See Section 1.6.3).
2 Principles of control and eradication

2.1 Introduction

There are several principles to be considered in the control and eradication of surra:

- quarantine and isolation of cases and exposed animals;
- movement controls and zoning;
- effective tracing and surveillance in domesticated and feral populations;
- testing of animals before movement out of the infected zone;
- treatment of animals;
- vector control (tabanids);
- selective depopulation of buffer zones; and
- slaughter of wild or uncontrolled animals.

Although limited outbreaks of surra have been eradicated in Australia and elsewhere, no country is known to have eliminated the disease once it has become well established.

2.2 Methods to prevent spread and eliminate pathogens

2.2.1 Quarantine and movement controls

Surra is not spread by direct contact between susceptible animals. However, quarantine and movement controls on affected and neighbouring properties aid in preventing the spread of *T. evansi* by the movement of diseased animals, and stringent movement restrictions may be implemented immediately the infected area has been identified. Initially, controls on movement and congregation should be imposed on susceptible domestic animals. These may be modified once the situation has been fully investigated.

Quarantine and movement controls can be imposed at several levels. Infected premises (IPs) and dangerous contact premises (DCPs) can be identified and a restricted area (RA) drawn around all IPs and DCPs (see Appendix 1). The shape and dimensions of the RA are determined by factors such as livestock concentrations, weather conditions, the distribution and movements of susceptible wild and feral animals, the topography and the presence of possible vectors. A level of movement control and surveillance applies in the RA.

A control area (CA) can be imposed around the RA. The purpose of the CA is to control movement of susceptible livestock for as long as is necessary to complete tracing and epidemiological studies. Less stringent movement controls and surveillance apply in the CA. Once the limits of the disease have been confidently defined, the CA boundaries and movement restrictions can be reassessed and modified.
If chemotherapy is used before movement from the CA, restrictions on treated animals can be less onerous than for nontreated animals, but movement must begin immediately after treatment because the arsenical drugs used for treatment have a period of therapeutic effectiveness of only a few hours.

Introduction of semen and embryos into a CA can minimise disruption to livestock breeding. However, semen from a CA should be regarded as potentially infected and not be allowed out.

Movement controls can be maintained until the disease is either eradicated or declared endemic.

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

Zoning

While there are no OIE guidelines specifically covering zoning for surra, zoning in accordance with OIE principles is an important strategy to reduce the social and economic impact of an outbreak of surra in Australia. This may facilitate exports and allow continuation of local, national and international competition and movement of livestock (particularly of horses and dogs originating from outside the infected zone).

The area at risk of infection by surra depends on the time of year, the geographical range of reservoir hosts and the distribution of competent insect vectors. If it can be established that the disease has only been introduced 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

Trace-back and trace-forward of all direct contacts with infected animals are vital. Tracing should include:

- all domestic and zoo animals;
- fresh meat slaughtered for pet food or for consumption by zoo animals; and
- semen.

It is possible that the first reported case will not be the index case. Trace-back may identify other cases and may establish how the disease entered Australia.

Trace-back and trace-forward periods are influenced by species, season and availability of resources.

2.2.3 Surveillance

Surveillance to determine the extent of an outbreak and to provide confidence that the outbreak has been controlled will vary greatly depending on the species in which the disease is detected and husbandry practices in the area of the outbreak. For example, the type of surveillance established after an outbreak of surra in a southern horse stud would be very different from that needed on a northern extensive cattle property.
Because of the variation in expression of disease between species, surveillance involving horses and dogs should be based on observation of clinical signs, supported by parasitological and serological testing, whereas in cattle, buffalo and small ruminants greater reliance should be placed on serological testing. Care must be exercised in interpreting the results of serological tests to detect infection in individual animals (see Section 1.4.3).

Where domesticated animals are concerned, each individual on a DCP should be examined and tested. However, when dealing with feral animals adjacent to an IP or DCP, statistically valid sampling to determine freedom is a more practical approach. That vectors are more likely to be active near water sources should be taken into consideration when surveying wild or feral animals. Horses and dogs present on IPs and DCPs will serve as useful sentinels, if examined regularly.

Although laboratory infections using very large infective doses can produce infective animals after a few days, under field conditions (in which natural infections from biting insects result in much smaller infective doses) it is unlikely that animals would become infective within one week. Seroconversion to *T. evansi* under field conditions generally occurs within 2–3 weeks after infection.

Given that the organism survives for less than a day outside a mammalian host, that seroconversion does not occur until 2–3 weeks after infection and that transmission is by mechanical rather than biological vectors, monthly testing of controlled, domestic animals over a period longer than 3 months is unjustified if the risk of exposure to infected animals has been removed (eg by treatment or by placing them in insect-proof enclosures). However, where uncontrolled hosts such as native or feral animals are potentially present, the period over which domesticated livestock should be under surveillance would be governed by the frequency and intensity of feral animal surveys.

2.2.4 Treatment of infected animals

If eradication of the disease is not possible, treatment of animals diagnosed with the disease is an effective option for control. Although melarsomine is the only drug with high efficacy against *T. evansi* (see Section 1.4.5), it is not currently registered by the Australian Pesticides and Veterinary Medicines Authority for use in Australia to treat surra. The withholding period for melarsomine is 14 days for milk and meat in camels. There are no data on the withholding period in other species. The likely detection time of melarsomine in racehorses is unknown.

Treatment of clinically affected horses should be carried out on the affected property. On an IP, animals with suspicious signs (eg recurrent fever) should be treated immediately with melarsomine and held during daylight hours in facilities that will reduce biting by tabanids.

2.2.5 Destruction of animals

All states and territories have the legal authority to destroy infected or suspect animals. However, a decision to destroy all susceptible animals on an IP (stamping out) because of an outbreak of surra would in many cases depend on compensation being available (see Section 3.5). Potentially serious complications of a stamping-out strategy for surra include the high monetary and genetic values of many horses and zoo animals, and emotional distress arising from the bond
between companion-animal owners and their horses, dogs and cats. Stamping out might be of value for an index case in a single, recently imported animal.

Although reliable chemotherapy is available, there may be circumstances in which it is appropriate to destroy individual animals (see the Destruction Manual).

### 2.2.6 Treatment of animal products and byproducts

Trypanosomes would not survive the pasteurisation of milk or freezing of meat.

At room temperature, the parasite does not survive longer than 24 hours after the death of the host animal, but it may survive several days in refrigerated meat. Carnivores can become infected after eating tissues from a freshly killed parasitaemic animal, but normal cooking processes rapidly destroy the organism.

See also Section 1.6.2.

### 2.2.7 Disposal

*T. evansi* cannot survive outside a live host for more than about 24 hours, so precautions usually taken when disposing of carcases or milk from suspect or infected animals may be relaxed accordingly. Care should be taken to deny access by carnivorous mammals to fresh carcases. Meat taken from infected or suspect carcases should not be fed uncooked to dogs within 24 hours of the death of the infected or suspect animal.

### 2.2.8 Decontamination

*T. evansi* is unlikely to survive for more than a few hours on animal handling equipment or on surfaces at animal handling facilities. Consequently, normal hygienic practices are adequate for the decontamination of premises during an outbreak and to prevent spread by fomites to other properties.

Specific methods of decontamination include heat, disinfectants, burial and washing. Possible treatments applying to various items include:

<table>
<thead>
<tr>
<th>Risk material</th>
<th>Possible decontamination methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>burial, flushing with water, disinfection, heat</td>
</tr>
<tr>
<td>Hypodermic needles</td>
<td>disinfection, heat</td>
</tr>
<tr>
<td>Carcases</td>
<td>heat, burial</td>
</tr>
<tr>
<td>Vehicles</td>
<td>washing, disinfection</td>
</tr>
<tr>
<td>Clothing</td>
<td>washing, disinfection</td>
</tr>
</tbody>
</table>

Given the fragility of *T. evansi*, disinfection with hypochlorites, phenolics, iodophores, quaternary ammonium compounds, acids or alkalis could be expected to rapidly destroy the organism, although specific trials of these compounds on *T. evansi* have not been reported in the literature. It is rapidly destroyed by heat.

### 2.2.9 Vaccination

There is no vaccine available to control surra.
2.2.10 Wild animal control

There is no information on the susceptibility of Australian native animals, other than the experimental infection of agile wallabies and pademelons (see Section 1.4.1). Therefore, if *T. evansi* were to enter an area in northern Australia, wallabies might be a good indicator animal (high mortality in free-living populations could be one of the first signs of an incursion).

The presence of feral animals would pose a serious threat to the control of surra. The wild animals most likely to carry surra are horses, donkeys, cattle, buffalo, camels, dogs, dingoes and pigs. In some circumstances, depopulation of these species may be an option. Other species that could be involved are goats and deer. Rabbits and rodents are unlikely to be important because of their high rate of mortality after infection, and because their nocturnal activity and small size reduce the chance that they will be bitten by tabanids.

For further details on wild animal surveillance and control, refer to the Wild Animal Response Strategy.

The distribution and movements of feral and Australian native animals should be considered when determining the boundaries of RAs and CAs.

2.2.11 Vector control

Tabanids

No control is used routinely against tabanids in extensive pastoral areas. Regional control might be achieved by insecticidal fogging of vegetation within one kilometre of swamps and other breeding and resting sites, such as densely wooded areas.

The application of residual synthetic pyrethroids to the host as an insect repellent may assist in reducing the frequency of feeding by tabanids but may have little overall effect on the risk of transmission of *T. evansi*.

Insect traps can also be useful, the most effective being the Manitoba trap (see Appendix 5). However, there is little information on the ecology of Australian tabanids to support this recommendation.

Since tabanids are most active in the middle of the day in sunlight (Coetzer et al 1994), housing animals during the day would offer them protection. Allowing animals to have access to field shelters could also help separate hosts from vectors. Rugs and hoods can also be used to decrease the exposure of horses to insect vectors.

Stable flies

Control of stable flies can be achieved by removing all horse dung from stables and horse paddocks every few days. Stacking the dung in a heap in the sun to promote heating through fermentation will kill the fly eggs. This is a practical measure in stables and small paddocks but becomes progressively less easy as the size of the paddock increases. Daily application of synthetic pyrethroid to animals may also provide an effective deterrent.
2.2.12 Sentinel and restocking measures

The use of sentinel animals to assess the effectiveness of control/eradication measures for surra will be imperfect because the disease may be present at low prevalence (in wild hosts), or because of possible fluctuations in the distribution and number of vector insects. Imperfect serological tests may also fail to detect the infection. However, clinical disease could be monitored.

Susceptible horses distributed near tabanid breeding locations during the summer months would provide the best means of monitoring the presence of the agent in residual hosts. Horses should be observed weekly and tested serologically each month with antibody-ELISA.

Sentinel animals may be drawn from local populations, provided they are seronegative, or drawn from free areas. Restocking can start two days after the last susceptible host is treated or removed.

2.2.13 Public awareness

Target groups for awareness promotion in a campaign against surra include:

- horse owners/carers — peak racing industry groups, horse councils, equestrian organisations, show societies, importers/exporters;
- cattle producers — peak beef and dairy industry groups;
- owners of all animals (other than birds and reptiles) and their associations;
- wildlife rangers, naturalists and land agents;
- residents within affected areas, including Indigenous communities;
- veterinary surgeons; and
- the community at large.

Key messages should include:

- surra’s long history in endemic areas, which demonstrates that humans are not susceptible to infection;
- the importance of reporting and investigating suspect animals, especially within the CA;
- the need for cooperation during extended surveillance programs and while movements are restricted;
- the means by which the risk of infection can be minimised (eg stabling, application of insect repellent, avoiding congregations of stock, isolating stock from tabanid breeding areas, moving stock by night); and
- why local wildlife populations may need to be destroyed for the sake of the broader wildlife population.

2.3 Feasibility of control in Australia

Three major factors determine the feasibility of controlling surra in Australia:

- the extent of the outbreak before detection;
- the location of the outbreak; and
• the species in which it occurs, particularly if feral and Australian native animals are involved.

For example, an outbreak on a southern horse stud would probably be rapidly detected, the movement of dangerous contacts would be known and the potential of the disease to overflow into feral animals would be limited. In such a situation, eradication would be entirely feasible.

However, if surra were first detected on a northern cattle property, initial signs might have been missed and the disease may have become established over a wide area in domestic, wild and feral animals before detection. Eradication in this situation might prove to be uneconomic, in which case efforts would concentrate on regionalising the disease and treating animals in specific cases.

Control could involve confining the disease to one or more regions, regular treatment of animals likely to show severe clinical signs, such as stock horses, and treatment before movement out of the infected region. Tests currently available are not sufficiently reliable to allow movement based only on the results of a negative test.
3 Policy and rationale

3.1 Overall policy

The policy is to eradicate surra where practicable; the initial response to an outbreak should have eradication as its goal.

However, this plan recognises that in some situations eradication will not be a viable option. The disease is insidious in some species, leading to the possibility of infection becoming well established before detection, both spatially and in a variety of wild and domestic species. In these circumstances, the policy is to establish a control program that would slow the spread of the disease and reduce any impact on trade.

A decision on whether to eradicate or control would be based on criteria such as:
- geographical spread;
- numbers of domestic animals involved;
- level of control over domestic species;
- establishment in feral or Australian native animals;
- location of the outbreak; and
- effect on trade.

The following strategies are recommended for eradication and control of surra.

ERADICATION:
- modified stamping out, involving quarantine, and slaughter or treatment with melarsomine of all infected and exposed susceptible animals on the index property; slaughter could include turnoff to abattoirs under controlled conditions;
- quarantine and movement controls on animals in declared areas to prevent the spread of infection;
- tracing and surveillance to determine the source and extent of the outbreak;
- establishment of an animal-free buffer zone around the restricted area;
- zoning to define infected and disease-free areas for trade purposes; and
- a public awareness campaign to facilitate cooperation from industry and the community.

CONTROL:
- treatment with melarsomine, where feasible, while the extent of the outbreak is being evaluated; and
- eradication strategies as described above, but with salvage-slaughter at abattoirs.

An uncontrolled outbreak of surra would cause production losses in the beef and dairy industries and an ongoing cost to the horse industry. Costs of control in horses would also affect the beef industry, in which horses are still used extensively to manage stock. The likely impact on Australian native fauna is largely unknown.

Surra in horses is notifiable internationally as an OIE Listed disease. In Australia, surra is an EAD Response Agreement Category 4 disease. Category 4 diseases are those for which the costs will be shared 20% by government and 80% by industry.
The chief veterinary officer (CVO) in the state or territory in which the outbreak 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 Emergency Animal Diseases (CCEAD), the state/territory and Australian governments, and representatives of the affected industries. The detailed control measures adopted will be determined using the principles of control and eradication (Section 2) and epidemiological information about the outbreak.

For further information on the responsibilities of the state/territory disease control headquarters and local disease control centres, see the Control Centres Management Manual, Part 1.

### 3.2 Strategy for control and eradication

The strategy is to eliminate the disease as quickly as possible by stamping out and other eradication measures, if feasible, in a small well-defined outbreak, or by using treatment and control measures in a less well-defined outbreak. The decision on whether to eradicate or control will be based on criteria such as:

- geographical spread;
- numbers of domestic animals involved;
- level of control over domestic species;
- establishment in feral or Australian native animals;
- location of the outbreak; and
- reaction from trading partners.

The action taken will depend on the manner and place in which a diagnosis is made, and on the confidence with which the geographical limit of infection can be assumed.

Clinical cases should either be euthanased immediately or treated with Cymelarsan (melarsomine hydrochloride) and isolated from tabanids by stabling, relocation away from tabanid breeding sites, rugging and application of insect repellent.

In-contact livestock should be treated with melarsomine and isolated from tabanids, or sent to slaughter. Movement to slaughter must be managed to avoid contact with vectors. Travelling animals should be spelled only at night and be slaughtered on the morning of arrival at the abattoir.

See Section 3.2.3 for further details on treatment and Section 3.2.7 for further details on insect control.
3.2.1 Stamping out

Conventional stamping out (destroying all potential hosts on an infected premises, as opposed to modified stamping out) is not recommended in most outbreaks. It may be an acceptable strategy in an outbreak that is detected early enough and in which all susceptible animals can be destroyed, or where there is confidence that residual hosts do not exist elsewhere.

Modified stamping out involves quarantine and slaughter or treatment with melarsomine of all infected and exposed susceptible animals on the index property. Slaughter could include turnover to abattoirs under controlled conditions. Valuable animals may be salvaged by treatment (see Section 3.2.3).

3.2.2 Quarantine and movement controls

The premises on which infected or suspect animals are detected will be subjected to quarantine and movement controls and will be officially declared an infected premises (IP). Adjacent premises will ordinarily also be quarantined and may be classed as suspect premises (SP). Susceptible animals on SPs may be subjected to testing.

Confinement of infected or suspect animals within specified limits maintains confidence that spread of the parasite by movement of animals to free areas is not occurring, and allows time for an epidemiological investigation to be undertaken so that more informed control measures can be implemented.

As vector transmission of *T. evansi* is by mechanical means only, and transfer from an infected animal is unlikely over distances greater than 200 metres, the IPs, SPs and immediately neighbouring premises may constitute the restricted area (RA) in the first instance while investigations into the extent of the infection are continuing.

Because vector transmission is limited to about 200 metres, the establishment of an animal-free buffer zone can prevent local spread of infection. This would require the removal from the buffer zone of all susceptible hosts, including domestic, feral and wild animals.

If it is considered that the parasite may be established in reservoir hosts, particularly feral or wild animals, a control area (CA) should be declared immediately. In the early part of the outbreak, the initial CA boundaries may correspond to state borders. The CA boundary can be modified as more information becomes available from investigations.

Dangerous contact premises (DCPs) are those that have received susceptible animals from an IP within a specified period (in this case 120 days, or twice the maximum incubation period). Where all stock on a premises are susceptible to clinical disease (e.g., horses), 120 days will be an adequate interval; where all stock are less susceptible (e.g., cattle) or where species of differing susceptibility are mixed (e.g., horses and cattle), a longer period must be considered. The need for, and size of, an RA to surround IPs and DCPs will be determined at the time, taking into account the time of year and likely intensity of vector activity.
Movement controls will be enforced until the status of declared areas can be redefined or until other strategies can be implemented (e.g., chemotherapy). Movement of animals out of the RA will be controlled.

The movements of wild animals should be considered when determining the boundaries of declared areas.

See Appendixes 1 and 2 for further information on declared areas and movement controls.

### Zoning

Once the epidemiological information has been collated and assessed and the disease distribution is defined, it may be advantageous to establish a zoning strategy with appropriate movement controls so that international trade, particularly in horses, dogs, and cats, may recommence at the earliest possible time. This should reduce the social and economic effects of the outbreak for those parts of the country remaining free of the disease.

There are no criteria in the *OIE Terrestrial Animal Health Code* for the zoning of surra. Acceptance of a zoning policy will have to be bilaterally negotiated with international trading partners, and this is likely to take some time. The feasibility of zoning will depend on the extent of the distribution of the disease and the range of host species involved in the outbreak.

The size of the infected zone will depend on the location of the index case, the potential for transmission from feral animal and wildlife reservoirs, the time of year, and other ecological factors that may have a bearing on vector activity and distribution. The infected zone should approximate the CA.

The movement of susceptible animals out of the infected area will only occur under conditions that require treatment before departure.

See Appendix 4 for further information on zoning.

#### 3.2.3 Treatment of infected animals

Infected animals may be effectively treated with melarsomine. As a risk-reduction measure, treatment, where it can be applied, is equivalent to destruction. Because of the short duration of prophylaxis, treatment timing should be managed with vector exposure strategies to reduce the risk of re-exposure. These strategies could include movement to a vector-free area, biting fly control or cessation of normal husbandry procedures.

Because the half-life of the drug is approximately 6 hours, the duration of effectiveness is only a few hours. All susceptible, exposed animals in a group must therefore be treated simultaneously.

Applying the same treatment strategy on SPs may eliminate the infection before it can be detected by serological or other methods.

Where infection is found in a recently imported horse or its cohort, prompt and simultaneous treatment with melarsomine of susceptible animals, whether infected or not, within a radius of 200 metres is appropriate. Treatment should include
animals likely to have been within this radius of the infected animal during the period of illness and since import. The treatment zone may need to be extended depending upon the results of tracing and surveillance.

See Section 1.4.5 for further details on treatment with melarsomine.

Where treatment is not an option for the owner or is impractical, animals within the zone should be slaughtered. The presence of chronically infected animals would reduce productivity and become an unavoidable financial burden on farming enterprises in the zone.

Movement of susceptible animals outside the zone should not be permitted without treatment.

Routine surgical procedures such as dehorning, castration and vaccination, where there is a risk of transfer of blood between animals on contaminated instruments, should be avoided (see Section 2.2). Where husbandry is necessary, instrument decontamination procedures, anti-fly treatments and additional surveillance of stock should be employed.

3.2.4 Treatment of animal products and byproducts

Fresh tissues are considered to be a risk if consumed by a susceptible species. Fresh, unchilled meat from infected carcases should not be fed to animals within 24 hours of death. Because of the uncertain survival period of the parasite under chiller conditions, chilled meat or milk should not be fed. Meat must be frozen for at least 72 hours before being fed to carnivores.

Humans are not at risk from surra. There is no risk from processed dairy products, including pasteurised milk, or from wool, other fibres, salted hides or materials that have been rendered.

3.2.5 Vaccination

No effective vaccine is available.

3.2.6 Tracing and surveillance

Tracing

Tracing will be used to determine movements of domestic animals susceptible to infection (not only to clinical disease) onto and off the IP during the period from 120 days before the first signs of clinical disease to the introduction of quarantine and movement controls.

Surveillance

All susceptible domesticated animals on IPs and DCPs should be examined weekly for clinical signs and undergo monthly testing using MAECT and antibody-ELISA for 3 months, followed by quarterly testing for a further 9 months. Where potentially susceptible native or feral animals are on or adjacent to the property, quarterly testing of susceptible domestic animals for longer than 12 months should be considered in the light of results from wild/feral animal surveys.
All other susceptible stock within the RA should be examined as often as is practically possible and be tested using MAECT and antibody-ELISA on a quarterly basis for one year.

Surveys of feral and wild species adjacent to IPs should take place each month for 3 months and then quarterly for at least a further year. Subsequent survey frequency will depend on the results and intensity of earlier surveys. The clustering effects caused by increased vector activity near water sources should be taken into consideration when designing the surveys.

Surveillance of vectors to detect *T. evansi* is appropriate, as PCR tests have proved effective for detecting the parasite in blood meal in the gut of tabanids. Manitoba traps could also be used to identify periods of peak vector activity, which would help determine the timing of serological surveys of susceptible domesticated and wild species.

### 3.2.7 Vector control

**Tabanids**

Biting of stock by tabanids could be reduced by isolating stock from swampy areas and other tabanid breeding sites, such as densely wooded areas. In addition, insecticidal fogging of vegetation in breeding sites should be considered.

Another option for the control of biting by tabanids is the daily application to the host of a residual synthetic pyrethroid, which acts as a repellent.

Traps can also be useful, the most effective being the Manitoba trap (see Appendix 5). However, there is little information on the ecology of Australian tabanids to support this recommendation.

Since tabanids are most active in the middle of the day in sunlight, housing animals during the day would offer them protection. Allowing animals to have access to field shelters could also help separate hosts from vectors.

**Stable flies**

In stables and small horse paddocks, stable flies should be controlled by removing all horse dung every few days and stacking it in a heap in the sun to promote heating (through fermentation) to kill the fly eggs. This measure is less practical in larger paddocks. Daily application of synthetic pyrethroid to animals and keeping them in stables during the day may also provide an effective deterrent.

Further details on vector control are given in Appendix 5.

### 3.2.8 Decontamination and disposal

Because of the short survival of the parasite outside the host and the low risk of infecting a second host other than by injection, intensive decontamination of an infected property or facilities within the property is not required. *T. evansi* is also rapidly destroyed by heat.

Fresh blood should be buried or washed away. Hypodermic needles used on infected animals should be burned, buried or disinfected with an approved
disinfectant. Appropriate disinfectants are hypochlorites, phenolics, iodophores, quaternary ammonium compounds, acids and alkalis. If medical waste is to be removed from a property for disposal as described in legislation, it should be placed in prescribed containers, which are then disinfected and placed in plastic bags. The plastic bags are then sealed and disinfected.

Carcases should be buried or secured unrefrigerated in a place where carnivores cannot gain access for at least one day. Infected carcases can be disposed of outside the RA, provided that these precautions are taken.

Animal tissues and blood should be washed from clothing, equipment or vehicles before they are taken from the RA.


3.2.9 Wild animal control
The distribution and density of susceptible wild animal hosts need to be determined early in the outbreak. Wild animal populations on IP's and DCPs may be eliminated if considered to be a risk.

Where elimination is decided on, wild pig and wild dog control programs should be instigated or intensified to reduce numbers, and kangaroo/wallaby numbers should be reduced by shooting. Destruction of feral, native and uncontrollable susceptible hosts should be undertaken before mass chemotherapy of domestic animals.

If animal-free buffer zones are created, ongoing depopulation of wild animals will be necessary across a much broader area to ensure that normal home-range movements of wild animals do not include the buffer zone.

For further information, see the Wild Animal Response Strategy.

3.2.10 Media and public relations
The industry and the media must be informed that prevailing circumstances will determine very strongly the most appropriate control measures. The rationale for control policies for a vector disease like surra will be more difficult to promote to the horse and livestock industries than previous disease control measures for diseases such as tuberculosis, brucellosis and foot-and-mouth disease, which have used stamping out. The pivotal role of vectors in disease distribution will be the most difficult aspect to convey to the livestock industry.

Public concern may be expressed over the destruction of wildlife and the environmental effects of vector control programs. The media strategy should take these issues into consideration.

For further information, see the Public Relations Manual.
3.3 Social and economic effects

3.3.1 Horses, dogs and cats

The diverse nature of the horse industry makes it difficult to determine the social and economic impact that an outbreak of surra and its control would have in Australia. Because Australian horses have not been exposed to the disease, mortality in infected animals and direct economic loss would probably be high if a reliable treatment were not readily available.

During the wet season in northern Australia, when environmental conditions are extremely favourable for the activity of tabanid vectors, disease is likely to be more widespread. Establishment of surra in areas of extensive livestock production would hinder the use of stockhorses to muster cattle and increase property management costs.

Quarantine of an IP could result in severe consequential loss if the premises is a racing stable, stud, agistment farm, equestrian centre, greyhound racing kennel, commercial boarding kennel or cattery, or tourist operation.

If an IP is in a major racehorse training centre, where horses are trained in closely contiguous premises, imposition of movement controls in the RA and CA could suspend racing until treatment is available and epidemiological investigations are completed. Treated horses might not be able to race for some time (the likely detection period for melarsomine in horses is uncertain). As an example of potential losses, total suspension of horse racing in Victoria for one month would affect around 77 race meetings, resulting in losses of almost $300 million (Racing Victoria, pers comm). There would be additional income loss by a significant number of full-time or part-time employees and by ancillary service providers. These losses would be greater during a major racing carnival. In some areas, the economic effect of a surra outbreak on greyhound racing would also be significant.

Significant but unquantifiable social disruption and economic impact would also result from the probable cancellation of a wide range of other local events at which susceptible host animals are assembled outdoors, such as agricultural and equestrian events, pony and adult riding club rallies, polo/polocrosse tournaments, and rodeos.

The emotional bond between many owners and their companion horses, dogs and cats is such that the social effect of deaths during an outbreak and delays in scheduled exports of dogs and cats would be considerable for individual owners. Deaths of clinically affected horses, dogs and cats, and their destruction for welfare reasons (if a reliable trypanosocidal treatment is not readily available), would result in widespread community concern and media attention.

3.3.2 Cattle and buffalo

There is a lack of objective information on the effect of surra on animal health and productivity in ruminants. In endemic areas, buffalo and cattle are often symptomless carriers of infection (Payne et al 1991b, Coetzer et al 1994), which leads to an assumption that surra does not cause disease in these species. Nevertheless, inapparent infections may cause significant production losses through decreases in milk yield, bodyweight gain and draught power, and increases in calving intervals. Epidemics with significant debility and mortality do
occur when the parasite is introduced to new regions or when susceptible animals are imported to endemic areas. A lack of data on the effect of *T. evansi* on productivity in beef and dairy cattle is not sufficient reason to assume that its effect in Australia would be inconsequential, particularly in extensively farmed areas where cattle are subject to intercurrent stress.

The cost of chemotherapy of cattle in extensive areas would be considerable, as it would include not only the cost of the drug but also the cost of mustering. Movement restrictions would cause loss of market opportunities and associated financial losses to nonaffected properties, and to support industries such as the livestock transport industry. This effect may be reduced by zoning and/or chemotherapy of animals before movement.

### 3.3.3 Exports

Major Australian markets for live horses (such as New Zealand, the European Union, Japan, Singapore, Macau and Hong Kong) are free of surra. Export trade and international horse competitions would be disrupted until new conditions for trade were negotiated with trading partners. Trade in horsemeat contributed approximately $14 million to Australian export income in 2002–03, with about 40,000 horses slaughtered annually for export (ABARE statistics, 2004). In an Australian outbreak of surra, exports of horses and horsemeat could be jeopardised, albeit with little scientific justification.

While the impact on export trade would be greatest for the horse industry, emerging markets for cattle and sheep in the European Union, Turkey and the Commonwealth of Independent States and for exported dogs and cats would also be affected.

### 3.4 Criteria for proof of freedom

There are no guidelines in the *OIE Terrestrial Animal Health Code* for proof of freedom from surra. However, active surveillance of the infected zone should be continued for a minimum of one year after the last case, and there must be no trypanocidal treatment of animals in the zone during the 9 months before declaration of freedom. Absence of any evidence of infection over a 3-year period should provide a sound case for declaration of freedom.

In zones where evidence of infection has not been recorded, declaration of zonal freedom from infection is automatic once an effective surveillance scheme is in place.

See Appendix 4 for further details on criteria that can be used to establish proof of freedom.

### 3.5 Funding and compensation

Surra is classified as a Category 4 emergency animal disease under the EAD Response Agreement between the governments of Australia and the livestock industries.

Category 4 diseases are those that could be classified as being mainly production loss diseases. While there may be international trade losses and local market disruptions, these would not be of a magnitude that would be expected to
significantly affect the national economy. The main beneficiaries of a successful emergency response to an outbreak of such a disease would be the affected livestock industries. For this category, the costs will be shared 20% by governments and 80% by the relevant industries (refer to the EAD Response Agreement for details).

Further information on cost-sharing arrangements can be found in the Summary Document and in the Valuation and Compensation Manual.

3.6 Strategies if the disease becomes established

If surra becomes established in Australia, control efforts will be aimed at controlling the infection within one or more zones to reduce the potential damage to trade, and ensuring that it does not spread outside the zone. If infection is detected outside the established zone, the policy will initially be to eradicate the disease from the new area.

Economically or socially valuable animals within the zone should be treated with Cymelarsan (see Section 3.2.3). Movement of susceptible animals outside the zone should not be permitted without treatment.
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 surra or the surra disease agent (*T. evansi*) 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 from 120 days before the first clinical signs to the introduction of quarantine; and

- premises contiguous with the IP (because of vector transmission).

Suspect premises (SP)
Premises classified as SPs will be those that contain animals that have possibly been exposed to *T. evansi*, such that quarantine and surveillance are warranted; OR animals not known to have been exposed to *T. evansi* but showing clinical signs requiring differential diagnosis.

The SP classification is a temporary classification because the premises contains animals that are suspected of having the disease. High priority should be given to clarifying the status of the suspect animals so that the SP can be reclassified as either an IP and appropriate quarantine and movement controls implemented, or as free from disease, in which case no further disease control measures are required.

Restricted area (RA)
In the first instance, while investigations into the extent of infection are continuing, the IPs, SPs and DCPs may constitute the RA.

The RA should cover an area of at least one-kilometre radius around the IP. It is important to prevent the spread of the disease by animal movements, although some local spread may still occur through movement of infected vectors (some overseas tabanids have a range exceeding two kilometres; Kettle 1984). A distance of one kilometre should ensure that the disease is contained if there are no illegal movements of animals.

In extensive farming areas, the RA boundary should extend to at least 50 kilometres beyond known infected premises until the results of surveillance testing can confirm the extent of infection. The boundary 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. The size of the RA will be determined taking into account the time of year, the likely range of vector activity and whether it is likely
that the parasite is established in reservoir hosts (see the Wild Animal Response Strategy).

Control area (CA)
The CA may initially be as large as a state or territory, but may be modified on the basis of epidemiological, meteorological, geographical and ecological data as they come to hand. Even in remote areas, the CA must include premises adjacent to the IP.

In establishing the boundaries of the CA, consideration should be given to long-term strategies if zones have to be developed.
## Appendix 2 Recommended quarantine and movement controls

### Premises

<table>
<thead>
<tr>
<th>Movement controls</th>
<th>Infected, dangerous contact and suspect premises</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Movement out of:</strong></td>
<td></td>
</tr>
<tr>
<td>– susceptible animals(^a)</td>
<td>Allowed after treatment and permit</td>
</tr>
<tr>
<td>– animal products and animal byproducts, other than semen and milk</td>
<td>Allowed without restriction</td>
</tr>
<tr>
<td>– semen</td>
<td>Prohibited</td>
</tr>
<tr>
<td>– milk</td>
<td>Can be released to an approved facility for pasteurisation</td>
</tr>
<tr>
<td>– embryos</td>
<td>Allowed without restriction</td>
</tr>
<tr>
<td>– farm products (eg hay, crops)</td>
<td>Allowed without restriction</td>
</tr>
<tr>
<td><strong>Movement in and out of:</strong></td>
<td></td>
</tr>
<tr>
<td>– people</td>
<td>Allowed without restriction</td>
</tr>
<tr>
<td>– vehicles</td>
<td>Livestock transport vehicles to be cleaned and sprayed with appropriate insecticide after each journey; other movements in and out allowed without restriction.</td>
</tr>
</tbody>
</table>

\(^a\) For susceptible animals, see Section 1.2
## Areas

<table>
<thead>
<tr>
<th>Quarantine and movement controls</th>
<th>Restricted area</th>
<th>Control area</th>
</tr>
</thead>
</table>

### Movement out of:
- **susceptible animals**: Controlled by inspection, treatment, application of insect repellent and permit. Controlled by inspection and permit; treatment not usually required.
- **semen**: Prohibited. As for RA.
- **milk**: Can be released to an approved facility for pasteurisation. As for RA.
- **meat**: Can be released after being hung in chiller for three days. As for RA.

### Movement within of:
- **susceptible animals**: Controlled by inspection, treatment, application of insect repellent and permit. Controlled by inspection and permit; treatment not usually required.

### Movement through of:
- **susceptible animals**: Controlled by inspection, treatment, application of insect repellent and permit. Controlled by inspection and permit; treatment not usually required.

### Movement in of:
- **susceptible animals**: Controlled by inspection, treatment, application of insect repellent and permit. Controlled by inspection and permit; treatment not usually required.

### Movement of susceptible animals from RA to CA:
Controlled by inspection, treatment, application of insect repellent and permit.

### Movement of susceptible animals from CA to RA:
Not applicable.

### Any movement of:
- **animal products and animal byproducts (other than meat, semen and milk)**: Allowed without restriction. As for RA.
- **people**: Allowed without restriction. As for RA.
- **specified equipment**: Movement subject to thorough decontamination. As for RA.
- vehicles  | Livestock transport vehicles moving inward and outward must be cleaned out and sprayed with appropriate insecticide after each journey | As for RA  

**Other considerations:**

- risk enterprises  | Not applicable | Not applicable  

- sales, shows or other events where animals from separately managed premises are gathered  | For susceptible species, controlled by inspection, treatment, application of insect repellent and permit |  

- stock routes, rights of way  | Not applicable | Not applicable
Appendix 3 OIE animal health code and diagnostic manual for terrestrial animals

OIE Terrestrial Code

The objective of the OIE Terrestrial Animal Health Code is to prevent the spread of animal diseases, while facilitating international trade in live animals, semen, embryos and animal products. This annually updated volume is a reference document for use by veterinary departments, import/export services, epidemiologists and all those involved in international trade.

The OIE Terrestrial Code is amended in May each year. The current edition is published on the OIE website at:

http://www.oie.int/eng/normes/mcode/A_summry.htm

There is no specific chapter in the code for surra. Relevant information is contained in Part 1 of the code, for example:

Chapter 1.3.5 Zoning, regionalisation and compartmentalisation

Chapter 1.3.6 Surveillance and monitoring of animal health

OIE Terrestrial Manual

The purpose of the OIE Manual of Standards for Diagnostic Tests and Vaccines for Terrestrial Animals is to contribute to the international harmonisation of methods for the surveillance and control of the most important animal diseases. Standards are described for laboratory diagnostic tests and the production and control of biological products (principally vaccines) for veterinary use across the globe.

The OIE Terrestrial Manual is updated approximately every four years. The current edition is available on the OIE website at:

http://www.oie.int/eng/normes/mmanual/A_summry.htm

The following chapter is relevant to this manual:

Section 2.5.15 Surra (Trypanosoma evansi)
Appendix 4 Procedures for surveillance and proof of freedom

As there is currently no OIE Terrestrial Animal Health Code chapter for surra, there are no internationally accepted guidelines on which proof of freedom can be based. The following is suggested as a guide.

Notification

Once an incursion of surra has been confirmed, the disease must be made notifiable by law in any state or territory affected (if this is not already the case). A media campaign may be required to ensure that producers, veterinarians, stock agents and others are aware of their responsibilities to report suspicious cases.

Period required to provide proof of freedom

In view of the often insidious nature of this disease, it is unlikely that proof of freedom after an outbreak in which wildlife or feral hosts are involved would be accepted internationally until after a period of three years. In a small, isolated outbreak, where no uncontrolled potential hosts exist, freedom could be achieved within one year.

Sentinels

Seronegative horses should be used as sentinel animals to detect residual infection on an IP where other susceptible animals have either been removed or treated. As the mouthparts of vectors do not remain infective for more than two days and Cymelarsan removes viable T. evansi from the blood of treated animals within a few hours of administration, sentinel horses should be introduced three days after removal or treatment of susceptible animals.

Surveillance

Where possible, all susceptible domesticated animals on IPs and DCPs should be examined weekly for clinical signs and undergo monthly serological testing (using antibody-ELISA) for three months, followed by quarterly testing for a further nine months. Where potentially susceptible native or feral animals are on or adjacent to the property, quarterly testing of susceptible domestic animals for longer than 12 months should be considered in the light of results from wild/feral animal surveys.

Where possible, all other susceptible domestic animals within the RA should be examined every four weeks and be tested serologically on a quarterly basis for one year. Where this is not possible, statistically valid serological surveys should be conducted quarterly.
Serological surveys should be designed to detect at least one seropositive animal with 95% confidence if the true prevalence of the disease is 5% of animals at risk. In view of deficiencies in the sensitivity and specificity of currently available serological tests, it is imperative that the sample size be calculated taking these deficiencies into account if serological screening is attempted. Sample sizes may be large as a result.

For example, if the antibody-ELISA is used for surveying a population of 1000 cattle, given that the sensitivity and specificity are 81% and 99.4% respectively, a sample size of 130 animals would be required. This level of precision (95% confidence of detecting a true prevalence of 5%) may not be achievable when using some of the other serological tests currently available.

Until more reliable serological tests become available, parasitological methods should always be used to confirm serological results. However, it will be necessary to increase the number of animals sampled or to sample on more than one occasion because the sensitivity of parasitological diagnosis is lower. For example, if PCR is used for surveying a population of 1000 cattle, given that estimates of the sensitivity and specificity are 30% and 100% respectively, a sample size of 194 animals would be required to detect a true prevalence of 5% with 95% confidence.

Where the number of animals at risk is sufficiently low and laboratory capacity exists, serious consideration should be given to conducting surveys using parasitological methods such as the mini-anion exchange centrifugation test, rather than serological tests.

Details of the published information on sensitivity and specificity of tests for *T. evansi* are given in Appendix 6.

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3 Calculated using Survey Toolbox (AusVet Pty Ltd, PO Box 3180, South Brisbane, Queensland 4101).
4 Calculated using Survey Toolbox.
Appendix 5  Procedures for vector monitoring and control

Monitoring

The population of tabanids may be monitored using Manitoba traps (Figure 1). The Manitoba trap uses a dark, heat-absorbing body to attract tabanids, which are then directed into a cone in which they are trapped. The addition of small amounts of carbon dioxide around the trap further increases its attractiveness. Traps should use black balls of 70 cm or more diameter and be baited with dry ice (to release CO₂). Trapping is most effective if carried out between 11 am and 2 pm, with the wind blowing towards the suspected breeding site at between 5 and 10 knots. Traps should be placed upwind from the breeding site so that the flies detect the emitted CO₂ and follow it to the trap. Tabanids also shelter in wooded areas near their breeding site, so traps should be located near such areas.

Figure 1  Manitoba trap
Control

Insecticidal fogging of tabanid breeding sites and the surrounding area within a radius of one kilometre, especially densely wooded areas, should be undertaken. However, tabanids are strong fliers; although few are caught in traps more than a few hundred metres from their breeding sites, the range of some overseas species can exceed two kilometres (Kettle 1984). Breeding places of the Australian species vary greatly. They include floating vegetation in swamps, mud of rivers and lagoons, damp soil, rotting vegetation, dry or beach sand, and rot holes in the trunks of casuarinas.

Control of stable flies should include insecticidal fogging of the area where animals are kept and daily collection and disposal of horse manure, where this is a practical option.

No data are available on the efficacy of different insecticides against tabanids, but aerial fogging with synthetic pyrethroids at commercially recommended concentrations should be considered as an initial strategy. The frequency of fogging and choice of insecticides should be determined from the results of monitoring activity as outlined above.


Appendix 6 Published information on sensitivity and specificity of tests for *T. evansi*

<table>
<thead>
<tr>
<th>Test*</th>
<th>Horses</th>
<th>Cattle</th>
<th>Buffalo</th>
<th>Camels</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Sen</td>
<td>Spec</td>
<td>Sen</td>
<td>Spec</td>
</tr>
<tr>
<td><strong>Serological tests</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibody-ELISA</td>
<td>–</td>
<td>95</td>
<td>89</td>
<td>92</td>
</tr>
<tr>
<td>IgG</td>
<td>95</td>
<td>81</td>
<td>99.6</td>
<td></td>
</tr>
<tr>
<td>IgM</td>
<td>30</td>
<td>98</td>
<td>88</td>
<td>55</td>
</tr>
<tr>
<td>Antigen-ELISA</td>
<td>63+</td>
<td>–</td>
<td>–</td>
<td>68+</td>
</tr>
<tr>
<td>CATT</td>
<td>–</td>
<td>100</td>
<td>72</td>
<td>98</td>
</tr>
<tr>
<td>Suratex</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td><strong>Parasitological tests</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Direct agglutination</td>
<td>94</td>
<td>97</td>
<td>–</td>
<td>–</td>
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<tr>
<td>HCT</td>
<td>71</td>
<td>–</td>
<td>15</td>
<td>–</td>
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<tr>
<td>RI</td>
<td>88</td>
<td>–</td>
<td>30</td>
<td>–</td>
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<tr>
<td>WBF</td>
<td>46</td>
<td>–</td>
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<td>–</td>
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<tr>
<td>MAECT</td>
<td>–</td>
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<tr>
<td><strong>Molecular tests</strong></td>
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<tr>
<td>PCR</td>
<td>–</td>
<td>–</td>
<td>~30</td>
<td>–</td>
</tr>
</tbody>
</table>

*a Definition of and information on each test are given below
Note: Only information from studies with designs considered capable of giving credible results have been included.

**Serological tests**

*Antibody-ELISA* — IgM and IgG antibody ELISAs using crude soluble parasite antigen. This is the most accurate of the available serological tests for the detection of recent infection with *T. evansi*. However, improved sensitivity is desirable and there may be scope to achieve this through choice of a better antigen and detection of specific isotypes that recognise the antigen.

*Antigen-ELISA* — Antigen-capture ELISA using *T. evansi*-specific monoclonal antibody. This test is not worth considering until a reliable source of the monoclonal antibody on which the test is built is secured.

*Card agglutination test (CATT) for trypanosomiasis* — available from Institute of Tropical Medicine, Laboratory of Serology, Antwerp, Belgium. This test is not recommended for routine use to detect new infection, as it does not become positive until the antigenic type it is designed to detect is expressed. This may not occur for many months after infection. In addition, conditions under which the test is performed must be carefully standardised in order to achieve repeatable results.

*Suratex* — commercial monoclonal antibody-based latex agglutination test using monoclonal antibodies from International Livestock Research Institute. There are few data to support claims for this test’s sensitivity or specificity, and quality control between batches is suspect. Limited testing of Australian bovine serum gave a specificity of 35% (Reid and Copeman 2003).
Parasitological tests

**Haematocrit centrifuge technique (HCT)** — the trypanosomes form a layer with the buffy coat (white blood cells) in a haematocrit tube and may be visualised at the interface of the buffy coat and plasma (Woo 1969).

**Rodent inoculation (RI)** (usually mice) — about 0.25–0.5 mL of blood (or buffy coat from 10 mL of blood) is inoculated intraperitoneally. Blood from the mouse is then checked (usually using tail-tip wet smears) every few days for the next 40–50 days for the presence of trypanosomes. Recent work has shown that this test will detect one *T. evansi* per 2 mL of blood (using buffy coat) (Reid et al 2001b).

Although blood should be inoculated into mice as soon as possible after collection, recent work has shown that the sensitivity of the RI test is not measurably diminished by 21 hours of refrigerated storage (in a lightproof container) of the blood under test (Reid et al 2001b). The use of RI is limited by the practicability of transporting and housing mice in the field.

There is no known difference in susceptibility to infection with *T. evansi* between different strains of laboratory mice. When tested experimentally, infection rates were the same in groups of BALB/c and CBA/CaH mice (Reid and Husein 1998).

**Wet blood film (WBF)** — a smear of a single drop of blood, examined microscopically for trypanosomes.

**Mini-anion exchange centrifugation test (MAECT)** — this test involves separation of trypanosomes from approximately 0.2 mL of blood (or buffy coat from 10 mL of blood) with a 2 mL DEAE-cellulose column followed by centrifugation of the eluate and examination of the sediment. Recent work has shown that this test will detect one *T. evansi* per 2 mL of blood (using buffy coat) (Reid et al 2001b).

**Molecular tests**

**Polymerase chain reaction (PCR)** — this test has been developed as part of the ACIAR-funded project AS1/200/009 and is suitable for use on fresh or frozen tissues, whole blood (0.25 mL) or blood dried on to Whatman FTA® cards. The sensitivity of this test is comparable to that of RI, and it can also be used to confirm the presence of *T. evansi* in tissue samples post mortem.
## Glossary

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<th>Term</th>
<th>Definition</th>
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<tr>
<td>Animal byproducts</td>
<td>Products of animal origin destined for industrial use, eg rawhides and skins, fur, wool, hair, feathers, hooves, bones, fertiliser.</td>
</tr>
<tr>
<td>Animal Health Committee</td>
<td>A committee comprising the CVOs of Australia and New Zealand, Australian state and territory CVOs, Animal Health Australia, and a CSIRO representative. The committee provides advice to PIMC on animal health matters, focusing on technical issues and regulatory policy (formerly called the Veterinary Committee). See also Primary Industries Ministerial Council (PIMC)</td>
</tr>
<tr>
<td>Animal products</td>
<td>Meat products and products of animal origin (eg eggs, milk) for human consumption or for use in animal feeding.</td>
</tr>
<tr>
<td>Australian Chief Veterinary Officer</td>
<td>The nominated senior veterinarian in the Australian Government Department of Agriculture, Fisheries and Forestry who manages international animal health commitments and the Australian Government’s response to an animal disease outbreak. See also Chief veterinary officer</td>
</tr>
<tr>
<td>AUSVETPLAN</td>
<td>A series of documents that describe the Australian response to exotic animal diseases, linking policy, strategies, implementation, coordination and counter-disaster plans.</td>
</tr>
<tr>
<td>Consultative Committee on Emergency Animal Diseases (CCEAD)</td>
<td>A committee of state and territory CVOs, representatives of CSIRO Livestock Industries and the relevant industries, and chaired by the Australian CVO. CCEAD convenes and consults when there is an animal disease emergency due to the introduction of an emergency animal disease of livestock, or other serious epizootic of Australian origin.</td>
</tr>
<tr>
<td>Control area</td>
<td>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).</td>
</tr>
<tr>
<td>Dangerous contact premises</td>
<td>Premises containing a dangerous contact animal(s) (see Appendix 1).</td>
</tr>
<tr>
<td>Decontamination</td>
<td>Includes all stages of cleaning and disinfection.</td>
</tr>
<tr>
<td>Disease Watch Hotline</td>
<td>24-hour freecall service for reporting suspected incidences of exotic diseases — 1800 675 888</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
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</tr>
<tr>
<td>ELISA</td>
<td>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.</td>
</tr>
<tr>
<td>Emergency animal disease</td>
<td>Includes exotic animal diseases and endemic diseases that warrant a national emergency response.</td>
</tr>
<tr>
<td>Emergency Animal Disease</td>
<td>Agreement between the Australian and state/territory governments and livestock industries on the management of emergency animal disease responses. Provisions include funding mechanisms, the use of appropriately trained personnel and existing standards such as AUSVETPLAN.</td>
</tr>
<tr>
<td>Exotic animal disease</td>
<td>Disease affecting animals (which may include man) and which do not presently occur in Australia.</td>
</tr>
<tr>
<td>Fomites</td>
<td>Inanimate objects (e.g. boots, clothing, equipment, vehicles, crates, packagings) that can carry the exotic agent and spread the disease through mechanical transmission.</td>
</tr>
<tr>
<td>Immunoglobulin</td>
<td>Antibody proteins</td>
</tr>
<tr>
<td>- IgG</td>
<td>The main form of immunoglobulin produced in response to an antigen. It is mainly found in body fluids.</td>
</tr>
<tr>
<td>- IgM</td>
<td>High molecular weight immunoglobulin; IgM antibodies are the first to be synthesised and released in response to a primary antigentic stimulation.</td>
</tr>
<tr>
<td>Incubation period</td>
<td>The period that elapses between the introduction of the pathogen into the animal and the occurrence of the first clinical signs of the disease.</td>
</tr>
<tr>
<td>Index property</td>
<td>The property on which the original case (index case) in a disease outbreak is identified to have occurred.</td>
</tr>
<tr>
<td>Infected premises</td>
<td>A premises on which an infected animal has been confirmed.</td>
</tr>
<tr>
<td>Local disease control centre</td>
<td>An emergency operations centre responsible for the command and control of field operations in a defined area.</td>
</tr>
<tr>
<td>Haemagglutination</td>
<td>Agglutination of red blood cells by a specific antibody or other substance.</td>
</tr>
<tr>
<td>Haemagglutination inhibition test</td>
<td>Test for the presence of an antibody</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Movement controls</td>
<td>Restrictions placed on movement of animals, people and things to prevent spread of disease.</td>
</tr>
<tr>
<td>National Management Group (NMG)</td>
<td>A group established to direct and coordinate an animal disease emergency. NMGs may include the chief executive officers of the Australian Government and state or territory governments where the emergency occurs, industry representatives, the Australian CVO (and chief medical officer, if applicable) and the chairman of Animal Health Australia.</td>
</tr>
</tbody>
</table>
  See Appendix 3 for further details. |
  See Appendix 3 for further details. |
| Primary Industries Ministerial Council (PIMC) | The council of Australian national, state and territory and New Zealand ministers of agriculture that sets Australian and New Zealand agricultural policy (formerly the Agriculture and Resource Management Council of Australia and New Zealand).  
  See also Animal Health Committee. |
<p>| 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. |
| 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, persons or things (see Appendix 1). |
| Risk enterprise                           | A horse or horse-related enterprise with a high potential for disease spread, e.g. a horse stud or artificial breeding centre.         |
| Sentinel animals                          | Animals used for the express purpose of detecting the presence of <em>Trypanosoma evansi</em>.                                                   |
| Stamping out                              | Eradication procedures based on quarantine and slaughter of all infected animals and animals exposed to infection.                      |</p>
<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tr>
<td>State/territory disease</td>
<td>The emergency operations centre that directs the disease control operations to be undertaken in the state/territory.</td>
</tr>
<tr>
<td>Surveillance</td>
<td>A systematic program of inspection, examination and testing of horses or things to determine the presence of <em>Taylorella equigenitalis</em>.</td>
</tr>
<tr>
<td>Susceptible species</td>
<td>Animals that can be infected with the disease</td>
</tr>
<tr>
<td>Suspect animal</td>
<td>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.</td>
</tr>
<tr>
<td>Suspect premises</td>
<td>Premises containing suspect animals that will be subject to surveillance <em>(see Appendix 1)</em>.</td>
</tr>
<tr>
<td>Tracing</td>
<td>The process of locating animals, persons or things that may be implicated in the spread of disease, so that appropriate action is taken.</td>
</tr>
<tr>
<td>Vector</td>
<td>A living organism (frequently an arthropod) that transmits an infectious agent from one host to another. <em>A biological</em> vector is one in which the infectious agent must develop or multiply before becoming infective to a recipient host. <em>A mechanical</em> vector is one that transmits an infectious agent from one host to another but is not essential to the life cycle of the agent.</td>
</tr>
<tr>
<td>Zoning</td>
<td>Dividing a country into defined infected and disease-free zones. A high level of movement control between zones will apply.</td>
</tr>
<tr>
<td>Zoonosis</td>
<td>Disease transmissible from animals to people.</td>
</tr>
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# Abbreviations

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<th>Description</th>
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<td>AAHL</td>
<td>CSIRO Australian Animal Health Laboratory, Geelong</td>
</tr>
<tr>
<td>CA</td>
<td>control area</td>
</tr>
<tr>
<td>CATT</td>
<td>card agglutination test</td>
</tr>
<tr>
<td>CCEAD</td>
<td>Consultative Committee on Emergency Animal Diseases</td>
</tr>
<tr>
<td>CSIRO</td>
<td>Commonwealth Scientific and Industrial Research Organisation</td>
</tr>
<tr>
<td>CVO</td>
<td>chief veterinary officer</td>
</tr>
<tr>
<td>DCP</td>
<td>dangerous contact premises</td>
</tr>
<tr>
<td>EDTA</td>
<td>ethylene diamine tetra-acetic acid (anticoagulant for blood)</td>
</tr>
<tr>
<td>ELISA</td>
<td>enzyme-linked immunosorbent assay</td>
</tr>
<tr>
<td>HCT</td>
<td>haematocrit centrifuge technique</td>
</tr>
<tr>
<td>IM</td>
<td>intramuscular</td>
</tr>
<tr>
<td>IP</td>
<td>infected premises</td>
</tr>
<tr>
<td>MAECT</td>
<td>mini-anion exchange centrifugation test</td>
</tr>
<tr>
<td>OIE</td>
<td>World Organisation for Animal Health</td>
</tr>
<tr>
<td></td>
<td>[Office International des Epizooties]</td>
</tr>
<tr>
<td>PCR</td>
<td>polymerase chain reaction</td>
</tr>
<tr>
<td>PNG</td>
<td>Papua New Guinea</td>
</tr>
<tr>
<td>QBC</td>
<td>quantitative buffy coat test</td>
</tr>
<tr>
<td>RA</td>
<td>restricted area</td>
</tr>
<tr>
<td>RI</td>
<td>rodent inoculation</td>
</tr>
<tr>
<td>SP</td>
<td>suspect premises</td>
</tr>
<tr>
<td>VAT</td>
<td>variable antigenic type</td>
</tr>
<tr>
<td>VSG</td>
<td>variable surface-coat glycoprotein</td>
</tr>
<tr>
<td>WBF</td>
<td>wet blood film</td>
</tr>
</tbody>
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References cited in text


and a PCR for *Trypanosoma evansi* diagnosis in experimentally infected water buffaloes. *Veterinary Parasitology* 97:23–33.


Nantulya VM (1990). Trypanosomiasis in domestic animals: the problems of
diagnosis. *Revue Scientifique et Technique des Office International des Epizooties*

by horse flies and deer flies (Diptera: Tabanidae). *Journal of Medical Entomology*

Prevalence of bovine hameo-parasites in Aceh Province of Northern Sumatra:

trypanosomiasis caused by *Trypanosoma evansi* on the island of Madura,

Payne RC, Waltner-Toews D, Djauhari D and Jones TW (1991a). *Trypanosoma evansi*
infection in swamp buffalo imported into Central Java. *Preventive Veterinary

and Luckins AG (1991b). *Trypanosoma evansi* infection in cattle, buffalo and horses

Payne RC, Sukanto IP, Partoutomo S and Jones TW (1994a). Efficacy of


ProMED-mail, [http://www.promedmail.org](http://www.promedmail.org), 7 Jul: 19990707.1125

ProMED-mail (2004). Trypanosomiasis – India (03). ProMED-mail,
[http://www.promedmail.org](http://www.promedmail.org), 16 Dec: 20041216.3323

ProMED-mail (2005). Trypanosomiasis – India (West Bengal). ProMED-mail,
[http://www.promedmail.org](http://www.promedmail.org), 20 Jan: 20050120.0197


Queiroz A, Cabello P and Jansen A (2000). Biological and biochemical
characterisation of isolates of *Trypanosoma evansi* from Pantanal of Matogrosso

Raina AK, Kumar R, Rajora VS, Sridhar, Singh RP (1985). Oral transmission of

Raisinghani PM, Bhatia JS, Vijas UK, Arya PL and Lodha KR (1980). Pathology of
experimental surra in camels (*Camelus dromedarius*). *Indian Journal of Animal
Sciences* 50:966–969.


**Further reading**

**General**


**Distribution, susceptible species**


Diagnosis

General


**Parasitological tests**


Olaho-Mukani W, Nyan’ao JMN and Ouma JO (1996). Comparison of Suratex, parasite detection and antigen-ELISA for the evaluation of treatment efficacy


**Serological tests (immunoassays)**


Molecular tests


Other tests


Culture of trypanosomes


Pathology


**Immunity**


**Epidemiology**


**Transmission/vectors**


**Treatment**


**Video/training resources**

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