

# EXOTIC Surra and Australian wildlife *Fact sheet*

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## Introductory statement

Surra (*Trypanosoma evansi*) is a blood borne protozoan parasitic disease transmitted by biting flies. It has long been recognized as a major threat to Australian livestock industries and wildlife. If surra were to enter Australia, it would prove very difficult to control and eradicate because of the widespread availability of feral and native animal reservoir hosts, and the ubiquity of the tabanid and perhaps muscid fly vectors of the disease (Thompson 2001). Surra is found in eastern Indonesian and Timor Leste, and there is a risk it will reach West Papua (Irian Jaya), through the movement of people and animals. If surra did reach West Papua it could then spread to Papua New Guinea (PNG) and Australia. Control of the disease in livestock in Australia could cost the cattle, sheep and horse industries millions of dollars. Surra is thus considered an exotic disease of concern for Australia (Reid 2002; DAWR 2016a).

## Aetiology

The aetiological agent is *Trypanosoma evansi*, belonging to the subgenus *Trypanozoon*. March flies (*Diptera: Tabanidae*), particularly species of the genus *Tabanus*, and stable flies (*Diptera: Muscidae*) belonging to the genus *Stomoxys* serve as mechanical vectors of infection (Hoare 1972), as well as vampire bats in South and Central America.

## Natural hosts

*T. evansi* is pathogenic in a range of domesticated and some wild animal species, predominantly horses, mules, donkeys, camels, llamas, deer, cattle, buffalo, cats and dogs. Asymptomatic, mild or chronic disease has been seen in sheep, goats, elephants and pigs. The primary host species varies with the geographic region, for example in southeast Asia infection is seen mainly in horses, cattle and buffalo.

## World distribution

Surra is endemic in China, the Indian subcontinent, northern Africa, the Middle East, South America, the Philippines, Bulgaria, parts of the former U.S.S.R. and parts of Indonesia (OIE 2012). The disease has become endemic throughout southeast Asia in recent time (Luckins 1988; DAWR 2016b).

## Occurrences in Australia

Surra was diagnosed in a consignment of imported camels at Port Hedland, Western Australia, in 1907 (Cleland 1907). These were destroyed and there has been no further evidence of the disease in camels, or any other species, in Australia. While currently absent from Australia and PNG, it is considered an exotic disease of concern for this country (Desquesnes et al. 2013; Thompson et al. 2014; DAWR 2016b).

The disease is nationally notifiable in Australia.

## Epidemiology

The severity of disease can vary with the strain of trypanosome and with host factors, including stress and concurrent infections. In some outbreaks, morbidity up to 50-70% with comparable mortality may occur. Mortality is generally high in horses, camels, dogs and cats and lower in cattle, buffalo, deer and other species. Mortality rates are low in cattle and buffalo raised in endemic areas, but movement of animals from a non-endemic area (Australia) into an endemic area (Indonesia) has resulted in high mortality rates. In *Equidae*, surra is often fatal within two weeks to four months.

The incubation period of the disease is generally between 5 and 60 days although longer periods of 3 months have been recorded.

Tabanid flies (also called march flies or horse flies), especially the genus *Tabanus*, are considered the primary vectors of surra (Nieschulz, reviewed by Krinsky 1976). At least 27 different species of *Tabanus* have been shown experimentally to transmit *T. evansi* (Luckins 1998), including *T. ceylonicus*, common to many countries in Southeast Asia and Australasia, and active during the wet and dry seasons in Cape York (Van Hennekeler 2007). There is also evidence that all *Tabanus* spp. are likely vectors of surra (Dieleman 1986; Luckins 1998). Other tabanid genera could transmit surra in Australia.

The method of transmission of surra by biting flies is direct, i.e. it occurs when flies take a meal on a surra-infected animal, with trypanosomes in the peripheral blood, and immediately afterwards feed on a healthy animal. Species of *Tabanus* and *Stomoxys* are interrupted feeders; when dislodged from a host they actively and persistently seek the nearest available host to continue feeding, a behaviour which renders them efficient mechanical vectors of diseases. One *Tabanus* fly can infect three animals successively (Dieleman 1986). Research in Australia has shown that species such as *T. pallipennis* can potentially bite up to nine individual animals in quick succession before completing a single blood meal, resulting in a high capacity to transmit surra (Muzari et al. 2010b). Iatrogenic transmission, for example as a result of herd vaccination, is a possible route of transmission which has not been studied (Reid 2002).

The most likely entry point for surra into Australia is with infected hosts being moved across the Torres Strait.

Agile wallabies (*Macropus agilis*) and dusky pademelons (*Thylogale brunii*) were found to be highly susceptible to experimental infection with a buffalo strain of *T. evansi* (Reid et al. 2001b). The agile wallaby is the most common species of macropodid in southern PNG and northern Australia. Studies have

demonstrated that macropods are a key host type for at least six of the most common tabanid species in North Qld (Muzari et al. 2010a). In addition, the preponderance of excellent reservoir hosts in the form of feral pigs and rusa deer in both PNG and Australia compounds the risk situation (Reid et al. 1999).

Australia has more than 250 species of *Tabanidae* including 25 species of *Tabanus* (42 species in PNG) with many in the north (Mackerras 1974; Van Hennekeler 2007), corresponding to a high geographical risk of surra incursion associated with the proximity to West Papua, Indonesia and Papua New Guinea. In addition, species of *Tabanus* were present for an average of 11 months of the year. Consequently, there is a confluence of risk factors in the most northern part of Cape York, which increases the risk of incursion and establishment of surra in this region (Mackerras 1974; Van Hennekeler 2007; Van Hennekeler et al. 2008). However, extension of the range of surra mediated by the movement of infected tabanids, especially over open water or open areas with little vegetation is unlikely. A much more important pathway for the spread of surra is through the movement of infected animals (hosts) such as deer or dogs, which are then available for local vectors to feed on and cause local transmission to naïve hosts. Infected deer swimming from PNG/ Irian Jaya to the northern Torres Strait islands or infected dogs moved illegally by boat are likely to be the most high-risk pathways into Australia if surra gained entry into West Papua.

## Clinical signs

The general clinical signs of *T. evansi* infection are not sufficiently pathognomonic for diagnosis (OIE 2012). In experimental infection of agile wallabies and pademelons, there was acute onset of signs, including anorexia, depression, ataxia, coma and death (Reid et al. 2001b). The disease in other susceptible animals is manifested by pyrexia, directly associated with parasitaemia, together with a progressive anaemia, loss of condition and lassitude. Recurrent episodes of fever and parasitaemia. Oedema, particularly of the lower parts of the body, urticarial plaques and petechial haemorrhages of the serous membranes are often observed. Although some deaths may occur in cattle and buffalo during the first six months, most animals recover and become carriers. Abortions have been reported in buffaloes and camels. A wide-based stance, loss of balance and hind limb proprioceptive deficits may also occur particularly in *Equidae*. In dogs, nervous signs may resemble rabies. In pigs, infections are usually asymptomatic or mild and in goats and deer they are chronic. There are indications that the disease causes immunodeficiencies (Desquesnes et al. 2013).

## Diagnosis

Diagnosis is by demonstration of *T. evansi* in blood, lymph nodes, skin exudates, liver, lungs or kidney. Trypanosomes tissues can be detected only when there is a high parasitaemia. Methods of parasite concentration and inoculation of laboratory animals have also been used. A variety of antibody detection tests have been introduced for laboratory and field use but few have been critically evaluated and standardised, and even fewer are used routinely in endemic areas (Reid et al. 2001a; Reid and Copeman 2002; Reid 2002; Reid and Copeman 2003). Among those that are used regularly in the laboratory are immunoenzyme assays, card agglutination tests and latex agglutination tests. For field use both card agglutination tests (CATT) for *T. evansi* and latex can be applied, yet an individual test format (pen side test) is currently unavailable. For declaring a disease-free status, serial testing (ELISA followed by re-testing of suspect samples by CATT) is recommended (OIE 2012). Cross reactions with other species of pathogenic trypanosomes may occur.

For serology, the CATT is the only commercially available test and the one routinely used at AAHL. PCR is useful for confirming the presence of infection but not as a first-line screening test. The haematocrit

centrifugation technique is still considered the best test but it is not easy to perform without training. A simple wet blood smear from a sick animal has a high likelihood of demonstrating infection because experimental work has shown that wallabies maintain very high parasitaemias until death (Reid and Copeman 2000; Reid et al. 2001a; Reid and Copeman 2002; Reid 2002; Reid and Copeman 2003). Real-time PCR has been used to detect surra and may be more sensitive than CATT (Taylor et al. 2008).

## Pathology

A cell-mediated immune response without tissue destruction was the primary lesion observed in experimentally infected wallabies, consistent with that in other host species (Damayanti et al. 1994; Reid et al. 2001b), there was no cellular destruction in wallabies.

Gross pathological changes have been reported in experimentally infected wallabies. Changes varied between animals and included pericarditis, serous atrophy of fat, splenomegaly and ulcerative gastritis and enteritis (Reid et al. 2001b). Consistent histological changes included lymphocyte and macrophage infiltration in the connective tissue of all organs examined. All animals had diffuse interstitial pneumonitis with thickening of the alveolar wall.

## Differential diagnosis

The differential diagnosis in horses includes African horse sickness, equine viral enteritis, equine infectious anaemia and chronic parasitism. In dogs, rabies should also be considered.

## Laboratory diagnostic specimens

Thick and thin blood films made, air-dried and fixed. Similar films made from needle biopsies of prescapular or precrural lymph nodes and any skin exudates. Post-mortem, impression smears collected from lungs, liver and kidney. Ten ml whole blood into heparin or EDTA with antibiotics added and 25 ml whole blood taken for serology; transported cold. The collection of samples can be simplified by using filter paper blood spots for later use in the ELISA, while for the CATT whole blood can be substituted for serum.

## Laboratory procedures

Because the parasites may be difficult to find in microscopic examination, detection may be improved almost tenfold by the haematocrit tube centrifugation technique or a modified miniature anion-exchange centrifugation technique (Woo 1969; Reid et al. 2001a). Despite a number of serological tests being used for surra none are sufficiently sensitive to prove that animals are free from infection and of the available antigen-detection assays none are reliable for detecting *T. evansi* with confidence (Reid 2002).

## Treatment

Drugs previously used to treat *T. evansi* infection in southeast Asia included suramin, diminazene aceturate and quinapyramine and more recently melarsamine hydrochloride (Reid 2002). In Australia, melarsamine is commercially available for treatment of canine heartworm and has a high efficacy against *T. evansi* (Kumar et al. 2016). A dose of 0.5mg/kg is likely to be safe and curative in most species.

## Prevention and control

Importation and movement of potentially infected animals is the highest established risk pathway and must be restricted and carefully monitored. Australia maintains a high level of vigilance through the ongoing activities of biosecurity services and the Northern Australia Quarantine Strategy.

## Surveillance and management

Effective surveillance for *T. evansi* is constrained by a lack of sensitive diagnostic tests and information on the distribution of the organism in Indonesia, particularly West Papua, where there is serological evidence but no parasitological confirmation of its presence (Reid 2002). Australia must maintain a high level of vigilance to reduce the likelihood of *T. evansi* entering and becoming established in this country, especially in the light of climate change which will affect both the geographic range and breeding season of tabanid flies, especially in more temperate regions.

*T. evansi* is considered a threat to Australia's unique biodiversity, our mammalian fauna having evolved in the absence of the disease and given evidence of its fatal effect on agile wallabies and pademelons (Reid et al. 2001b; Thompson et al. 2014).

Effective surveillance for the potential vectors of surra can be achieved using the Nzi trap (Mihok 2002) baited with octenol. For some Australian tabanid species there is a correlation between catches from this trap and tabanid burden on animals in the same area. In studies on spatial distribution of Australian tabanids, fly abundance was much higher in savannah woodlands than open grassland areas (Muzari 2010). Therefore, surveillance for the presence of tabanids is best conducted in woodland habitats.

## Research

Further development of molecular tests for *T. evansi*, such as PCR, to improve diagnostic accuracy in detecting the organism in blood and tissues are warranted given their greater advantage in test sensitivity and specificity over parasitological and serological tests. Additionally, they can be applied directly to clinical samples.

## Human health implications

Surra is not generally considered to be a disease risk for humans however there has been one confirmed case of clinical disease in an immunodeficient human in India who developed a range of clinical signs and responded to anti-trypanosome medication (Powar et al. 2006). The human case was subsequently linked to a deficit in the Apo-1 gene of the person infected (Lun et al. 2009).

## Conclusions

Surra is currently absent from Australia, PNG and West Papua. However, its likely introduction into West Papua (Irian Jaya); the numerous reservoir hosts available in both PNG and Australia, especially agile wallabies, rusa deer and pigs; the ubiquity of its tabanid vectors, especially members of the genus *Tabanus*; and the proximity of Australia to PNG combine to create a high risk of its entry to this country, either by incursion of infected animals or movement of infected tabanid flies. If surra were to enter Australia it is improbable that the disease could be controlled given the spectrum of potential feral and native reservoir

hosts. Control of the disease in livestock alone would cost the cattle sheep and horse industries millions of dollars.

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## To provide feedback on this fact sheet

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