**Importance**

Surra is one of the most important diseases of animals in tropical and semitropical regions. This protozoal disease can affect most domesticated mammals and some wild species. When surra is first introduced into a region, the morbidity and mortality rates can be high. In the early 1900s, an outbreak in Mauritius killed almost all of the Equidae on the island. More recently, severe outbreaks have been reported in the Philippines, Indonesia and Vietnam. In addition to illness and deaths, surra causes economic losses from decreased productivity in working animals, reduced weight gain, decreased milk yield, reproductive losses and the cost of treatment.

**Etiology**

Surra is caused by infection with the protozoal parasite *Trypanosoma evansi*. This organism belongs to the subgenus *Trypanozoon* and the Salivarian section of the genus *Trypanosoma*.

**Species Affected**

*T. evansi* has a wide host range. This organism can cause disease in most domesticated mammals and some wild or zoo animals; clinical cases have been reported in horses, mules, donkeys, camels, deer, cattle, water buffalo, sheep, goats, pigs, cats and dogs, as well as captive elephants, Himalayan black bears (*Selenarctos thibetanus*), tigers and jaguars. Experimentally infected wallabies and bandicoot rats also become ill. In South America, infections with *T. evansi* have been reported in wide variety of wildlife including capybara (*Hydrochaeris hydrochaeris*), coati (*Nasua nasua*), peccaries (*Tayassu spp.*), feral pigs, armadillos (*Euphractus sp.*), vampire bats (*Desmodus rotundus*), insectivorous and fruit bats (*Artibeus sp.*, *Platyrhinus sp.*, *Carollia sp.*, *Myotis sp.* and *Noctilio sp.*), marsupials (*Gracilinanus sp.* and *Monodelphis sp.*) and small mammals in the genera *Thrichomys*, *Clyomys*, *Oecomys* and *Dasyprocta*. Young pigeons can be infected by subcutaneous and intraperitoneal injection.

**Geographic Distribution**

Surra is enzootic in Africa, the Middle East, many parts of Asia, Central and South America, and the Canary Islands of Spain. In 2006, an outbreak was reported among camels in France.

**Transmission**

Unlike some other trypanosomes, *T. evansi* does not require a biological vector. This organism, which can be found in blood and tissues, is transmitted mechanically by biting flies. *Tabanus* spp. appear to be the most important mechanical vectors, but transmission by members of the genera *Haematopota*, *Chrysops*, *Lyperosia*, *Stomoxys*, *Musca* and *Atylotus* has also been reported. Ticks have been proposed as possible mechanical vectors, and iatrogenic transmission can probably occur on needles and surgical instruments. Vampire bats have also been implicated in transmission in South and Central America. Carnivores can also become infected after feeding on infected tissues. Oral transmission might occur during fighting. Transmission in milk and by the venereal route might also be possible.

**Incubation Period**

In the Equidae, the incubation period varies from 5 to 60 days.

**Clinical Signs**

Surra can be an acute, subacute or chronic disease. Some animals die rapidly; in other cases, clinical signs may persist for months. Animals can also carry *T. evansi* subclinically. Common clinical signs in most species include fever (which is intermittent in chronic cases), weight loss or wasting, lethargy, signs of anemia, enlargement of the lymph nodes and dependent edema. Urticaria, jaundice and petechial hemorrhages of the mucous membranes may also be seen. Facial and laryngeal edema and conjunctivitis have been reported in dogs, and dyspnea,
coughing and diarrhea occurred in some experimentally infected goats. Neurological signs have been reported in horses, dogs, cattle, deer, water buffaloes and captive Himalayan black bears. Horses in South America frequently develop ataxia, with gradually progressive paresis of the hindquarters accompanied by muscle atrophy. Other neurological signs such as head tilt, circling, hyperexcitability, blindness, proprioceptive deficits and paddling movements have also been seen. Infertility, abortions and/or stillbirths have been documented in buffalo, camels and horses. Testicular lesions in camels and experimentally infected goats suggest that, in some species, male fertility might also be impaired. *T. evansi* causes leukopenia, which may result in immunosuppression.

**Post Mortem Lesions**

The gross lesions tend to be nonspecific, and may include wasting or emaciation of the carcass, subcutaneous edema, signs of anemia, enlargement of the spleen and lymph nodes, and petechiae on some internal organs. Muscle atrophy may be noted, particularly in the hindquarters. Hydrothorax and ascites are sometimes seen. The lungs may also be affected; congestion, consolidation, edema, emphysema, hemorrhages and pneumonia have been reported. Cardiac lesions including hydropericardium, pericarditis and evidence of cardiomyopathy or myocarditis occur in some animals.

In some horses with neurological signs, the cerebral hemispheres may be swollen and the gyri flattened. There may be severe edema and malacia, with the white matter becoming yellow, gelatinous and friable. Subpial hemorrhages may also be present.

**Morbidity and Mortality**

The severity of the disease can vary with the strain of *T. evansi* and with host factors including stress, concurrent infections and general health. Severe outbreaks can occur when surra is introduced into disease-free areas or susceptible animals are moved into endemic regions. Morbidity rates as high as 50-70%, with comparable mortality, can be seen in a herd. In the Philippines, where severe epizootics occurred after the introduction of *T. evansi*, approximately 20% of the horses, water buffalo and cattle became ill, with an overall mortality rate of 5%. During one outbreak in Brazil in 2005, at least 100 horses died. Of 23 horses examined, all nine animals with neurological signs and 11 of 14 horses without CNS signs died. The three surviving horses were in poor condition.

Reproductive success may also be affected. One study reported that the calving rate in high-risk areas for surra was half that of a low risk area. Although this difference may have been caused by general debilitation, it might also have been the result of abortions and stillbirths, and/or decreased fertility in infected bulls. The number and severity of surra outbreaks appears to be increasing.

**Diagnosis**

**Clinical**

Surra should be considered when the clinical signs include anemia, weight loss and dependent edema. Neurological signs, especially hindlimb weakness, may be seen in some animals. Sudanese herders identify infected camels by changes in the color and odor of their urine, caused by the excretion of α-keto acids from trypanosome catabolism.

**Differential diagnosis**

Other diseases that cause edema, anemia, wasting and/or neurological signs should be considered. In horses, the differential diagnosis includes African horse sickness, equine viral arteritis, equine infectious anemia, equine babesiosis and chronic parasitism. In horses with encephalitis, equine herpesvirus type 1 myeloencephalopathy, Eastern, Western or Venezuelan equine encephalitis, equine protozoal myeloencephalitis, West Nile virus infection and rabies should also be considered.

**Laboratory tests**

A presumptive diagnosis can be made if organisms consistent with *T. evansi* are detected in the blood, lymph nodes or other tissues by direct examination. Parasites may be found in wet blood films and thick or thin stained blood smears. Wet unstained blood films are examined by light microscopy, using 200x power to detect the motile trypanosomes. Thin smears fixed in methyl alcohol can be stained with Giemsa, and unfixed thin smears may be stained with May–Grünwald stain followed by Giemsa. Thick smears are air dried and stained with Giemsa. Other stains such as Field’s stain or Diff Quick® can also be used. Thick or thin films should be examined by light microscopy at high magnification. Thick films have the advantage of being able to detect parasites in low numbers; however, the morphology of the parasite is difficult to determine. *T. evansi* can be difficult to find in the blood, especially in mild or subclinical cases. It cannot be definitively identified by direct examination, as it resembles other trypanosomes.

Detection can be improved with parasite concentration techniques including mini anion-exchange chromatography, hemolysis methods that use sodium dodecyl sulphate (SDS) to destroy the erythrocytes (i.e., wet blood film clarification or hemolysis centrifugation) and by hematocrit centrifugation or the dark-ground/phase-contrast buffy coat technique. The latter two methods rely on the concentration of trypanosomes near the buffy coat after centrifugation.

Techniques to detect *T. evansi* antigens, including latex agglutination, enzyme-linked immunosorbent assays (ELISAs) and immunohistochemistry, have been published. In some horses with neurological signs, parasites were found in the brain with immuno-histochemistry, even when they were not visible in hematoxylin and eosin stained sections. This technique has also been used to detect *T.
evansi in the brains of cattle, hog deer and buffalo. Recombinant DNA probes may be used to specifically identify *T. evansi* in blood or tissues, but are not in routine use. Polymerase chain reaction (PCR) assays, including a species-specific technique, have also been published.

Serology can aid diagnosis. Commonly used serological tests include ELISAs, card agglutination and latex agglutination. Other assays have also been used. All serological tests have not been validated or standardized, and cross-reactions can occur with other trypanosomes. Some serological tests may not detect certain variants of the organism (e.g., type B in Kenya). An ELISA that detects an invariant surface glycoprotein of *T. evansi* has been published.

Animal inoculation studies in rats or mice may occasionally be used to diagnose surra. These tests are very sensitive and can detect low levels of parasites, but they are also time consuming.

**Samples to collect**

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

Blood samples for smears and wet films should be collected from live animals during a febrile period. Because *T. evansi* tends to occur in the deep blood vessels when parasitemia is low, samples should be taken from deeper blood vessels with a syringe, as well as from peripheral vessels such as the small veins in the ear or tail. Anticoagulated fresh blood is used in all concentration methods. For some techniques, samples may be collected into heparinized capillary tubes. Concentration techniques that use hemolysis can also be done on meat. Samples sent to the laboratory should be kept cold. Parasitemia is often intermittent in chronically infected animals, and repeated sampling of the blood may be necessary.

Lymph node biopsies can also be used to detect the parasites in live animals; samples are often taken from the prescapular or precrural lymph nodes. Slides are made similarly to blood. Trypanosomes may be found in various tissues at necropsy. They have also been detected in the cerebrospinal fluid (CSF) of a horse with neurological signs.

Serum should be collected for serology.

**Recommended actions if surra is suspected**

**Notification of authorities**

Surra should be reported to state or federal authorities immediately upon diagnosis or suspicion of the disease.

Federal: Area Veterinarians in Charge (AVIC): www.aphis.usda.gov/animal_health/area_offices/


**Control**

The introduction of surra often results in the disease becoming enzootic; however, the parasite was prevented from becoming established in the U.S. in 1906 and in Australia in 1907. If the outbreak is detected early, *T. evansi* might be eradicated by quarantines, movement controls and the isolation or slaughter of infected animals. Trypanosomes cannot survive for long periods outside the host, and disappear quickly from the carcass after death. Controlling arthropod vectors is important in preventing new infections. Flies are most infective during the first few minutes after feeding on an infected host; after eight hours, they no longer transmit the parasites. Healthy animals can be confined to stables during the day, as tabanids preferentially feed in sunlight.

In endemic areas, surra is usually controlled by treating infected animals with antiparasitic drugs. The choice of drugs may be limited in some areas. Drug resistance can be seen, and some drugs may have toxic effects. No vaccines are available.

**Public Health**

Only one human infection with *T. evansi* has been documented. This case occurred in a 45-year-old Indian farmer who had a genetic defect in apolipoprotein L1, a protein that confers resistance to nonzoonotic animal trypanosomes. The symptoms included intermittent fever, chills, sweating and neurological signs. Antiparasitic treatment was successful. A study conducted in the farmer’s village did not detect trypanosomes in the blood of any other humans, although some people were seropositive.

**Internet Resources**

http://www.spc.int/rabs/

The Merck Veterinary Manual
http://www.merckvetmanual.com/mvm/index.jsp

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/
References


Herrera HM, Abreu UG, Keuroghlian A, Freitas TP, Jansen AM. The role played by sympatric collared peccary (Tayassu tajacu), white-lipped peccary (Tayassu pecari), and feral pig (Sus scrofa) as maintenance hosts for Trypanosoma evansi and Trypanosoma cruzi in a sylvatic area of Brazil. Parasitol Res. 2008;103(3):619-24.


Laha R, Sasmal NK. Detection of Trypanosoma evansi infection in clinically ill cattle, buffaloes and horses using various diagnostic tests. Epidemiol Infect. 2009 Apr 15:1-3. [Epub ahead of print]


*Link defunct as of 2009*