TRYPANOSOMA EVANSI INFECTIONS (INCLUDING SURRA)

AETIOLOGY

Classification of the causative agent

Order Kinetoplastida; family Trypanosomatidae; Genus *Trypanosoma*; Subgenus Trypanozoon, Species *Trypanosoma evansi. T. equinum* in South America is a dyskinetoplastic variant of *T. evansi* and not a separate species.

Transmitted mechanically from infected blood of animals, and is not capable of cyclical development in tsetse *Glossina spp*. Morphologically indistinguishable from *T. brucei.*

Resistance to physical and chemical action

Chemicals/Disinfectants:	Controlling arthropod vectors and preventing access to host species is important in preventing new infections. Disinfection does not prevent spread of disease (blood-borne parasite). One minute exposure to ultraviolet light prevents infection.
Survival:	Trypanosomes only survive short periods outside the host. *T. evansi* disappears quickly from the carcass after death. Flies no longer transmit the parasites after 8 hours.

EPIDEMIOLOGY

T. evansi has a wide host range. In some countries incidence of surra increases significantly furing the rainy season when biting fly populations have greatly increased. Surra affects mainly camels and horses but buffaloes and cattle are also affected. Other species that develop severe disease include donkeys, mules, deer, llamas, dogs, cats, cattle and buffalo. Sheep, goats, pigs and elephants may occasional develop mild or chronic disease. Camel raising in Africa and buffalo production in Asia are severely affected.

Hosts

- Pathogenic in most domesticated animals and some wild animals
- Domesticated animals: Horses, mules, donkeys, cattle, buffalo, camels (dromedary and Bactrian), llamas, pigs, sheep, goats, dogs and cats
- Most important single cause of morbidity and mortality in camels
- Wild animals: deer, capybara (reservoir host) and other species
- New world camelids in South America are experimentally susceptible but natural disease has not been reported despite presence in cattle and horses
- Reservoir hosts to camels and horses: cattle, buffalo, capybara, and vampire bat
- Rats and mice are highly susceptible as experimental hosts for detecting subclinical (nonpatent) infections

Transmission

- Direct life cycle with no intermediate host
- Agent is transmitted from animal to animal mechanically by hematophagous flies, including *Tabanus spp*. and *Musca* spp.
    - Also *Lyperosia*, *Stomoxys* and *Atylotus* genera. Tabanids (horse flies) are the most significant vectors
- Vampire bats in South and Central America are hosts, reservoirs and vectors of *T. evansi*; they transmit *T. evansi* mechanically in their saliva, and may develop high parasitaemia which may kill the bat. Recovered bats serve as carriers
- Carnivores may become infected after ingesting infected meat
- Transmission in milk and during coitus has been documented

Sources of infection

- Blood from infected animals; occasionally meat and milk
- *T. evansi* frequently localises extravascularly in tissues including the central nervous system

Occurrence

T. evansi has a wide distribution in Asia, North Africa (extending into tsetse areas with *T. brucei* infections) and Central and South America.

The main host species varies with the geographical region. Horses are most often affected in South America; horses, mules, buffalo, and deer in China (People's Rep. of); horses, cattle, and buffalo in South-East Asia; and camels in the Middle East and Africa.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE *World Animal Health Information Database* (WAHID) Interface [http://www.oie.int/wahis/public.php?page=home] or refer to the latest issues of the *World Animal Health* and the OIE *Bulletin*.

DIAGNOSIS

Clinical diagnosis

Morbidity and Mortality: Camels living in northeastern Africa may have infection rates of 20–70%. Case fatality rate in untreated horses and camels is nearly 100%. Surra in cattle and buffalo tends to be chronic with a much lower CFR. Animals subjected to stress, like malnutrition, pregnancy, and physical labour, are more susceptible to disease.

The disease is often rapidly fatal in camels and horses, but may also be fatal in buffalo, cattle, llama and dogs, however these host species may develop mild or subclinical infections. Nervous signs are common in horses. Dogs may also have nervous signs that resemble rabies. Infections in deer are usually chronic with oedema, anaemia, emaciation and nervous signs.

Clinical signs are suggestive but diagnosis must be confirmed by a laboratory.

The disease in susceptible animals, including cattle, buffalo, camels (dromedary and bactrian), horses, pigs, sheep and goats, is manifested by:

- Incubation period in horses, mules, donkeys and camels varies from 5–60 days
- Fever, directly associated with parasitaemia – recurrent episodes occur during the course of disease
- Progressive anaemia, weight loss and icterus
- Progressive weakness and lethargy
- Oedematous swellings of the lower parts of the body: legs, briskets and abdomen (gravity dependent)
- Urticarial plaques in the skin
- Petechial haemorrhages of the serous membranes (eyelids, nostrils and anus)
- Abortions reported in buffaloes and camels
- Immunodeficiency
- Death may occur in 2 weeks to 4 months, chronic infections may last up to 2 years

Lesions

Post-mortem lesions are nonspecific and may include:

- emaciation of the carcass
- anaemia
- petechial haemorrhages on some internal organs
- hydrothorax and ascites
- enlarged lymph nodes and spleen

Differential diagnosis

- Horses: African horse sickness, equine viral arteritis, equine infectious anaemia, chronic parasitism, dourine
- Camels: tsetse-transmitted trypanosomosis, anthrax, chronic parasitism
- Cattle: babesiosis, anaplasmosis, theileriosis (East Coast Fever), haemorrhagic septicaemia, anthrax, chronic parasitism, malnutrition
- Dogs: rabies if neurological signs

Laboratory diagnosis

T. evansi is not known to have a zoonotic potential. It should be handled in the laboratory in accordance with the principles outlined in Chapter 1.1.2 *Biosafety and biosecurity in the veterinary microbiology laboratory and animal facilities.*

Samples

Parasite identification

- Dried thick and thin blood smears during the febrile phase stained with Giemsa
- Dried thick and thin smears from needle biopsies of prescapular and precrural lymph node aspirates
- Smears from any skin exudates
- Anticoagulated blood in EDTA and/or heparin (10 ml)
- Cerebrospinal fluid
- Impression smears of lungs, liver, and kidney at post mortem

Serological tests

- Serum samples (10–20 ml of serum)

Procedures

Identification of the agent

- Direct identification of the parasite in stained thick or thin blood films or wet mounts.
    - Diagnostic sensitivity is increased significantly by concentrating the parasites in the buffy coat layer of a heparinised microhaematocrit tube
    - The buffy coat is then examined directly at low power (Woo's method) or in a wet preparation with phase-contrast or dark-ground microscopy (Murray's method)
    - Sensitivity is also increased when used at the herd versus individual animal level
    - Parasitaemias are highly variable during the course of infection: high during early infection, low during chronic infection, and almost nil in healthy carriers
    - Mini-anion exchange centrifugation technique: simplified method for detecting low parasitaemia by separating salivarian trypanosomes from host red blood cells
- Direct identification of the parasite in lymph node biopsy smears from fine needle aspirates
- Animal inoculation to reveal subclinical infections: rats or mice; should be limited as far as possible and only used if fully justified
- Recombinant DNA probes: detect trypanosomes in infected blood or tissue; experimental
- Polymerase chain reaction (PCR)
    - More sensitive test than direct identification and similar sensitivity to mouse inoculation
    - False negatives can occur when parasitaemias are very low, which occurs frequently with chronic infections
- Antigen detection: yet reached a satisfactory level to be recommended for routine diagnosis

Serological tests

- Indirect immunofluorescent antibody test: useful when screening s small number of samples
- Antibody detection ELISA: very useful for large-scale surveys
    - ELISA using variable surface glycoproteins from a *T. evansi* RoTat 1.2 clone successfully differentiated *T. evansi* from *T. brucei*. Protocols are available for equines, camelidae and water buffaloes
- Card agglutination test: also makes use of *T. evansi* RoTat 1.2 clone
- Latex agglutination test: currently under evaluation

- Immune trypanolysis test: detects specific 'trypanolytic' antibodies directed against a given parasitic strain able to induce trypanolysis in the presence of complement. It is performed with *T. evansi* variable antigen type RoTat 1.2

There are OIE Reference Laboratories for Surra (see OIE Web site: http://www.oie.int/en/our-scientific-expertise/reference-laboratories/list-of-laboratories/).

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 2.1.17 *Trypanosoma evansi infections* (including surra) the latest edition of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques".

PREVENTION AND CONTROL

Surra is one of the most important diseases of camels. Camel raising in Africa and buffalo production in Asia are severely affected by surra. As in tsetse-transmitted trypanosomosis, losses are due to reduced productivity, mortality and cost of treatment. Control of surra can be difficult as there is no vector specificity and a wide range of hosts.

Sanitary prophylaxis

- Control measures are aimed at the host rather than vector, unlike Nagana
- Control measures include detection and treatment of infected animals, prophylactic treatment of susceptible animals, and protection of animals from biting flies and vampire bats

Medical prophylaxis

- Drugs such as suramin, prothridium and isometamidium chloride (as a prophylactic) and diminazene aceturate (curative) can be used although drug resistance has been reported
- For camels melarsomine (cymelarsan) is very effective (curative) against *T. evansi*
- So far this drug is only registered for use in camels
- No vaccines are available nor likely in the near future because of the ability of trypanosomes to rapidly change their surface glycoproteins to avoid the immune response

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated April 2013.