TRYPANOSOMOSIS (TSETSE-TRANSMITTED)

AETIOLOGY

Classification of the causative agent

Order Kinetoplastida; family Trypanosomatidae; Genus *Trypanosoma*; Subgenus Nannomonas (*T. congolense*), Subgenus Duttonella (*T. vivax*), and Subgenus Trypanozoon (*T. brucei* ssp).

Flagellated protozoan parasites that live in the blood, lymph and various tissues of their vertebrate hosts: *Trypanosoma congolense, T. vivax,* and to a lesser extent *T. brucei brucei*. *T. uniforme* and *T. simiae* are other, less common tsetse-transmitted species.

T. congolense and *T. vivax* are mainly intravascular parasites while *T. brucei* has an affinity for tissues.

Several types of *T. congolense* can be distinguished by molecular biology; the most common and pathogenic one in cattle is the type "savannah" (large variation in pathogenicity within the savannah subgroup), the other ones (type 'forest' and 'Kilifi' or Kenya coast) are less pathogenic and have different host affinity.

Mixed trypanosome infections with two or three species are common.

Resistance to physical and chemical action

Disinfectants/chemicals:	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).
Survival:	These agents can only survive in blood, body fluids and tissues of animal hosts and within tsetse flies. Mechanically transmitted *T. vivax* cannot survive long outside the host. Agents disappear within a few hours after death of the vertebrate host.

EPIDEMIOLOGY

Tsetse flies infest 10 million square kilometres and affect 37 countries, mostly in Africa, where it is known as 'Nagana'. It is the most economically important livestock disease of Africa, especially of cattle.

Hosts

- Wild animals: natural hosts
  - At least 30 species, including greater kudu (*Tragelaphus strepsiceros*), warthog (*Phacohoerus aethiopicus*), bushbuck (*Tragelaphus scriptus*), bush pig (*Potamochoerus porcus*), African buffalo (*Syncerus caffer*), African elephant (*Loxodonta africana*), white rhinoceros (*Ceratotherium simum*), black rhinoceros (*Diceros bicornis*), wild Equidae, lion (*Panthera leo*) and leopard (*Panthera pardus*).
  - Usually show no clinical signs since host and parasite are in equilibrium
  - Enormous reservoir of trypanosomes
- Tsetse fly (*Glossina*): biological vector
  - 23 species in sub-Saharan Africa between latitudes 14°N and 29°S are competent, but primarily *G. morsitans*, *G. palpalis* and *G. fusca*
  - Grouped according to preferred habitat: savannah, riverine and forest
  - Remain infected by trypanosomes for life
  - Trypanosome life cycle involves cyclical development in the tsetse fly, taking up to 3 or more weeks depending on trypanosome species and ambient temperature
- Domestic animals: incidental hosts; cattle most important economically
  - *T. congolense*: cattle, pigs, goats, sheep, horses, and dogs
  - *T. vivax*: cattle, horse, sheep, and goats

    - *T. brucei brucei*: cattle, horses, dogs, cats, camels, sheep, goats, and pigs
- Trypanotolerant breeds
    - West African indigenous taurine breeds: N'Dama, Baoule, Muturu, Laguna, Somba and Dahomey
    - East African zebu breeds: Orma Boran and Maasai zebu
    - Indigenous breeds of small ruminants: West African dwarf sheep and goats, and East African goats
- Reservoirs: many wild animals, trypanotolerant animals, chronically infected animals, tsetse flies
- Laboratory rodents, especially rats and mice
    - For revealing subpatent infections of *T. brucei brucei* (and *T. evansi*) infections, but does not work for some *T. congolense* strains and *T. vivax* rarely infects them.
- Humans: Sleeping Sickness caused by *T. brucei gambiense* and *T. brucei rhodesiense*;
    - The animal trypanosomes very rarely cause human infection, but they do share animal reservoirs (wild and domestic) and tsetse vectors

Transmission

- Cyclical transmission: trypanosomes are transmitted through the bite of an infected tsetse fly. Tsetse flies get the infection when feeding on an infected animal; after implementation of the parasitic cycle in the fly (15–21 days) it becomes infective and may remain infective for the rest of its life. Transmission occurs in the early stage of the blood feeding, when the fly inject some saliva before sucking the blood of its host.
- Mechanical transmission: Biting flies, especially tabanids and stomoxes, but possibly other biting insects (including tsetse flies) are the mechanical vectors of *T. vivax*. Mechanical transmission can occur when interrupted feeding is re-started on a new host; thus it is efficient inside a group of animals but has little chance to occur at distance. Trypanosomosis due to *T. vivax* has thus spread to some areas of Africa free or cleared of tsetse, and also in Central America and South America.
- Others: vertical transmission can occur intra-utero and during partum.
- For *T. brucei* per-orale transmission can even occur after the birth, when contaminant blood or other fluids can be ingested by the calf. Perorale transmission is also common for carnivore when eating fresh infected prey.

Sources of infection

- Blood, lymph and other fluids of infected animals

Occurrence

African animal trypanosomosis occurs where the tsetse fly vector exists in Africa, between latitude 15°N and 29°S. *T. vivax* can also be transmitted mechanically by biting flies, and thus is also found in parts of Africa free or cleared of tsetse, and parts of Central and South America.

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*.

For specific information for Latin American trypanosomose can be found in [http://www.oie.int/boutique/index.php?page=ficprod&id_produit=46&fichrech=1&lang=en]

DIAGNOSIS

Incubation period is generally 8-20 days. *T. congolense* usually becomes apparent in 4–24 days, *T. vivax* in 4–40 days, and *T. brucei brucei* highly variable.

Clinical diagnosis

Disease is classically acute or chronic, and is affected by poor nutrition, concurrent diseases, and other stressors. Trypanosomosis in cattle is usually chronic – some may slowly recover but usually relapse when stressed. The most important clinical sign is nonregenerative anaemia, and the most common reason animals are unable to function normally. The major clinical signs are:

- intermittent fever
- anaemia
- oedema
- lacrimation
- enlarged lymph nodes
- abortion
- decreased fertility
- loss of appetite, body condition and productivity
- early death in acute forms
- emaciation and eventual death in chronic forms often after digestive and/or nervous signs

When tsetse challenge is high, morbidity is usually also high. All three species of trypanosomes will eventually cause death in their hosts unless treated.

Lesions

Post-mortem lesions are nonspecific and are usually related to anaemia and the prolonged antigen-antibody response:

- emaciation and serous atrophy of fat
- enlarged lymph nodes, liver and spleen
- excessive fluid in the body cavities and subcutaneous oedema
- petechial haemorrhages
- lymphoid tissue may be atrophic in the terminal phases as the animal is too debilitated to mount an immune response, and severe myocarditis is common
- *T. brucei brucei* tends to invade tissues to cause inflammation and/or degeneration of multiple tissues, in addition to anaemia

Differential diagnosis

Acute trypanosomosis with fever:

- Babesiosis
- Anaplasmosis
- Theileriosis (East Coast Fever)
- Haemorrhagic septicaemia
- Anthrax

Chronic trypanosomosis with anaemia and emaciation:

- Helminthosis
- Malnutrition
- Other haemoparasitoses

Laboratory diagnosis

Samples

Parasite identification

- Plain blood or anticoagulated blood in EDTA and/or heparin (10 ml)
- Needle biopsies of prescapular and precrural lymph node aspirates for *T. vivax* and *T. brucei brucei*
- Cerebrospinal fluid for *T. brucei*

Serological tests

- Serum samples (10–20 ml)

Procedures

Identification of the agent

- Direct examination of fresh blood or buffy coat between slide and cover slide can sometimes lead to species identification, based on the epidemiological situation and on typical size, shape and movements of the parasites but fixation and staining is required for a reliable species identification
  Direct identification of the parasite in wet or dry-stained thick or thin blood films.
    - 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). Buffy coat can also be smeared and stained.
    - 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. Widely used in human medicine but not suitable for large scale screening of animal samples.
    - In-vitro cultivation: based on the cultivation of procyclic forms of trypanosomes, species differentiation is not possible; success has been irregular over many years

- Polymerase chain reaction (PCR)
    - Highly specific and more sensitive test than direct identification
    - Can identify parasites at subgenus, species or subspecies level
    - False negatives can occur when parasitaemias are very low (<1 trypanosome per ml), which occurs frequently with chronic infections, or when primers do not recognise all isolates of a particular trypanosome species

Serological tests

- Antibody detection ELISA: very useful for large-scale surveys
- Indirect fluorescent antibody test

Both tests have high sensitivity and genus specificity, but their species specificity is generally low. At present they can only be used for presumptive diagnosis of trypanosomosis. Antibodies persist on average 3-4 months after curative treatment or self-cure, but may last up to 13 months.

For more detailed information regarding laboratory diagnostic methodologies and vaccines, please refer to Chapter 2.4.18 Trypanosomosis (tsetse-transmitted) in the latest edition of the OIE *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques".

PREVENTION AND CONTROL

Trypanosomosis is a major constraint to ruminant livestock production in many areas of Africa.

Sanitary prophylaxis

- Land spray of insecticide, bush clearing and elimination of game animals destroy valuable animal resources and also leads to soil erosion; they have been abandoned.
- Control and eradication of tsetse vector
  - Insecticides: synthetic pyrethroids applied directly on the animal as a spray or pour-on offers great promise; insecticide foot bath are also under evaluation;
  - Sterile male technique: potentially valuable since females mate only once in a lifetime but production facilities are expensive and can only be apply at the end of the eradication campaign, when the density of remaining flies is very low;
  - Pheromone baited tsetse traps that attract and catch tsetse flies: simple, cheap, non-polluting, and readily accepted by local communities
- Good husbandry of animals at risk and avoid contact with tsetse flies as much as possible
- Introduction and development (selective cross breeding) of trypanotolerant animals. Cattle breeds, like the N'Dama and West African Shorthorn, have been in West Africa for centuries and have developed innate resistance to trypanosomes. They are infected by tsetse flies but do not show clinical disease. However, these breeds have not been readily accepted because they are small in size and low in milk producing. Cross breeding is however a common practice.

Medical prophylaxis

- Drugs such as isometamidium chloride and quinapyramine sulphate and chloride can be used as prophylactic during transhumance or high seasonal parasitic pressure;
- Curative drugs are diminazene aceturate and quinapyramine methylsulfate which can be used as curative and sanative. Chemoresistance may occur and care must be taken due to the presence of fake drugs on some markets.
- No vaccines are available nor likely in the near future because of the ability of trypanosomes to rapidly change variable surface glycoproteins (VSG) in their coats to avoid an effective Immune response (antigenic variation). This also leads to establishment of prolonged infections with intermittent parasitaemias. There are estimated to be about 1,000 VSGs, in the trypanosomal coat, which switch genetically as antibodies are produced by the host.

For more information, please refer to the technical notes edited by CIRDES on (1) strategic control of trypanosomoses [http://www.cirdes.org/IMG/pdf/F_3_anglais.pdf]; (2) trypanocide use [http://www.cirdes.org/IMG/pdf/F_8_anglais.pdf] and (3) insecticide treatments: [http://www.cirdes.org/IMG/pdf/F_2_anglais.pdf]

REFERENCES AND OTHER INFORMATION

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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.