TRYPANOSOMA EVANSI INFECTIONS (SURRA)
Definition.

Surra, caused by *Trypanosoma evansi*, is one of the most important diseases of animals in tropical and semitropical regions. This trypanosoma was discovered in India more than a hundred years ago by Evans (1880). While surra is particularly serious in equids and camels, infections and clinical cases have been reported in most domesticated mammals and some wild species. *T. evansi* is transmitted mechanically by various tabanids and other flies, and it can readily become endemic when introduced into a new area. The morbidity and mortality rates in a population with no immunity can be high. 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.

Aetiology.

Surra is caused by the protozoal parasite *Trypanosoma evansi*. Order Kinetoplastida; family Trypanosomatidae; this organism belongs to the subgenus *Trypanozoon* and the Salivarian section of the genus *Trypanosoma*. Two genetic types of *T. evansi*, type A and type B, have been recognized. Most isolates worldwide belong to type A. Type B, which is not recognized by some diagnostic tests, has only been detected in parts of Africa as of 2015. Whether *T. evansi* should be considered a distinct species, separate from *T. brucei*, is controversial.
**Trypanosoma evansi** - Source en.wikipedia.org

**Epidemiology.**

Surra is enzootic in Africa, the Middle East, many parts of Asia, and Central and South America. *T. evansi* has a wide host range. In some countries incidence of surra increases significantly during the season when biting fly populations have greatly increased. Equids, Bactrian camels (*Camelus bactrianus*) and dromedaries (*Camelus dromedarius*) are highly susceptible to disease. Camel also act as reservoir host.

It is an arthropod-borne disease; mechanical transmission by biting insects is the most important mode of transmission in camels. *T. evansi* does not require a biological vector. This organism, which can be found in blood and tissues, is transmitted mechanically by biting insects. Members of the deerfly and horsefly family, Tabanidae (e.g., the genera *Tabanus*, *Atylotus*, *Chrysops*, *Lyperosia*, and *Haematopota*) and flies in the genus *Stomoxys* spp. are thought to be the most important vectors.
Additional means of transmission include iatrogenic spread on contaminated needles or surgical instruments, and the ingestion of infected tissues. Trypanosomes cannot survive for long periods outside the host, and disappear relatively quickly from the carcass after death. Flies no longer transmit the parasites after 8 hours.

*T. evansi* is not currently considered to be zoonotic, a few cases have been reported in humans. It is uncertain whether all of these infections occurred in people who are unusually susceptible or the disease is underdiagnosed.

**Clinical Signs**
Surra can be an acute, subacute or chronic disease, with the severity of the clinical signs differing between individual animals, as well as between species. Surra can attack camels at any age, even foetuses. There is a particularly high incidence of infection in juvenile camels shortly after weaning. Numerous environmental and host factors influence the course of the disease, such as other infections, nutritional status, age, pregnancy, previous exposure or immunosuppression by other diseases, and stress.
In a typical case, the camel loses weight, develops a drooping hump, is unable to walk long distances, and may or may not develop oedema of the feet, brisket, underbelly and eyelids; the coat becomes rough. In the initial attack of fever there may be lacrimation, shivering, reduced appetite and mild diarrhoea. The animal always shows progressive anaemia and fluctuating body temperature with initial peaks of fever up to 41 °C. Later, the appetite is relatively unimpaired and the temperature may become normal or slightly elevated. The mucous membranes are pale and the packed cell volume (PCV) drops to below 25% (v/v), sometimes as low as 10% (v/v). Petechial haemorrhages of the serous membranes (eyelids, nostrils and anus).

The herders may notice a characteristic odour of the camel's urine and identify infected animals by this sign alone. The odour of the urine may be due to ketone bodies, which were found to be elevated in trypanosome-infected camels. Abortion in all stages of pregnancy is common. Death of the newborn calf ensues within two weeks. Lactating females produce less milk, and cases of blindness and central nervous lesions have been reported to be sequelae of trypanosomiasis. The herd eventually reaches an endemic disease situation. Some animals may carry trypanosomes for years whereas others never do. Within such a group there are all forms and stages of surra from new infections to subclinical and chronic conditions. Incubation period in camels varies from 5–60 days. A lethal outcome is relatively rare, but mortality may reach 20%. Post-mortem examination reveals no absolutely typical signs, but some degree of anaemia is often visible. Skeleton and heart muscles are pale, and there are signs of dehydration, pericardial effusion, enlarged lymph nodes and splenomegaly.

Diagnosis.

Identification of the agent.
The general clinical signs of T. evansi infection are not sufficiently pathognomonic for diagnosis. Laboratory methods for detecting the parasite are required. The organism may be difficult to find, especially in mild or subclinical cases, and parasitemia is often intermittent in chronically infected animals. In early infection or acute cases, when the parasitaemia is high, examination of wet blood films, stained Blood smears or lymph node materials might reveal the trypanosomes. In more chronic cases, or more generally when the parasitaemia is low, the examination of thick blood smears, as well as methods of parasite concentration and the inoculation of laboratory rodents are required. In apparently healthy carriers (animals without clinical signs), parasites are rarely observed and mouse inoculation gives the best results. Several primer pairs targeting the subgenus (Trypanozoon) or the species-specific (T. evansi) parasitic DNA sequences are available for diagnosis by polymerase chain reaction (PCR). PCR is more sensitive than parasitological examination, but it may give false-negative results when the
parasitaemia is very low; in these cases, suspicion of potential carriers can only be confirmed by serological examination.

a) Direct microscopic examination
Blood sampling
Trypanosoma evansi is a parasite of the blood and tissues. As for other trypanosomes, it is recommended that blood for diagnosis be obtained from peripheral ear or tail vein, even if the jugular vein is most often preferred for practical reasons. However it should be realised that less than 50% of infected animals may be identified by examination of blood. Peripheral blood is obtained by puncturing a small vein in the ear or tail. Deeper samples are taken from a larger vein by syringe. Cleanse an area of the ear margin or tip of the tail with alcohol and, when dry, puncture a vein with a suitable instrument (lancet, needle). Ensure that instruments are sterilised or use disposable instruments to avoid iatrogenic transmission of the infection by residual blood.

i) Wet blood films.
Place a small drop of blood (2–3 μl) on to a clean glass slide and place over it a cover-slip to spread the blood as a monolayer of cells. Examine by light microscopy (200 ×) to detect any motile trypanosomes. Improved visualisation can be obtained with dark-ground or hase-contrast microscopy (200–400 ×). The sensitivity of this method is low, approximately 10 trypanosomes per μl, which is frequent in early or acute infections only.

ii) Stained thick smears.
Place a large drop of blood (10 μl) on the centre of a microscope slide and spread with a toothpick or the corner of another slide so that an area of approximately 1.0–1.25 cm in diameter is covered. Air-dry for 1 hour or longer. Place the slide in a horizontal position, stain the unfixed smear with Giemsa’s Stain (one drop of commercial Giemsa + 1 ml of phosphate-buffered saline, pH 7.2), for 25 minutes. After washing and drying, examine the smears by light microscopy at a magnification of 500× with oil immersion. The advantage of the thick smear technique is that it concentrates the drop of blood into a small area, and thus less time is required to detect the parasites, which are more visible owing to the haemolysis of the unfixed red cells. The disadvantage is that the trypanosomes may be damaged in the process, and the method is therefore not suited for species identification in case of mixed infections.

iii) Stained thin smears.
Place a small drop of blood (3–5 μl) at one end of a clean microscope slide and draw out a thin film in the usual way. Air-dry briefly and fix in methyl alcohol for 1 minute and allow to dry. Stain the smears in Giemsa (one drop Giemsa + 1 ml PBS, pH 7.2) for 25 minutes. Pour off stain and wash the slide in tap water and dry. Nowadays, fast stains are most often used, which allow fixation and staining within a few seconds. Slides are then washed in tap water and dried. Examine at a magnification of 400–1000× with oil immersion. This technique permits detailed morphological studies and identification of the Trypanosoma, but it is of a very low sensitivity (it can detect parasitaemia >500,000 trypanosomes/ml of blood).
Lymph node biopsies or oedema fluid. Samples are usually obtained from the prescapular or precrural (subiliac) lymph nodes. Select a suitable node by palpation and cleanse the site with alcohol. Puncture the node with a suitable gauge needle, and draw lymph node material into a syringe attached to the needle. Expel lymph on to a slide, cover with a cover-slip and examine as for the fresh blood preparations. Fixed thin or thick smears can also be stored for later examination. Similar examination can be done by collection of oedema fluid.

**Control.**

The control of a vectorial disease is classically divided into two sections: pathogen control and vector control. There are also various alternative means of controlling transmission, which can be combined as “means to prevent the infection.” In the case of surra, in the absence of a vaccine against trypanosomes (due to a large repertoire of variable surface antigens), disease control is principally based on the use of trypanocides and preventive management methods to protect animals from infection.

**Chemical control of parasite.**

As a blood parasite, *T. evansi* can be killed by injecting various trypanocidal drugs, providing that concentration of the chemical in the serum is lethal for the parasite. However, treatment might fail in the case of extravascular invasion or chemoresistance. Trypanocides can be divided into two categories. The “curative drugs” are used for treatment and have a short-term effect. They can kill the parasites, although they do not always eliminate 100% of them. The “curative/preventive drugs” are used for chemoprophylaxis. They not only kill parasites but also prevent any new infection or new circulation of parasites, due to the remanence of a sustainable curative dose in the serum of animals under chemoprophylaxis.

Melarsomine dihydrochloride is the latest trypanocide was first available for commercial use in 1992. It is used to control surra in camels via deep intramuscular injection at a dose rate of 0.25 mg/kg bw. Which can be increased up to 0.5 mg/kg bw if fully curative (sterilising) treatment is required.

Quinapyramine belongs to the group of aminoquinidine derivatives. Quinapyramine methyl-sulphate can be used to treat the infection by subcutaneous injection at a dose of 5 mg/kg bw. A more effective combination of quinapyramine sulphate and quinapyramine chloride can be used as a curative/preventive drug against *T. evansi* in camels, administered by subcutaneous injection at a dose of 8 mg/kg bw. Local tolerance is sometimes low. However, the drug is quite efficient and the chemoprophylactic effect can last up to 4 months.

**Vector Control.**

In endemic areas, it is difficult to control the biting flies that transmit *T. evansi*; however, some animals may be protected with insecticides/repellents, traps, insect screens/netting in stables.
and/or other controls. Flies are most infective soon after feeding on an infected host (e.g., in the first half hour), and the highest probability of transmission is to nearby hosts. Because tabanids are persistent feeders and do not usually leave one animal to bite another more than 50 meters away.

In camels kept close to the tsetse belt, some cases of *T. brucei* have been recorded. *T. congolense* infections are fatal to camels. Therefore, camels should not be allowed to enter the tsetse belt unless they are permanently protected with the use of chemicals. Consequently, *T. evansi* is not found in the tsetse belt.

**Disinfection**

There is limited need for disinfectants, due to the fragility of trypanosomes in the environment, and no studies appear to have examined disinfectant susceptibility specifically for *T. evansi*. The closely related organism *T. brucei* can be inactivated by various agents including 0.05% sodium hypochlorite, 70% ethanol, 2% TriGene™, 0.1% hand soap, 2% formaldehyde and 0.05% glutaraldehyde. The temperature reported to kill 100% of trypomastigotes is 50°C.

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