## *Trypanosoma evansi*

An acute form of the disease, which is generally fatal unless treated, occurs in horses, donkeys, mules, cattle, buffalo, deer, camels, llamas, dogs, and cats. and is transmitted by horse-flies, and also by the vampire bat, *Desmodus rotundus*, in South-America.

### Location in the host

These parasites are parasites of the blood stream and tissue fluids.

### Life cycle and Transmission

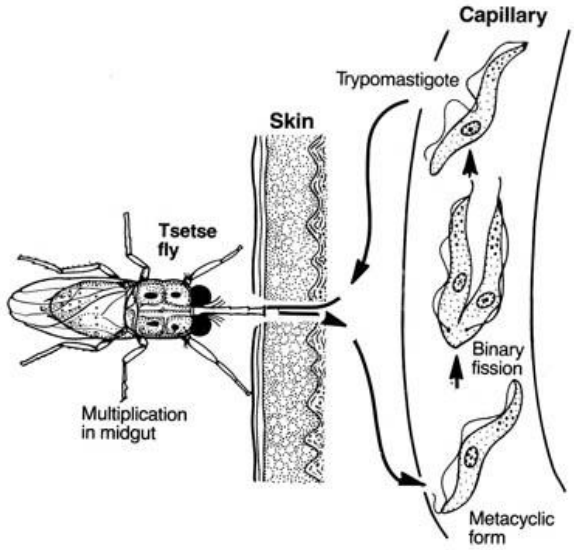
*Trypanosoma evansi* is spread mechanically by an arthropod vector. The vector varies geographically. All blood sucking flies can transmit the disease, although the 'horse' fly, *Tabanus*, and 'stable' fly, *Stomoxys* are the most common vectors in South-East Asia. The *Tse-Tse fly* is the most seen in different countries in Africa .

*Trypanosoma* species have a limited survival time in the mouth parts of a vector, so therefore time between feedings have to be short.

*Trypanosoma evansi* isn’t only spread by mechanical transmission, but also during nursing, copulation or ingestion of infected tissues by carnivores.

Unlike other trypanosomes species, whose life cycle needs an intermediate host before reaching the adult state, *Trypanosoma evansi* doesn’t undergo development in an intermediate host. *Trypanosoma*

*evansi* multiplies in a mammalian host by the process of longitudinal binary fission in the trypomastigote stage




**Pathogenesis**

*Trypanosoma evansi* which causes the disease known as trypanosomosis (Surra) . The degree of pathogenicity depends on what species the host is, the virulence of the *Trypanosoma evansi* strain, and the dose received by the host. Many species such as dogs, horses and rats, have been shown to have immunological reactions to the infection, such as anemia due to decrease in erythrocytes and hemoglobin. Although not fully understood at this point in time, some theories believe that the erythrocytes could acquire trypaonosomal antigen, resulting in a negative immunological reaction.

**Clinical signs**

Animals that have been infected show loss of appetite, weight loss, anaemia, odema, fever, salivations, lacrimation, and abortion. The

proteases that are released during infection of T.evansi might degrade the host tissue proteins and are a huge force in the pathogenesis. That is why scientists are looking at immune targeting of these proteases to protect the infected host.

**Diagnosis**

Some conventional parasitological techniques (CPT) such as wet blood film, and stained blood smears are used because so far, the best identifier is looking at the blood of the potentially infected host. Other tissues can be looked at, but the gold standard is identifying trypanosomes in the blood.

## *Histomonas meleagridis*

**Agent and host range:**

- *Histomonas meleagridis* is a cosmopolitan parasite affecting gallinaceous fowl (turkeys, chickens, pheasant, quail, & grouse)

Turkeys - highly susceptible to infection & most infected turkeys die (Blackhead disease) Chickens - easily infected but usually a milder form of disease

**Site of infection** - cecum & liver

**Morphology**

- various trophozoite stages (flagellate & amoeba forms, i.e. pleomorphic) 5-30 um - no cysts

**Life Cycle**

- divides by asexual, binary fission - direct transmission - fecal-oral route possible but considered rare - cloacal drinking (turkeys & SPF chickens)

transmission possible - indirect transmission is the most common route by the cecal nematode, *Heterakis gallinarum* - trophozoites are ingested by the nematode & then trophs penetrate the tissues of the worm & finally become incorporated into developing nematode eggs - birds become infected by ingesting *Heterakis* eggs in soil or by eating earthworms (paratenic host of the nematode) - by either route, *Histomonas* liberated into the intestine will penetrate the cecal wall & passes to the liver via the portal circulation.

**Pathogenesis**

- disease results when *Histomonas* penetrates the cecal wall & invades the liver via the blood stream - cecal lesions - cecum becomes thickened (edematous) & lumen is filled with yellow caseous smelly exudate - liver lesions - circular depressed (bulls eye) yellow-green to grey areas of necrosis (1-2 cm) which may coalesce to involve the entire liver - appear by approximately 10 days post infection .

**Clinical signs**

- hunched appearance, droopy wings & tail, ruffled feathers - anorexia & emaciation, weakness & depression

- head may (or may not) turn black or cyanotic (due to deficient oxygenation of the blood) - foul smelling, brilliant yellow sulfur-coloured diarrhea - mortality high in young turkeys (50-100%) - low mortality in chickens, but up to 30% has been reported in young birds

**Diagnosis**

- brilliant yellow (sulfur) feces combined with the cecal & liver lesions

- Histomonas may also be demonstrated histologically.

Lect.3

1- ……. Is a parasites of the blood stream and tissue fluids.

2- *Trypanosoma evansi* is spread mechanically by…….

3- define surra disease?

4- the final hosts of *T.evansi* are …….

5- *Histomonas meleagridis* affecting ……

6- *Histomonas meleagridis* sites of infection are ……

7- why the head of birds becomes black with *H. meleagridis*?

8- enumerate the clinical signs with histomoniasis?