Paper of the week:

Steverding, D. 2008. The history of African trypanosomiasis. Parasites and Vectors. 2008: 1:3. http://www.parasitesandvectors.com/content/1/1/3

Phylum Protozoa or Protista

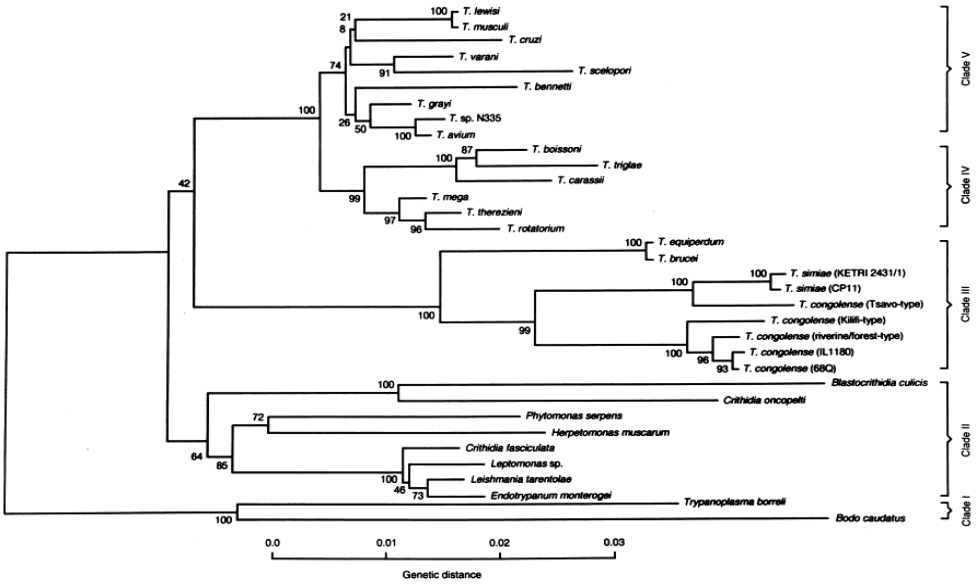
Class Kinetoplastida – have kinetoplast – a large darkly staining body in the mitochondrion.

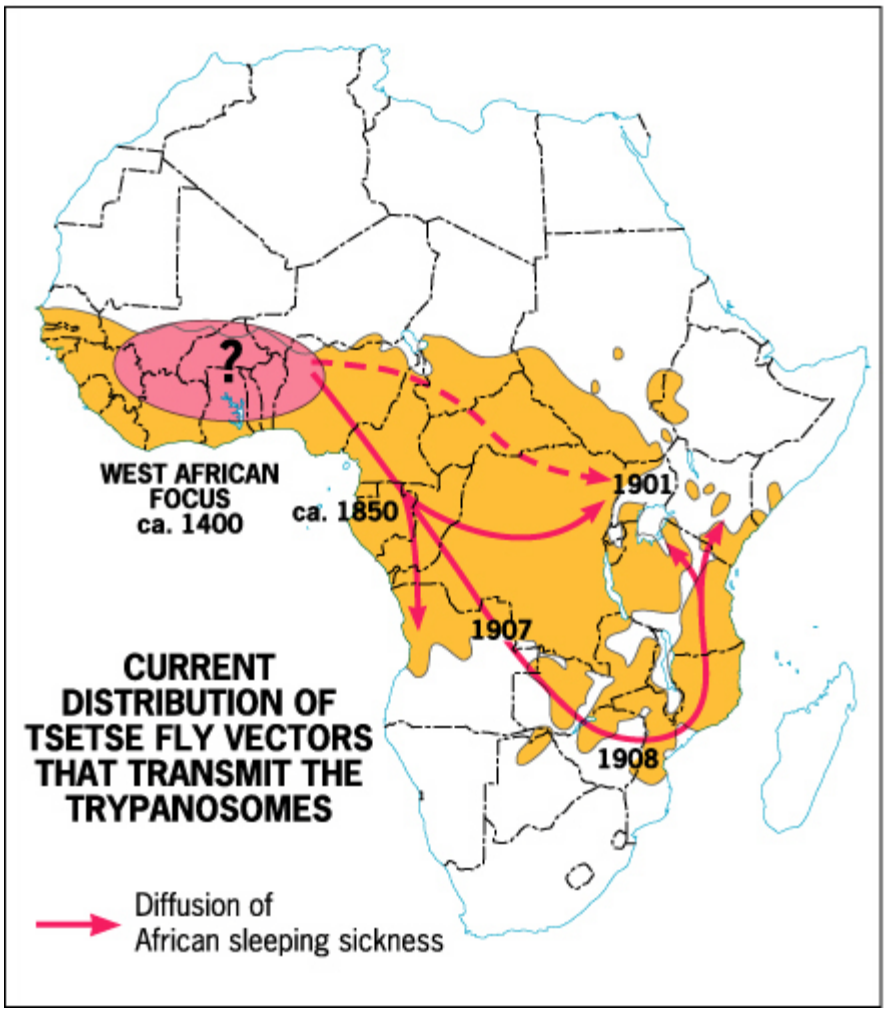
History of tsetse fly and sleeping sickness in Africa. Also see Book by McKelvey, J.J. Jr. 1973. Man against Tsetse. Cornell Univ. Press, Ithaca and London. 306 pp.

Several species occur in mammals in Africa.

- **SALIVARIAN TRYPANOSOMES** – Those that develop in the anterior end of the arthropod host and are transmitted to the vertebrate host via a bite. – African forms of *Trypanosoma*
- **STEROCORARIAN TRYPANOSOMES** – Those that develop in the posterior end of the arthropod host and are transmitted to the vertebrate host via a fecal deposit of infective stages. – S. Amer.

Phylogenetic diversification of the trypanosomes. See:





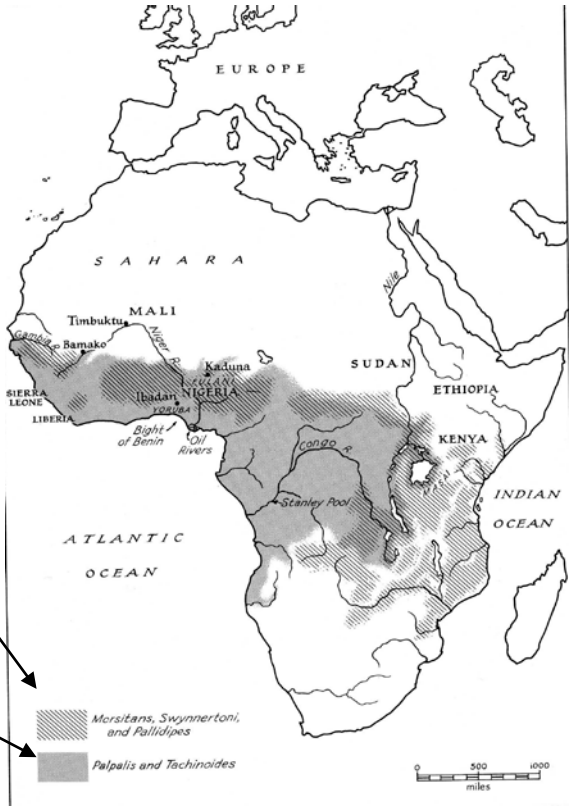
The African forms are the Salivarian types.

Distribution: Africa – in area where Tsetse flies occur = *Glossina* spp. 8 million sq km. Between 14° North latitude and 20° South latitude.

Map 1 – from explanation by McKelvey 1973.

Map 2 – from McKelvey, 1973.

Distribution of *Glossina mositans*, *G. swynnertoni*, *G. pallidipes* and *G. palpalis* and *G. tachinoides*



Human trypanosomiasis – African Sleeping Sickness – caused by:

1. *Trypanosoma brucei gambiense* – most common in west and central Africa. Chronic form of the disease called sleeping sickness.
2. *Trypanosoma brucei rhodesiense* – most common in east and southern Africa. Acute form of the disease.

Vector: ***Glossina*** – NOT AN INTERMEDIATE HOST AS THERE IS LITTLE EVIDENCE FOR SEXUAL REPRODUCTION OF THE TRYPANOSOMES IN EITHER MAMMAL OR FLY HOST. There is some….

Stages of disease:

Early stage of disease – hemolymphatic phase – restriction of trypanosomes to blood and lymphatic system of humans. Fever, headache, joint pain, itching.

Late stage of disease – neurological phase – presence of parasites in the cerebrospinal fluid. Extreme lethargy, confusion, eventually coma and untreated, death.

T.b. rhodesiense (untreated will cause death in a few months) – acute sleeping sickness.

T.b. gambiense (untreated will cause death within a few years) – chronic sleeping sickness.

Reservoir hosts for both human and domestic animal disease forms are both wild and domestic livestock. Wild mammals are generally resistant or can be infected – showing few symptoms.

Animal trypanosomiasis- disease NAGANA of domestic livestock – caused by:

3. *Trypanosoma brucei brucei – ZOONOTIC* - occur in wild mammals –
4. *Trypanosoma congolense – ZOONOTIC* - occur in wild mammals –
5. *Trypanosoma vivax – ZOONOTIC* - occur in wild mammals – Africa – South and Central America

Other species of trypanosomes.

6. *Trypanosoma evansi- ZOONOTIC - widespread* - Causes **SURRA** in domestic animals. Mechanical transmission. Horse flies and other mechanical means of transmission. World wide in more tropical areas, probably transferred around by people.
7. *Trypanosoma equinum -ZOONOTIC - South America.*
8. *Trypanosoma equiperdum* **DOURINE** in domestic horses and donkeys.

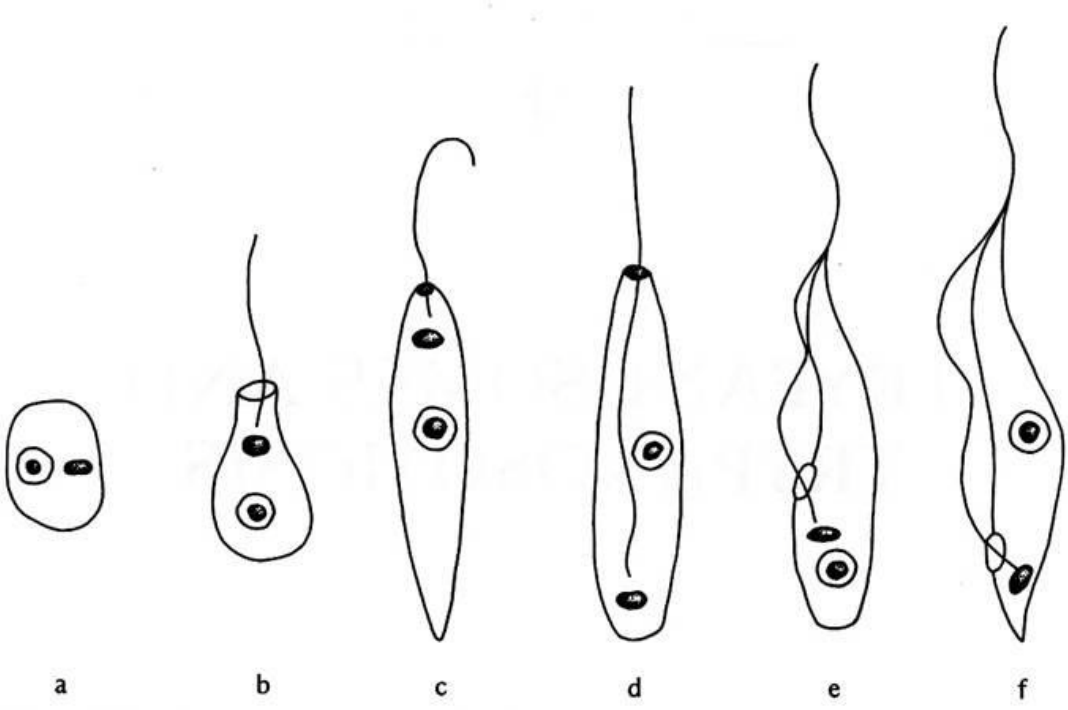
Evolution – ribosomal RNA sequence analysis in a phylogenetic framework shows deep time-phylogenetic split of salivarian from other trypanosomes. See fig. above. Trypanosomes were probably originally insect or other arthropod parasites and commensals that then became adapted to living in mammals.

-commensals of early insects – 380 mya.

-tsetse flies appeared in the fossil record about 35 mya.

Morphology –

a. *Leishmania* – amastigote (no external flagellum)
b. *Crithidia* – choanomastigote (collar cell with flagellum in center on apical part of cell)
c. *Leptomonas* – promastigote (flagellum arising from near anterior end)
d. *Herpetomonas* – opistomastigote (flagellum arising from near posterior exiting anterior)
e. *Blastocrithidia* – epimastigote (flagellum arising midbody exiting the body there too and attached with memberane to anterior end).
f. *Trypanosoma* – trypomastigote (flagellum arising at posterior of body, exiting and forming an undulating membrane toward anterior end and passing past anterior end).

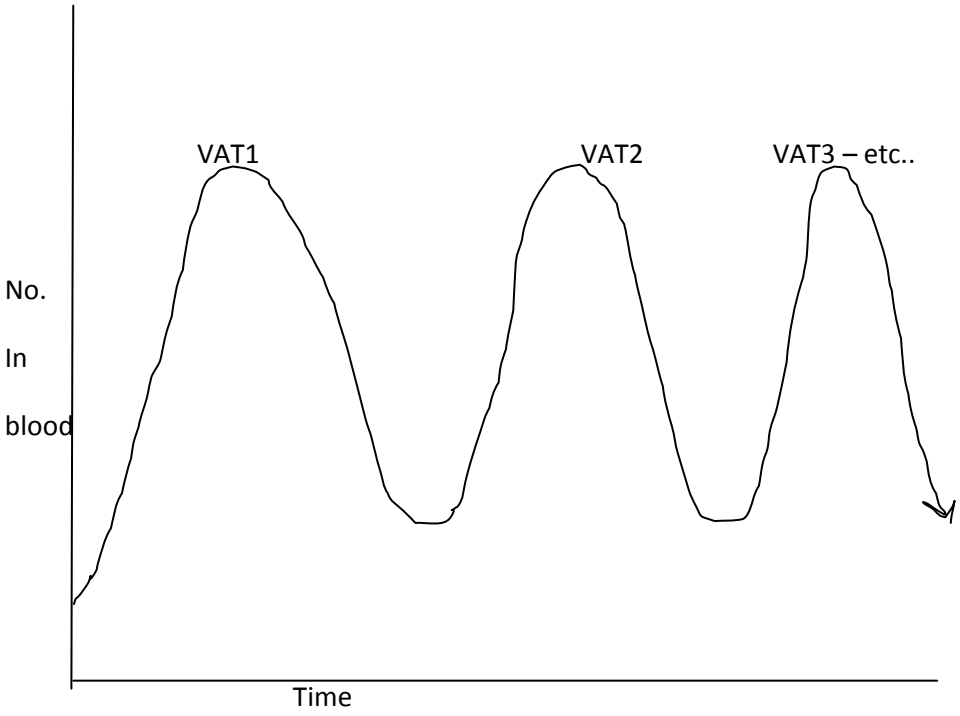


(a), amastigote, (b) choanomastigote, (c) promastigote, (d) opisthomastigote, (e) epimastigote, (f) trypomastigote

Some molecular biology and immunology –

Antigenic Variation. *Trypanosoma* spp. show what is called antigenic cycling. They are able to cycle through a variable antigen on their outer cell coat, presenting a different antigenic motif to the host antibody defense system every few days.

In this way, the parasite persists in a host in plain-sight of the host immune system, but the immune system cannot clear the infection. Variable Antigen Types cycle through a series of glycoproteins that coat the whole surface of the trypanosome. In a single host, there are many VATS but only one major population at any one time.



Control. – removal of vegetation. Wholesale removal of reservoir hosts, ineffective drugs.

SECTION STERCORARIA

Development of these trypanosomes occur in the posterior end of the insect host.

1. ***Trypanosoma cruzi*** - North America, Central America, South America
2. ***Trypanosoma lewisi-*** cosmopolitan in *Rattus* and probably widespread in other rodents.
3. ***Trypanosoma rangeli*** - Dogs cats and Humans in central and SA. All over Colombia and Panama.
4. ***Trypanosoma theileri*** - Cosmopolitan in cattle - vectors are Tabanidae

Life cycle.

In Bug.

Hemipteran - *Triatoma* **- bug feeds on blood** of mammal. Sucks up trypomastigote forms from a blood meal of a mammal.

These then turn into short epimastigote forms that reproduce in the midgut of the insect. The metacyclic trypomastigotes (non dividing) forms are then pooped out onto the skin or mucus membranes of the mammal host. A bug becomes infective in 8 – 10 days after feeding.

Defecates Trypomastigotes on skin or mucus membranes of mammal. Trypomastigotes enter blood stream (chagoma or sign of Romana). Trypomastigotes turn into amastigotes in subcutaneous cells of the skin. Chagoma may form.

In Mammal.

Trypomastigotes released into blood stream and penetrate either the -

Reticuloendothelial system (strain.) turn into amastigotes.

Myotropic strain. - gets into muscle and forms pseudocysts. Can infect smooth muscle, and cardiac muscle. Also as amastigotes.

Acute and chronic stages.

Ddiagnosis – xenodiagnosis.