

## AFRICAN TRYPANOSOMIASIS

**NOTE:** The following paragraphs offer information that may not be available in any regular text on parasitology. I thought it might be valuable to have a broader picture of African trypanosomiasis since it remains a major disease of humans and animals in Africa.

Trypanosomiasis are important diseases of man and animals in Africa especially in the regions south of the Sahara and in Sudan and southern Ethiopia. Although human trypanosomiasis was described by Atkins in 1724, it was not until 1901 that Forde discovered the causative organism in the blood of a West African. Bruce and Navarro in 1903 established that species of flies of the genus *Glossina* were the intermediate hosts and vectors of trypanosomiasis in man and animals. Since then, a tremendous effort had been made in the fight against these diseases in both man and animals with the result that they have been brought under rather dubious control.

It is estimated that species of *Glossina* capable of transmitting one form or another of trypanosomiasis occupy over four million square miles of fertile African land. The principal vectors of *T. gambiense* are *G. palpalis* and *G. tachinoides*, which are riverine species and live along rivers and lakes where dense vegetation borders these bodies of water. *G. longipalpis*, which is a forest species important in isolated areas of West Africa. The principal vectors of *T. rhodesiense* are *G. morsitans*, a savannah and bush-thicket species that feeds principally on undulates and travels long distances following the migration of game and cattle. *G. swynnertoni*, of lesser importance, is similar to *G. morsitans* habitat and feeding preferences. *G. pallidipes* is a forest fly that will feed on man and is a proven vector of *T. rhodesiense* in East Africa.

Both sexes of *Glossina* (tsetse flies) act as intermediate hosts and biological vectors of African trypanosomiasis. To become infected, the fly must feed on an infected individual within 24 hours after emerging for the pupa. About two days after the fly takes an infective meal, trypomastigotes (trypanosomes) begin to multiply in the lumen of the midgut and hindgut. Between the 10<sup>th</sup> and 15<sup>th</sup> day, they develop into long slender forms in the proventriculus. From the 16<sup>th</sup> to 29<sup>th</sup> day, they migrate through the esophagus, hypopharynx, and salivary ducts to the salivary glands where they develop first into epimastigotes (crithidia). In the salivary glands, they develop into metacyclic forms and infective trypomastigotes. After 18 to 21 days, the bite of the infected flies may transmit the infection to a susceptible host, man and animal.

Human African trypanosomiasis or African sleeping sickness results from infection with trypanosomes of the Brucei sub-group that is comprised of three species, *Trypanosoma gambiense*, *T. rhodesiense*, and *T. brucei*. *T. gambiense* and *T. rhodesiense*, the two species infecting man, differ in their ability to infect various laboratory, domestic and wild animals; the severity of the resulting disease; the species of intermediate host; and their resistance to drugs. These differences impact on one's ability to make an accurate diagnosis.

Human trypanosomiasis occurs over most of Africa within certain geographic limitations between 15° N and 20° S latitude where suitable vectors are present. There are from 10,000 to 20,000 new cases reported annually, most of which are *T. gambiense* and occur in West Africa. West African trypanosomiasis, resulting from

Infection with *T. gambiense*, is endemic along the coast of the continent from Senegal on the north to Angola to the south. It extends eastward into Kenya, Tanzania, and Sudan. East African trypanosomiasis, resulting from infection with *T. rhodesiense*, is found in Mozambique, Zambia, Malawi, Tanzania, Kenya, Uganda, Ethiopia, and Sudan.

In human Infections, after an incubation period of a few days, there is a rapid multiplication of forms (by longitudinal fission) and many small, irregular-sized forms may be seen in peripheral blood, many in division. As the disease progresses, the number of trypanosomes in the blood is reduced making diagnosis from peripheral blood more difficult.

Gambian trypanosomiasis usually manifests itself as a chronic disease of long duration. It is often diagnosed during the early stages before involvement of the central nervous system. As the disease proceeds, encephalitis, the "sleeping sickness" stage, develops. Clinical manifestations are usually suggestive and most often lead to an accurate diagnosis. Laboratory animals are poorly susceptible and rarely are inoculated as a diagnostic procedure. Occasionally, cases are acute and patients may succumb before the disease manifests itself in the central nervous system. If cases are recognized relatively early, even after neurological symptoms begin, the infection usually can be successfully treated. Patients who die, usually succumb to intercurrent diseases such pneumonia.

Rhodesian sleeping sickness is a much more insidious disease and proceeds through the early stages more rapidly. It may reach the central nervous system before symptoms are recognized. When symptoms do appear, the numbers of forms in the peripheral blood are often insufficient to find in peripheral blood and make a diagnosis. In suspected cases, from 5 to 10 ml of peripheral blood can be inoculated into the peritoneal cavity of a young laboratory rat. If the patient is infected, the blood will contain a few parasites and numerous trypomastigotes will develop in the rat within 4 or 5 days, usually in sufficient time to treat the patient successfully. In cases where diagnosis takes longer than two weeks, the outcome is serious and infection may be fatal.