

AFRICAN ANIMAL TRYPANOSOMIASIS

(Nagana, Tsetse Disease, Tsetse Fly Disease)

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Definition [top](#)

African animal trypanosomiasis (AAT) is a disease complex caused by tsetse-fly-transmitted *Trypanosoma congolense*, *T. vivax*, or *T. brucei brucei*, or simultaneous infection with one or more of these trypanosomes. African animal trypanosomiasis is most important in cattle but can cause serious losses in pigs, camels, goats, and sheep. Infection of cattle by one or more of the three African animal trypanosomes results in subacute, acute, or chronic disease characterized by intermittent fever, anemia, occasional diarrhea, and rapid loss of condition and often terminates in death. In southern Africa the disease is widely known as nagana, which is derived from a Zulu term meaning "to be in low or depressed spirits"— a very apt description of the disease.

Etiology [top](#)

African animal trypanosomiasis is caused by protozoa in the family Trypanosomatidae genus *Trypanosoma*. *T. congolense* resides in the subgenus *Nannomonas*, a group of small trypanosomes with medium-sized marginal kinetoplasts, no free flagella, and poorly developed undulating membranes. In east Africa, *T. congolense* is considered to be the single most important cause of AAT. This trypanosome is also a major cause of the disease in cattle in west Africa. Sheep, goats, horses, and pigs may also be seriously affected. In domestic dogs, chronic infection often results in a carrier state.

T. vivax is a member of the subgenus *Duttonella*, a group of trypanosomes with large terminal kinetoplasts, distinct free flagella, and inconspicuous undulating membranes. *T. vivax* is a large (18-26 μm long) monomorphic organism that is very active in wet-mount blood smears. Cattle, sheep, and goats are primarily affected. Although this organism is considered to be less pathogenic for cattle than *T. congolense*, it is nevertheless the most important cause of AAT in west African cattle. This trypanosome readily persists in areas free of tsetse flies (for example, in Central and South America and in the Caribbean), where it is transmitted mechanically by biting flies or contaminated needles, syringes, and surgical instruments.

T. brucei brucei resides in the subgenus *Trypanozoon*. *T. b. brucei* is an extremely polymorphic typanosome occurring as short, stumpy organisms without flagella, long slender organisms with distinct flagella, and intermediate forms that are usually flagellated. Horses, dogs, cats, camels and pigs are very susceptible to *T. b. brucei* infection. Infection of cattle, sheep, goats and sometimes pigs results in mild or chronic infection. This last observation, although widely accepted, has been called into question by Moulton and Sollod (13), who cite evidence that this organism is widespread in east and west Africa and that it can cause serious disease and high mortality in cattle, sheep, and goats.

Host Range [top](#)

Cattle, sheep, goats, pigs, horses, camels, dogs, cats, and monkeys are susceptible to AAT and may suffer syndromes ranging from subclinical mild or chronic infection to acute fatal disease. Rats, mice, guinea pigs, and rabbits are useful laboratory species.

More than 30 species of wild animals can be infected with pathogenic trypanosomes, and many of these remain carriers of the organisms. Ruminants are widely known to be active reservoirs of the trypanosomes. Wild Equidae, lions, leopards, and wild pigs are all susceptible and can also serve as carriers of trypanosomes.

Geographic Distribution [top](#)

The tsetse-fly-infested area of Africa extends from the southern edge of the Sahara desert (lat. 15° N.) to Angola, Zimbabwe, and Mozambique (lat. 20° S.). Of the three African animal trypanosomes, only *T. vivax* occurs in the Western Hemisphere in at least 10 countries in the Caribbean and South and Central America

Transmission [top](#)

In Africa, the primary vector for *T. congolense*, *T. vivax*, and *T. b. brucei* is the tsetse fly. These trypanosomes replicate in the tsetse fly and are transmitted through tsetse fly saliva when the fly feeds on an animal. The three main species of tsetse flies for transmission of trypanosomes are *Glossina morsitans*, which favors the open woodland of the savanna; *G. palpalis*, which prefers the shaded habitat immediately adjacent to rivers and lakes; and *G. fusca*, which favors the high, dense forest areas. Trypanosomiasis is also mechanically transmitted by tsetse and other biting flies through the transfer of blood from one animal to another. The most important mechanical vectors are flies of the genus *Tabanus*, but *Haematopota*, *Liperosia*, *Stomoxys*, and *Chrysops* flies have also been implicated. In Africa, both *T. vivax* and *T. b. brucei* have spread beyond the "tsetse fly belts" (20), where transmission is principally by tabanid and hippoboscid flies.

The vector for *T. vivax* in the Western Hemisphere remains unknown, but several species of hematophagous (especially tabanid and hippoboscid) flies are believed to serve as mechanical vectors.

Incubation Period [top](#)

The incubation period for *T. congolense* varies from 4 to 24 days; for *T. vivax*, from 4 to 40 days; and for *T. b. brucei*, from 5 to 10 days.

Pathogenesis [top](#)

Initial replication of trypanosomes is at the site of inoculation in the skin; this causes a swelling and a sore (chancre). Trypanosomes then spread to the lymph nodes and blood and continue to replicate. *T. congolense* localizes in the endothelial cells of small blood vessels and capillaries. *T. b. brucei* and *T. vivax* localize in tissue. Antibody developed to the glycoprotein coat of the trypanosome kills the trypanosome and results in the development of immune complexes.

Antibody, however, does not clear the infection, for the trypanosome has genes that can code for many different surface-coat glycoproteins and change its surface glycoprotein to evade the antibody. Thus, there is a persistent infection that results in a continuing cycle of trypanosome replication, antibody production, immune complex development, and changing surface-coat glycoproteins.

Immunologic lesions are significant in trypanosomiasis, and it has been suggested that many of the lesions (e.g., anemia and glomerulonephritis) in these diseases may be the result of the deposition of immune complexes that interfere with, or prevent, normal organ function. The most significant and complicating factor in the pathogenesis of trypanosomiasis is the profound immunosuppression that occurs following infection by these parasites. This marked immunosuppression lowers the host's resistance to other infections and thus results in secondary disease, which greatly complicates both the clinical and pathological features of trypanosomiasis.

Clinical Signs [top](#)

Because simultaneous infections with more than one trypanosome species are very common (18), and simultaneous infection with trypanosomes and other hemoparasites (*Babesia* spp., *Theileria* spp., *Anaplasma* spp., and *Ehrlichia* spp.) frequently occurs, it is difficult to conclude which clinical signs are attributable to a given parasite. Few adequately controlled studies have been made, and thus a "typical" clinical response to each trypanosome is difficult to reconstruct. What follows is a summation of the syndromes observed in field and experimental cases of trypanosomiasis caused by each of the three African animal trypanosomes.

The cardinal clinical sign observed in AAT is anemia. Within a week of infection with the hematic trypanosomes (*T. congolense* and *T. vivax*) there is usually a pronounced decrease in packed cell volume, hemoglobin, red blood cell, and white blood cell levels, and within 2 months these may drop to below 50 percent of their preinfection values. Also invariably present are intermittent fever, edema and loss of condition (Fig. 2). Abortion may be seen, and infertility of males and females may be a sequel. The severity of the clinical response is dependent on the species and the breed of affected animal and the dose and virulence of the infecting trypanosome. Stress, such as poor nutrition or concurrent disease, plays a prominent role in the disease process, and under experimental conditions, where stress may be markedly reduced, it is difficult to elicit clinical disease.

T. congolense is a hematic trypanosome found only in the blood vessels of the animals it infects. It does not localize and multiply outside blood vessels. Infection with *T. congolense* may result in peracute, acute, or chronic disease in cattle,

sheep, goats, horses, and camels. Pigs often develop a milder disease; chronic disease is common in dogs. The incubation period is followed by intermittent febrile episodes, depression, lethargy, weakness, loss of condition, anemia, salivation, lacrimation, and nasal discharge. As the disease progresses, loss of condition and hair color changes from black to metallic brown are seen. The back is often arched and the abdomen "tucked up." Accelerated pulse and jugular pulsation occur and breathing is difficult. Anemia is a prominent sign. Early in the infection, the organisms are readily demonstrable in blood smears, but, as the disease progresses to its acute and chronic forms, organisms are most readily demonstrated in lymph node smears.

T. vivax has a variable incubation period, and, although it is considered to be less virulent for cattle than *T. congolense*, mortality rates of over 50 percent can occur. There seems to be a marked variation in the virulence of different strains of *T. vivax*, but it remains the most important cause of trypanosomiasis of cattle, sheep, and goats in west Africa. It causes mild disease in horses and chronic disease in dogs. *T. vivax* is often difficult to find in blood smears and can also be demonstrated in lymph node smears.

T. brucei brucei has a relatively short incubation period and causes severe to fatal infection in horses, camels, dogs, and cats. It usually causes mild, chronic, or subclinical disease in cattle, sheep, goats, and pigs. A febrile response occurs in the horse 4-14 days after infection. This is followed by recurrent febrile reactions. The heartbeat and respiration may be accelerated, and loss of condition and weakness are seen, whereas the appetite remains good. Progressive anemia and icterus, and edema of the ventral regions, especially the male genitalia, are characteristic. The organisms are not always easily perceived in blood smears and are best demonstrated in tissue smears or sections, (e.g., lymph nodes). Infected animals die in a few weeks or several months, depending on the virulence of the strain of *T. b. brucei*.

The marked immunosuppression resulting from trypanosome infection lowers the host's resistance to other infections and causes in secondary disease, which greatly complicates both the clinical and pathological features of trypanosomiasis.

Gross Lesions [top](#)

No pathognomonic change is seen in AAT. Anemia, edema, and serous atrophy of fat are commonly observed. Subcutaneous edema is particularly prominent and is usually accompanied by ascites, hydropericardium, and hydrothorax. The liver may be enlarged, and edema of lymph nodes is often seen in the acute disease, but they may be reduced in size in the chronic disease. The spleen and lymph

nodes may be swollen, normal, or atrophic. Necrosis of the kidneys and heart muscle and subserous petechial hemorrhages commonly occur. Gastroenteritis is common, and focal polioencephalomalacia may be seen. A localized lesion (chancre) may be noted at the site of fly bite, especially in goats. The anemic blood changes are anisocytosis, poikilocytosis, polychromasia, and punctate basophilia. All, some, or none of the above may be seen.

The lesions caused by the trypanosomes in susceptible host species vary considerably, depending on the species and strain of trypanosome and the species and breed of host animal affected. The hematic trypanosomes (*T. congolense* and *T. vivax*) cause injury to the host mainly by the production of severe anemia, which is accompanied in the early stages of the disease by leukopenia and thrombocytopenia. In the terminal stages of the disease caused by the hematic trypanosomes, focal polioencephalomalacia probably results from ischemia due to massive accumulation of the parasites in the terminal capillaries of the brain.

The lesions resulting from *T. b. brucei* (a tissue parasite) are remarkably different from those seen with the hematic trypanosomes. Anemia is an important lesion, but much more dramatic are the inflammation, degeneration, and necrosis resulting from cellular invasion of various organs. Marked proliferative changes reflecting immunologic response are observed in most body tissues.

Diagnosis [top](#)

Field Diagnosis [top](#)

Trypanosomiasis should be suspected when an animal in an endemic area is anemic and in poor condition. Confirmation depends on the demonstration of the organism in blood or lymph node smears.

In the early phases of infection, especially with *T. vivax* and *T. congolense*, the parasite can readily be observed by microscopic examination of a wet-mount of blood slides. Thick blood films and stained with Giemsa are also a good technique (Fig. 1), but in thin fixed blood films, which are favored for species identification, the parasites may be hard to demonstrate. When parasitemia is low, smears of buffy coat (obtained by microhematocrit centrifugation) can be useful for demonstration of the parasites. Because *T. congolense* tends to associate with the erythrocytes, it is essential that buffy coat and adjacent erythrocytes be included in the smear to ensure demonstration of the parasite.

Stained lymph node smears are a very good method for diagnosis, especially for *T. vivax* and *T. b. brucei*. In chronic *T. congolense* infection, the parasites localize

in the microcirculation of the lymph nodes and in other capillary beds, allowing diagnosis by examination of lymph node smears or smears made with blood collected from the ear. Early in infection, blood smears are optimal for the demonstration of *T. congolense*.

These conventional techniques of microscopic examination for the presence of trypanosomes are still widely used, but newer and far more sensitive methods are beginning to supplant them. The antigen-detecting enzyme-linked immunosorbent assay is extremely sensitive for the detection of trypanosomiasis in cattle and goats (12, 25), and species-specific DNA probes have been shown to detect simultaneous infection of cattle with *T. vivax*, *T. b. brucei*, and *T. congolense* when conventional methods revealed only single infections (18).

Specimens for the Laboratory [top](#)

To perform the preceding and more sensitive procedures, the following specimens should be submitted to the laboratory from several animals: serum, blood with the anticoagulant EDTA, dried thin and thick blood smears, and smears of needle lymph node biopsies.

Control and Eradication [top](#)

Vector Control [top](#)

Fly eradication and drug prophylaxis are the only effective trypanosomiasis control methods now available. Several approaches to fly control have been used with varying degrees of success.

Discriminative bush clearing, extensively used in early tsetse fly eradication campaigns, has been locally useful because it eliminates the breeding places of the tsetse. But, to be completely effective, bush clearing requires ecologically unacceptable destruction of vast areas of brush and forest. It is still a useful procedure when used locally in conjunction with other control methods.

Game elimination, and thus elimination of the main source of bloodmeals for the tsetse, was used in early eradication campaigns.

This was an ineffective and wasteful procedure.

Application of the sterile male technique (as used in screwworm eradication in the United States) received considerable attention in the 1980's. Early problems with

breeding of the male flies have been overcome, and field trials have been done in both east and west Africa to determine the effectiveness of this approach in vector control. In limited trials, this procedure has reduced fly populations.

Ground and aerial spraying with insecticides and the use of synthetic pyrethroids on cattle have lowered fly densities in some areas, but widespread use would require considerable international cooperation and expense. Widespread application of insecticide has the tremendous disadvantage of also eradicating many other arthropods, several of which are desirable. The recent introduction of odor-baited targets impregnated with insecticides is proving promising as a means of reducing the tsetse fly.

Chemotherapy and Chemoprophylaxis [top](#)

The use of drugs for the prevention and treatment of trypanosomiasis has been important for many decades, but the rapidity with which the trypanosomes have developed resistance to each drug introduced has tremendously complicated this approach to controlling the disease. In spite of this, some of the older chemoprophylactic drugs such as the quinapyramine derivatives Antrycide and Antrycide Prosalt are still used and give effective protection against *T. b. brucei* infection in horses, camels, and cattle for up to 3 months. The drug pyrrithidium bromide (Prothidium and AD2801) is useful in the prophylaxis of *T. vivax* and *T. congolense* infections in cattle, sheep, and goats and can give protection for up to 6 months. The most widely used of the newer chemoprophylactic drugs (and also the least expensive) is isometamidium chloride (26). This drug, in use for over 20 years and sold under the trade names Samorin, Trypamidium, and M&B 4180A, is excellent for the prophylaxis of all three African animal trypanosomes, and gives protection for 3-6 months. The development of resistance to this drug has been reported in both east and west Africa. Homidium bromide has also been found to be an effective chemoprophylactic drug in Kenya, and the newly introduced arsenical Cymelarsan is effective in treatment of *T. b. brucei* infection.

A very widely used chemotherapeutic drug is diminazine aceturate (Berenil), which is effective against all three African animal trypanosomes. The isometamidium drugs are also excellent chemotherapeutic agents as are the quaternary ammonium trypanocides Antrycide, Ethidium and Prothidium.

Although extensively used in trypanosomiasis control, chemoprophylaxis is an expensive, time-consuming, and thus unsatisfactory long-term solution to the problem of African animal trypanosomiasis.

Immunization [top](#)

No vaccine is currently available for African animal trypanosomiasis.

Trypanotolerance [top](#)

It has long been recognized that certain breeds of African cattle are considerably more resistant to African trypanosomiasis than others. This is especially true of the west African short-horned cattle (Muturu, Baoule, Laguna, Samba, and Dahomey) and the N'Dama, which is also of west Africa. These cattle have existed in the region for over 5,000 years. Susceptibility studies have shown the N'Dama to be the most resistant breed followed by the smaller west African short-horned cattle, but the large and more recently introduced Zebu is the most susceptible (15). The mechanisms of trypanotolerance have been extensively studied, and it is now well established that trypanotolerance has a genetic basis (13, 17). Trypanotolerance in sheep and goats has also been described, but the mechanisms of the tolerance phenomenon have not been defined.

Public Health [top](#)

The three AAT trypanosomes are considered to be nonpathogenic for humans. *T. b. brucei*, although not causing human disease, is closely related to *T. b. gambiense* and *T. b. rhodesiense*. The latter is the cause of human sleeping sickness, a very debilitating and often fatal disease considered to be of major public health significance in 36 sub-Saharan countries of west, central, and east Africa with 50 million people at risk (18). In west and central Africa, a chronic form of human sleeping sickness is caused by *T. b. gambiense*, which uses humans as its major host but also infects pigs. In east and southern Africa, *T. b. rhodesiense* is the cause of a much more acute form of human sleeping sickness. This trypanosome also infects cattle, bushbuck (*Tragelaphus scriptus*), and probably many other wild animals that may serve as reservoirs of the parasite.

GUIDE TO THE LITERATURE [top](#)

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