

Tsetse-transmitted Animal Trypanosomiasis

This group of diseases caused by protozoa of the genus *Trypanosoma* affects all domestic animals. The major species are *T congolense* , *T vivax* , *T brucei brucei* , and *T simiae* .

See Table below for Tsetse-transmitted Animal Trypanosomes for the animals mainly affected by these tsetse-transmitted trypanosomes and the geographic areas where tsetse-transmitted trypanosomiasis occurs. Cattle, sheep, and goats are infected, in order of importance, by *T congolense* , *T vivax* , and *T brucei brucei* . In pigs, *T simiae* is the most important. In dogs and cats, *T brucei* is probably the most important. It is difficult to assign an order of importance for horses and camels. *T vivax* may occur outside tsetse-infested areas of sub-Saharan Africa.

<i>Trypanosoma spp</i>	<i>Animals Mainly Affected</i>	<i>Major Geographic Distribution</i>
<i>T congolense</i>	Cattle, sheep, goats, dogs, pigs, camels, horses, most wild animals	Tsetse region of Africa
<i>T vivax</i>	Cattle, sheep, goats, camels, horses, various wild animals	Africa, Central and South America, West Indies*
<i>T brucei brucei</i>	All domestic and various wild animals; most severe in dogs, horses, cats	Tsetse region of Africa
<i>T simiae</i>	Domestic and wild pigs, camels	Tsetse region of Africa

The trypanosomes that cause tsetse-transmitted trypanosomiasis (sleeping sickness) in humans, *T brucei rhodesiense* and *T brucei gambiense* , closely resemble *T brucei brucei* from animals, and suitable precautions should be taken when working with such isolates. Domestic animals may act as reservoirs of human infections

Transmission & Epidemiology: Most tsetse transmission is cyclic and begins when blood from a trypanosome-infected animal is ingested by the fly. The trypanosome loses its surface coat, multiplies in the fly, then reacquires a surface coat and becomes infective. *T brucei spp* migrate from the gut to the proventriculus to the pharynx and eventually to the salivary glands; the cycle for *T congolense* stops at the hypopharynx, and the salivary glands are not invaded; the entire cycle for *T vivax* occurs in the proboscis. The animal-infective form in the tsetse salivary gland is referred to as the metacyclic form. The life cycle in the tsetse may be as short as 1 wk with *T vivax* or extend to a few weeks for *T brucei spp*

Tsetse flies (genus *Glossina*) are restricted to Africa from about latitude 15°N to 29°S. The 3 main species inhabit relatively distinct environments— *G morsitans* usually is found in savanna country, *G palpalis* prefers areas around rivers and lakes, and *G fusca* lives in high forest areas. All 3 species transmit trypanosomes and all feed on various mammals.

Mechanical transmission can occur through tsetse or other biting flies. In the case of *T vivax* , *Tabanus* spp and other biting flies seem to be the primary mechanical vectors outside the tsetse areas, as in Central and South America. Mechanical transmission requires only that blood containing infectious trypanosomes be transferred from one animal to another.

Pathogenesis: Infected tsetse inoculate metacyclic trypanosomes into the skin of animals, where the trypanosomes grow for a few days and cause localized swellings (chancres). They enter the lymph nodes, then the bloodstream, where they divide rapidly by binary fission. In *T congolense* infection, the organisms attach to endothelial cells and localize in capillaries and small blood vessels. *T brucei*

species and *T vivax* invade tissues and cause tissue damage in several organs.

The immune response is vigorous, and immune complexes cause inflammation, which contributes to the signs and lesions of the disease. Antibodies against the surface-coat glycoproteins kill the trypanosomes. However, trypanosomes have multiple genes that code for different surface-coat glycoproteins that are not vulnerable to the immune response; this antigenic variation results in persistence of the organism. The number of antigenic types of glycoprotein that can be made is unknown, but exceeds several hundred. Antigenic variation has prevented development of a vaccine and permits reinfections when animals are exposed to a new antigenic type.

Clinical Findings & Lesions: Severity of disease varies with species and age of the animal infected and the species of trypanosome involved. The incubation period is usually 1-4 wk. The primary clinical signs are intermittent fever, anemia, and weight loss. Cattle usually have a chronic course with high mortality, especially if there is poor nutrition or other stress factors. Ruminants may gradually recover if the number of infected tsetse flies is low; however, stress results in relapse.

Necropsy findings vary and are nonspecific. In acute, fatal cases, extensive petechiation of the serosal membranes, especially in the peritoneal cavity, may occur. Also, the lymph nodes and spleen are usually swollen. In chronic cases, swollen lymph nodes, serous atrophy of fat, and anemia are seen.

A presumptive diagnosis is based on finding an anemic animal in poor condition in an endemic area. Confirmation depends on demonstrating trypanosomes in stained blood smears or wet mounts. The most sensitive rapid method is to examine a wet mount of the buffy coat area of a PCV tube after centrifugation. Other infections that cause anemia and weight loss, such as babesiosis, anaplasmosis, and theileriosis, should be ruled out by examining a stained blood smear.

Various serologic tests measure antibody to trypanosomes, but their use is more suitable for herd and area screening than for individual diagnosis. Tests for detection of circulating trypanosome species-

specific antigens in peripheral blood are becoming available for both individual and herd diagnosis, although their reliability remains unproven.

Treatment & Control: Several drugs can be used for treatment. Most have a narrow therapeutic index, which makes administration of the correct dose essential. Drug resistance occurs and should be considered in refractory cases.

Control can be exercised at several levels, including eradication of tsetse flies and use of prophylactic drugs. Tsetse flies can be partially controlled by frequent spraying and dipping of animals, spraying of insecticides on fly-breeding areas, use of insecticide-impregnated screens, bush clearing, and other methods. Animals can be given drugs prophylactically in areas with a high population of trypanosome-infected tsetse. Drug resistance must be carefully monitored by frequent blood examinations for trypanosomes in treated animals.

In west Africa, several breeds of cattle have been identified that show innate resistance to trypanosomiasis and play a valuable role in reducing the impact of the disease in this area. However, resistance may be lost due to poor nutrition or heavy tsetse challenge.

Control is ideally achieved by combining methods to reduce the tsetse challenge and by enhancing host resistance with prophylactic drugs.