# African Animal Trypanosomiasis

*Nagana, Tsetse Disease, Tsetse Fly Disease, African Animal Trypanosomosis*

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## Importance

African animal trypanosomiasis is a parasitic disease, caused by several species of *Trypanosoma*, that can result in illnesses and significant economic losses in cattle, other livestock, pets and wild mammals from anemia, loss of condition and effects on reproduction. Untreated cases can be fatal, with a high mortality rate in some outbreaks, and only a limited number of drugs are available for treatment. The causative agents mainly circulate in parts of Africa that contain their biological vector, the tsetse fly; however, some species can also be found outside these regions due to mechanical transmission and the movement of infected animals. One organism, *Trypanosoma vivax*, has become established in South America, where tsetse flies are absent but various biting flies serve as mechanical vectors.

## Etiology

African animal trypanosomiasis is the disease caused by the members of the protozoan genus *Trypanosoma* (family Trypanosomatidae, Salivarian section) that are transmitted by tsetse flies and cause illnesses in animals but do not ordinarily affect humans. They currently comprise several organisms in the *Trypanosoma* subgenera *Nannomonas*, *Duttonella*, *Trypanozoon* and *Pycnomonas*, including the species *Trypanosoma* (*Nannomonas*) *congolense*, *T. (Nannomonas) simiae*, *T. (Nannomonas) godfreyi*, *T.* (*Duttonella) vivax*, *T. (Duttonella) uniforme*, *T.* (*Trypanozoon*) *brucei* subsp. *brucei* and *T. (Pycnomonas) suis.* A *T. suis*-like organism was recently detected in domestic and wild ruminants and appears to be a distinct species, and there may be other agents that have not yet been discovered. Species of trypanosomes, as well as different isolates of a given organism, can differ in virulence.

African animal trypanosomiasis traditionally excludes two tsetse-transmitted parasites, *T. brucei* subsp. *gambiense* and *T. brucei* subsp. *rhodesiense*, which evade the innate resistance humans possess against other trypanosomes and cause a disease known as human African trypanosomiasis or sleeping sickness. However, neither of these organisms is exclusive to humans. *T. b. rhodesiense* is a zoonotic parasite maintained in livestock and wildlife, and while *T. b. gambiense* is mainly thought to have human reservoirs, it has been reported in animals.

African animal trypanosomiasis also excludes two non-tsetse transmitted trypanosomes of animals, *T. evansi*, which causes surra, and *T. equiperdum*, the agent of dourine. (These two diseases are described in separate factsheets.) However, *T. evansi* and *T. equiperdum* are closely related to *T. brucei* subsp. *brucei*, and whether all three organisms should be considered distinct species or reclassified as subspecies of either *T. brucei* or *T. evansi* is controversial.

## Species Affected

One or more of the organisms that cause African animal trypanosomiasis have been found in many mammals, including all domestic animals and a number of free-living or captive wildlife such as various wild ruminants (e.g., African buffalo, *Syncerus caffer*), cervids, antelopes, equids, rhinoceroses, hippopotamuses (*Hippopotamus amphibius*), giraffes (*Giraffa camelopardalis*) and other ungulates; African elephants (*Loxodonta africana*); some wild felids, canids and hyaenids; wild suids; nonhuman primates; and rodents. However, there are some mammals, such as the African hedgehog *Atelerix albiventris*, that are resistant to infection due to trypanolytic activity in the blood or other factors. Reptiles and birds carry their own species of trypanosomes, but *T. vivax* DNA was detected by PCR in crocodiles and monitor lizards (*Varanus ornatus*) in Africa. Whether this organism can become established in reptiles or is only inoculated transiently by insects remains to be determined.

Some trypanosomes seem to have a broader host range than others. While agents such as *T. congolense*, *T. vivax* and *T. b. brucei* can be found in a number of hosts, with reservoirs in cattle, small ruminants, pigs and some wildlife, *T suis* only seems to infect domestic pigs and wild African suids such as warthogs (*Phacochoerus* spp.). *T. godfreyi* and *T. simiae* also affect pigs, but the latter organism has been detected in domestic ruminants, camels, horses and some wildlife as well.

Clinical cases of African animal trypanosomiasis have been documented in a number of species including cattle, water buffalo, sheep, goats, pigs, various equids, camels, South American camelids, dogs, cats and some captive or free-living wildlife. *T. vivax* and *T. congolens*e are considered to be the major pathogens in ruminants, while *T. b. brucei* is thought to be of lesser significance. Many clinical cases in dogs have been caused by *T. congolense*, although other organisms can also be responsible.

### *Zoonotic potential*

People are not usually susceptible to the trypanosomes that cause African animal trypanosomiasis. The trypanolytic activity of apolipoprotein L-I (apoL-I), found in human blood, is thought to be the major innate defense mechanism. Nevertheless, there have been a few reports of human infections caused by *T. b. brucei*, *T. vivax* and *T. congolense*. Whether these cases occurred in people with genetic defects in apoL-I proteins, were caused by trypanosome isolates with elevated resistance to human apoL-I, or happened for other reasons is currently unclear.

Some of the reported cases might have been mistakes. Most were not described fully, and many are not definitive, as they predate modern identification methods and were diagnosed by the parasite's morphology alone. However, one infection with *T. b. brucei* occurred after accidental inoculation in a laboratory, and one of 7 volunteers experimentally infected with this organism in the 1940s became infected for 3 weeks. Two clinical cases of sleeping sickness in people infected with *T. b. brucei* or *T. congolense* were diagnosed by PCR-based confirmation of the organism's identity. In the latter case, the person was infected with both *T. congolense* and an unidentified subspecies of *T. brucei*, which might have been one of the usual agents of sleeping sickness in humans, *T. b. gambiense* or *T. b. rhodesiense*. Therefore, the contribution of *T. congolense* to this illness, if any, is uncertain. There is also a survey from Chad, published in 2021, that reported finding nucleic acids of *T. congolense* in 11 of approximately 800 people tested by PCR. This result remains to be confirmed, particularly as the blood was not examined for live parasites.

## Geographic Distribution

Most of the organisms that cause African animal trypanosomiasis are endemic only in areas of sub-Saharan Africa where their biological vectors can be found. This region, called the tsetse fly belt, is located roughly between latitudes 10°N and 20-30°S; however, not all parts of this region are suitable habitats for the flies. A few species of tsetse flies have been detected in the southwestern Arabian Peninsula.

*T. vivax*, can also become established outside tsetse fly areas, where it is transmitted by various biting insects acting as mechanical vectors. Outside Africa, this organism currently occurs in parts of South and Central America and the Caribbean, and it was identified recently in Iran.

## Transmission

Tsetse flies (*Glossina* spp.) are the biological vectors for the trypanosomes that cause African animal trypanosomiasis, and transmit these organisms in their saliva during bites. Tsetse fly feeding preferences vary, with some species displaying a strong preference for certain hosts (e.g., cattle) even when their abundance is low, while others are less selective. Trypanosomes must develop for one to a few weeks in the fly before they reach the infective stage. *T. vivax* has the shortest cycle.

Trypanosomes can also be transmitted iatrogenically by fomites exposed to blood or tissues, such as surgical instruments or needles/ syringes, and mechanically by various biting flies. Mechanical transmission is particularly significant for *T. vivax*, which can be maintained by this route outside the tsetse fly belt. Tabanidae (horseflies) are thought to be its most important insect vectors in South America. Stable flies (*Stomoxys* spp.) might also play a role, though some experiments suggest that, at best, they transmit *T. vivax* very inefficiently.

Carnivores can probably also become infected by eating tissues from infected animals, with transmission experiments suggesting that this must occur immediately after death. Trypanosomes do not survive long outside a living host and disappear rapidly from the carcass, though *T. brucei* is reported to remain alive for a few hours in blood, and for up to a few days if it is refrigerated under certain conditions (e.g., in blood with adequate oxygen). Transplacental transmission of African animal trypanosomes has been reported in several hosts. There is also some speculation about the possibility of venereal transmission, based on the detection of *T. vivax* nucleic acids in the semen of experimentally infected goats and cattle, together with the ability of some other trypanosomes, such as *T. equiperdum*, to spread by this route.

## Disinfection

There is limited need for disinfection, due to the fragility of trypanosomes in the environment; however, *T. brucei* and *T. vivax* are reported to be susceptible to a number of agents including sodium hypochlorite, 50-70% ethanol, chlorhexidine, iodides, hydrogen peroxide, some quaternary ammonium compounds, formaldehyde, glutaraldehyde and 0.1% hand soap. Somewhat longer contact times and/or higher concentrations were needed to decontaminate blood, compared to surfaces, when certain agents were used. Trypanosomes are also inactivated by heating to 50°C (122°F).

## Incubation Period

The incubation period is variable, as some animals can carry trypanosomes subclinically for months or years before becoming symptomatic. Acute illnesses in ruminants have been reported after incubation periods ranging from 4 days to approximately 2 months, and most cases in dogs are thought to become apparent in about 1-3 weeks, though some may take up to 2 months or more.

## Clinical Signs

Ruminants, including cattle, water buffalo, sheep and goats, can be infected with trypanosomes either with or without clinical signs. Most clinical cases in these species are chronic, but acute illnesses are also possible. Common signs include an intermittent fever, signs of anemia (e.g., pale mucous membranes, lethargy), lymphadenopathy and weight loss, which may be rapid. Some animals initially have a localized swelling (chancre) at the site of the fly bite; however, this usually remains unnoticed. There may also be a decreased appetite, dependent edema (including submandibular edema), ocular lesions (conjunctivitis, keratitis, corneal opacity), diarrhea, reduced milk production and occasional cases with neurological signs. Reproductive effects can include abortions, premature births, perinatal losses and damage to the male reproductive organs (e.g., orchitis, epididymitis) with reduced semen quality. Jaundice has been reported in some outbreaks, possibly as the result of coinfection with organisms such as *Anaplasma marginale*. Anemia, thrombocytopenia and leukopenia (early) or leukocytosis are common laboratory findings, and hypoglycemia may be seen in the acute stage.

Trypanosomes can also cause immunosuppression, and concurrent infections may complicate the disease. While some animals may recover spontaneously, and others can carry the organisms long-term without significant clinical signs, untreated acute or chronic illnesses can be fatal, with acutely ill animals sometimes dying within weeks. Sudden deaths have also been reported occasionally, particularly in sheep and goats. Animals that recover clinically may relapse when stressed.

A variation on this pattern, an acute hemorrhagic syndrome that might be mediated by antibodies to red blood cells, has been reported in cattle infected with some isolates of *T. vivax*. This syndrome, which can be rapidly fatal, has mostly been documented in East Africa, and is characterized by enlarged lymph nodes, signs of severe anemia, and widespread visceral and mucosal hemorrhages accompanied by weight loss, which may be severe. The hemorrhages often affect the gastrointestinal tract, but in one outbreak, the main sign was bleeding from the ears.

Clinical signs in other animals are reported to be similar to those in ruminants, though information about some species is limited. Neurological signs seem to be more common in some species, such as equids and dogs, than ruminants, and while donkeys are often reported to have subclinical infections, cerebral trypanosomiasis with progressive cerebral dysfunction, spinal ataxia and cranial nerve deficits is reported to affect both horses and donkeys in the Gambia. Gastrointestinal signs in some hosts can include vomiting as well as diarrhea, with rare reports of acute hemorrhagic vomiting and diarrhea in dogs. In one unusual case, a dog with clinical signs of trypanosomiasis presented with seizures and severe hypoglycemia that was refractory to treatment with glucose and oral sugar supplementation but responded to elimination of the trypanosomes.

There is relatively little information about pigs, which often seem to carry trypanosomes subclinically; however, *T. simiae* was reported to cause a hyperacute illness in this species, with death often occurring within 48 hours of the initial signs, while experimental *T. suis* infection was characterized by severe acute disease in piglets and chronic infections in adults.

## Post Mortem Lesions

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The gross lesions are nonspecific and often include an enlarged spleen, indications of anemia, and petechiae on serosal surfaces, particularly in the peritoneal cavity. Lymphadenopathy is common in the acute stage, but later the lymph nodes may be enlarged, normal or atrophied. Carcasses often have evidence of weight loss and serous atrophy of fat, and may be emaciated. Additional lesions in some animals can include an enlarged liver, subcutaneous edema, excessive fluid in the body cavities and pericardial sac, pulmonary edema, keratitis, cardiac damage (e.g., myocarditis) and inflammation caused by immune complexes in the kidneys, blood vessels and other tissues.

## Diagnostic Tests

A presumptive diagnosis can be made in an endemic area if trypanosomes are observed by direct microscopic examination of blood, lymph nodes (e.g., smears of needle biopsies), edema fluid or tissues collected at necropsy. Organisms are most readily found in the blood during the initial stages of the infection. They are less likely to be detected in chronically ill animals, and are almost never seen in healthy carriers. Detection can be improved with parasite concentration techniques such as hematocrit centrifugation, the quantitative buffy coat method, mini anion–exchange chromatography and the dark-ground/phase-contrast buffy coat technique.

Stained thick or thin blood smears are usually used to look for trypanosomes, but motile organisms can be observed in wet films. Thick films have the advantage of being able to detect small numbers of parasites, while parasite morphology is easier to see in thin films. An organism's morphology and size may suggest its subgenus and/or species. However, some trypanosomes cannot be distinguished visually, animals can be infected concurrently with more than one species of trypanosome, and unusual morphologies have been reported occasionally. Care must also be taken not to confuse avirulent organisms such as *Trypanosoma theileri*, or species of unknown health significance such as *T. caninum*, with pathogens.

Clinical cases can also be diagnosed by PCR, and other genetic tests such as loop-mediated isothermal amplification (LAMP) methods have been described in the literature. PCR tests that identify trypanosomes at the genus, species or subspecies level may suggest the organism's identity; however, definitive confirmation may require a level of analysis that is impractical for routine diagnosis, as several PCR tests and/or sequencing may be needed to distinguish some organisms.

Trypanosomes can be isolated by *in vitro* cultivation or inoculation into rats or mice, but these methods are generally reserved for special circumstances due to their cost and the time required. Only some pathogenic species will grow *in vitro*, and nonpathogenic trypanosomes can readily overgrow cultures, while animal welfare is a concern for animal inoculation.

Serology is mainly used in surveillance or to identify infected animals before transport to trypanosome-free areas; however, the presence of antibodies could provide supporting evidence for trypanosomiasis outside endemic regions and a clinical case might be diagnosed by a rise in titer. Commonly used serology tests in cattle include the indirect fluorescent antibody test and ELISAs. Immunoblotting has also been described, but it is not in routine use and is generally unavailable outside reference laboratories. The various trypanosomes found in animals can cross-react in serological tests.

## Treatment

African animal trypanosomiasis can be treated with antiparasitic (trypanocidal) drugs, which have varying efficacy against different organisms. The number of effective drugs is limited, and few or no agents may be readily available outside endemic areas. Protocols for less common hosts should be chosen carefully, as the efficacy and toxicity of a particular drug may differ between animal species. Drug resistance is a significant issue in many regions.

Treatment is most effective when begun early in the course of the disease. Cases with neurological signs can be difficult to cure, due to the poor penetration of most of these drugs into the CNS. Depending on the dose and other factors, treatment may be clinically curative without completely eliminating the parasite, and relapses are possible.

## Control

### *Disease reporting*

Veterinarians who encounter or suspect African animal trypanosomiasis should follow their national and/or local guidelines for disease reporting. In the U.S., this disease should be reported immediately to state or federal authorities.

### *Prevention*

Although most of the organisms that cause African animal trypanosomiasis can only become established where tsetse flies are present, *T. vivax* has become endemic in some areas where it is transmitted mechanically by other biting flies. Stamping out (e.g., quarantines, movement controls and the euthanasia of infected animals), combined with vector controls, might be able to eliminate this organism from a disease-free area if its introduction is recognized promptly. Once it has entered vector populations, eradication is usually impossible.

In endemic areas of Africa, trypanosomiasis is primarily controlled by reducing the numbers of tsetse flies with traps, insecticides and other means, and by treating infected animals that act as reservoirs. Tsetse fly eradication campaigns have been conducted in some locations, with varying success. Repellents, such as tsetse repellent collars for cattle, are also being investigated. In addition, the impact of this disease can be reduced by good animal management, with adequate nutrition and the treatment of concurrent illnesses, as well as by selecting breeds resistant to clinical signs ("trypanotolerant" breeds). Chemoprophylaxis with antiparasitic drugs, which may be combined with vector control (e.g., deltamethrin-impregnated collars in dogs), is sometimes used to protect susceptible livestock or dogs introduced to an endemic area.

It is difficult to control the many biting flies that transmit *T. vivax* mechanically outside tsetse fly areas, but some degree of protection might be provided by insecticides/repellents, traps, insect screens/netting in stables, and other insect controls. Mechanical vectors are most infective soon after feeding on an infected host, and the highest probability of transmission is to nearby hosts. Tabanids, which are thought to be the most important insect vectors, are persistent feeders and do not usually leave one animal to bite another more than 50 meters away. For this reason, it may be useful to separate infected from uninfected animals, and to keep highly susceptible species away from herds that may be infected subclinically.

## Morbidity and Mortality

Infections with trypanosomes tend to be most prevalent where these organisms circulate in tsetse flies and less common where they are only transmitted mechanically. Some animals remain subclinically infected, and may or may not eventually clear the organism, while others develop chronic illnesses of varying severity or acute cases that may be rapidly fatal. Asymptomatically infected animals sometimes develop clinical signs if they are stressed by malnutrition, concurrent illnesses, transport or overwork. Recently infected ruminants often respond promptly to treatment, but chronic cases tend to recover more slowly.

Factors that can affect the severity of the illness include the virulence of the isolate, its dose, and various characteristics of the host, such as previous exposure to trypanosomes, concurrent infections and general health. Some breeds of cattle and small ruminants in endemic areas are genetically resistant to the development of clinical signs, a phenomenon known as trypanotolerance. Some trypanotolerant breeds tend to self-cure and eliminate the organism; others can remain persistently infected while developing few or no signs of illness and maintaining productivity. Trypanotolerance can be specific to an organism, and is not absolute; trypanotolerant animals may require treatment in areas where the challenge intensity is high.

In fully susceptible, untreated ruminants, trypanosomes sometimes result in 50-100% mortality within months, especially when poor nutrition or other factors contribute to debilitation. However, African animal trypanosomiasis is now mostly a disease of high morbidity but low mortality in Africa, due to the availability of antitrypanosomal drugs.

While epizootics with high fatality rates do occur, especially when susceptible livestock are introduced into endemic regions or tsetse flies spread into a new area, they seem to be infrequent. This disease overall seems to be milder in South America, though outbreaks can be an issue in some areas and reproductive losses may also be significant, with mortality from abortions and neonatal deaths exceeding 50% in some outbreaks.

The patterns of disease in other animals range from sporadic clinical cases to outbreaks. Some dogs in Africa develop acute or chronic illnesses; however, reported infection rates in this species range from < 10% to 30%, suggesting that mild or subclinical infections might also be common. Dogs from outside this region appear to be more susceptible. In one analysis, 13% of 148 French military dogs sent to Africa with only vector control as protection became infected and 3% died of trypanosomiasis. (The infection rate decreased to 2% when dogs were protected by both chemoprophylaxis and deltamethrin-treated collars.) Adult pigs often seem to be relatively resistant to clinical signs, but young animals can be more susceptible.

Among wildlife, clinical cases are seen occasionally in captive animals, but are infrequently reported in free-living hosts. Although the stress of captivity might contribute to higher susceptibility, sporadic deaths and illnesses in free-living wildlife are probably underdiagnosed. Wild species that did not co-evolve with trypanosomes are more likely to become ill. In some incidents, trypanosome-naive wild white rhinoceroses (*Ceratotherium simum*) moved into tsetse habitats developed severe or fatal illnesses, though infections in healthy indigenous black rhinoceros (*Diceros bicornis*) often seem to be subclinical.

## Internet Resources

The Merck Veterinary Manual

United States Animal Health Association.
Foreign Animal Diseases

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

OIE Reference Laboratory for Animal Trypanosomoses of African origin

WOAH Terrestrial Animal Health Code

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