

African Animal Trypanosomiasis

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Importance

African animal trypanosomiasis (AAT) is a parasitic disease that causes serious economic losses in livestock from anemia, loss of condition and emaciation. Many untreated cases are fatal. AAT is found mainly in those regions of Africa where its biological vector, the tsetse fly, exists. One organism, *Trypanosome vivax*, has become established in South America, where it is transmitted by biting flies acting as mechanical vectors. Protecting animals from trypanosomiasis is difficult in endemic areas, as bites from tsetse flies and a variety of other insects must be prevented. A tsetse fly eradication program being conducted in Africa may help control this disease, as well as other forms of trypanosomiasis that affect humans.

Etiology

Trypanosomes are protozoan parasites in the family Trypanosomatidae. Most trypanosomes are transmitted by tsetse flies. Two tsetse-transmitted parasites, *T. brucei gambiense* and *T. brucei rhodesiense*, cause human African trypanosomiasis (HAT)/sleeping sickness, which affects both humans and animals.

The remaining tsetse-transmitted trypanosomes primarily affect animals and cause African animal trypanosomiasis. The most important species in this disease are *Trypanosoma congolense*, *T. vivax* and *T. brucei* subsp. *brucei*. *T. congolense* can be classified into three types, which are called the savannah, forest and kilifi types. Other species such as *T. simiae* and *T. godfreyi* can also cause AAT. Some trypanosome infections in Africa cannot be identified as any currently recognized species. Concurrent infections can occur with more than one species of trypanosome.

Species Affected

Trypanosomes can infect all domesticated animals; clinical cases have been described in cattle, water buffalo, sheep, goats, camels, horses, donkeys, alpacas, llamas, pigs, dogs, cats and other species. In parts of Africa, cattle are the main species affected, due to the feeding preferences of tsetse flies; in effect, they can shield other domesticated animals such as goats and pigs from the effects of trypanosomiasis. More than 30 species in the wild or zoos, including ruminants such as white-tailed deer, duikers, antelope and African buffalo, as well as wild Equidae, lions, leopards, warthogs, capybaras, elephants, nonhuman primates and various rodents are also known to be susceptible to infection. *T. vivax* DNA has been found by PCR in crocodiles and monitor lizards (*Varanus ornatus*) in Africa, but whether this organism can become established in reptiles or it is merely inoculated transiently by insects remains to be determined. Experimental infections can be established in laboratory animals including mice, rats, guinea pigs and rabbits.

The host preferences of each trypanosome species may differ, but *Trypanosoma congolense*, *T. vivax* and *T. brucei brucei* have a wide host range among domesticated animals. *T. godfreyi* and *T. suis* occur in pigs. *T. simiae* appears to be most important in pigs, but it has also been reported by PCR in camels, horses and cattle.

Geographic Distribution

Trypanosomes can be found wherever the tsetse fly vector exists. Tsetse flies are endemic in Africa between latitude 15° N and 29° S, from the southern edge of the Sahara desert to Zimbabwe, Angola and Mozambique. Trypanosomes, particularly *T. vivax*, can spread beyond the “tsetse fly belt” by transmission through mechanical vectors. *T. vivax* is also found in South and Central America and the Caribbean, areas free of the tsetse fly.

Transmission

Most trypanosomes must develop for one to a few weeks in tsetse flies (*Glossina* spp.), which act as biological vectors. When an infected tsetse fly bites an animal, the parasites are transmitted in the saliva. Trypanosomes can also be spread by fomites and mechanical vectors including surgical instruments, needles, syringes and various biting flies including horse flies (Tabanidae).

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T. vivax in South America does not require tsetse flies to develop; it is transmitted mechanically. The most important vectors are thought to be Tabanidae and stable flies (*Stomoxys* spp.). In Africa, the relative importance of mechanical transmission for *T. vivax* is controversial.

The immune response is unable to completely eliminate trypanosomes, and animals can become inapparent carriers. These inapparent infections can be reactivated if the animal is stressed. Transplacental transmission can also occur.

Incubation Period

The incubation period for African animal trypanosomiasis ranges from 4 days to approximately 8 weeks. Infections with more virulent isolates have a shorter incubation period.

Clinical Signs

Most cases of trypanosomiasis are chronic, but acute disease, which may be fatal within a week, can also occur. The first sign of trypanosomiasis may be a localized swelling (chance) at the site of the fly bite, but this usually remains unnoticed. The primary clinical signs are an intermittent fever, signs of anemia, lymphadenopathy and weight loss. Animals lose condition and become progressively emaciated. Milk yield may be decreased in dairy animals. Neurological signs, dependent edema, cardiac lesions, diarrhea, keratitis, lacrimation, appetite loss and other clinical signs have also been reported. Effects on reproduction include abortions, premature births and perinatal losses, as well as testicular damage in males. Deaths are common among chronically infected animals, and animals that recover clinically may relapse when stressed. Sudden deaths have been reported in small ruminants infected with *T. vivax*. Trypanosomes can cause immunosuppression, and concurrent infections may complicate this disease.

An acute hemorrhagic syndrome has been reported among cattle infected with *T. vivax* in Africa. Affected animals have enlarged lymph nodes and signs of severe anemia, and they develop widespread visceral and mucosal hemorrhages, particularly in the gastrointestinal tract. In one outbreak, the main hemorrhagic sign was bleeding from the ears. Weight loss can be severe. This syndrome can be rapidly fatal.

Few clinical cases have been described in dogs and cats. A chronic infection in an imported Jack Russell terrier, which may have been exacerbated by prednisone treatment, was characterized by an acute, fatal episode of severe anemia and abdominal distension with hepatosplenomegaly and ascites. The dog is reported to have had mild, fluctuating abdominal distension for six months. In two dogs, the clinical signs included acute hemorrhagic vomiting and diarrhea, with seizures; the stress of travel may have resulted in the exacerbation of a chronic

condition. Subclinical infections have also been reported in this species.

Post Mortem Lesions [Click to view images](#)

The gross lesions are nonspecific. In the acute stage, the lymph nodes and spleen are enlarged, and petechiae are common on the serosal membranes, particularly in the peritoneal cavity. More chronic cases may have serous atrophy of fat and signs of anemia, and the lymph nodes may be enlarged, normal or atrophied. Subcutaneous edema, excessive fluid in the body cavities, pulmonary edema and an enlarged liver may also be seen. Wasting or emaciation is common. Some trypanosomes can directly damage tissues, resulting in lesions such as keratitis or cardiac damage. Immune complexes also cause inflammation and damage in a variety of tissues, including the kidneys and blood vessels.

Morbidity and Mortality

Morbidity and mortality vary with the breed of the animal, as well as the strain and dose of the infecting organisms. Some breeds of African cattle and small ruminants are genetically resistant to the development of clinical trypanosomiasis, a phenomenon known as trypanotolerance. Trypanotolerant breeds of cattle include West African Shorthorn (which may also be known as Muturu, Baoule, Laguna/ Lagune, Namchi, Samba/ Somba or Dahomey cattle) and N'Dama.

Although acute cases can be seen, trypanosomiasis is often a chronic disease in susceptible animals. The morbidity rate is high, and many untreated animals infected with *T. vivax*, *T. brucei brucei* or *T. congolense* eventually die. In cattle infected with some strains, the mortality rate can reach 50-100% within months after exposure, particularly when poor nutrition or other factors contribute to debilitation. Other isolates may cause subacute disease, followed by spontaneous recovery and the disappearance of trypanosomes from the bloodstream. The roles of different trypanosome species and mixed infections on the severity of disease in each host species are incompletely understood. However, some of the savannah type strains of *T. congolense* are among the most virulent isolates for cattle. West African isolates of *T. vivax* can cause acute disease in cattle, while East African isolates tend to result in chronic infections. A few East African isolates of this organism cause an acute hemorrhagic syndrome with a mortality rate of 6-35%.

Different patterns of disease occur in Africa and South America. In South America, where *T. vivax* is transmitted strictly by mechanical vectors, trypanosomiasis occurs in cattle as epizootics separated by a few years when the disease appears to be silent and subclinical. After a series of outbreaks, animals tend to be immune and maintain the parasites in very low numbers. Once several years have passed and most of the population is once again susceptible, the occurrence of high parasitemia in a number of carriers, when combined with the proliferation of mechanical

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vectors, leads to another epizootic. When these conditions are met, the morbidity rate can be very high. In Africa, where the parasites are transmitted by tsetse flies, trypanosomiasis appears as a persistent endemic disease. Although seasonal changes can occur in the morbidity rate, the disease is never completely absent. Whether cyclical patterns for *T. vivax* will emerge in Africa with the eradication of tsetse flies is unknown.

Diagnosis

Clinical

Trypanosomiasis should be a consideration in endemic areas when an animal is anemic and in poor condition. Animals imported from these areas can be subclinical carriers and may become ill when they are stressed.

Differential diagnosis

Other diseases that cause anemia and weight loss including babesiosis, anaplasmosis, hemochromatosis, theileriosis, malnutrition or helminth infestation should be ruled out.

Laboratory tests

A presumptive diagnosis can be made if pathogenic trypanosomes are observed in the blood, lymph nodes or other tissues by direct examination. The organisms must be distinguished from avirulent trypanosomes such as *T. theileri*. Parasites may be found in wet blood films and thick or thin stained blood smears. Wet unstained blood films are examined by light microscopy (condenser aperture, phase-contrast or interference contrast), using 400x power to detect the motile trypanosomes. Thin smears fixed in methyl alcohol can be stained with Giemsa or May-Grünwald stain. Thick smears are air dried and stained with Giemsa. Other stains may also be used. Thick or thin films should be examined by light microscopy at high magnification. A presumptive species identification can be made based on the parasite's morphology in thin films. More than one species may be found. Thick films have the advantage of being able to detect parasites in low numbers; however, the morphology of the parasite is difficult to determine. Trypanosomes can be difficult to find in the blood, especially in animals with chronic disease or healthy carriers. Detection can be improved with parasite concentration techniques including mini anion-exchange chromatography, hematocrit centrifugation, the quantitative buffy coat method or the dark-ground/phase-contrast buffy coat technique. The latter three methods rely on the concentration of trypanosomes near the buffy coat after centrifugation.

Animal inoculation studies in rats or mice may occasionally be used to diagnose AAT. This technique is very sensitive and can detect low levels of parasites, but it is also time consuming.

Most currently used tests do not identify trypanosomes to the species or subspecies level. *In vitro* cultivation, with

species identification by techniques such as restriction fragment length polymorphism (RFLP), isoenzyme electrophoresis or DNA hybridization, is possible for some trypanosomes. Polymerase chain reaction (PCR) assays can identify parasites at the genus, species or subspecies level. PCR may not identify all species or some genotypes. Antigen detection techniques are not currently in use.

Serology can be helpful in diagnosis. Two serological tests, the indirect fluorescent antibody test and enzyme-linked immunosorbent assays (ELISA), are routinely used to identify seropositive cattle. Because reactions to previous infections can be detected, serology is useful only for a presumptive diagnosis. Cross-reactions can occur with other trypanosomes such as *T. theileri*, which is not pathogenic, and *T. evansi*, which causes surra.

Samples to collect

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease. Although the trypanosomes that cause African animal trypanosomiasis are not known to be zoonotic, trypanosomes related to *T. brucei brucei* infect humans, and non-zoonotic trypanosomes might cause disease in people with certain genetic defects. Precautions are recommended when handling blood, tissues and infected animals.

Anticoagulated fresh blood, dried thin and thick blood smears, and smears of needle lymph node biopsies can be submitted from live animals. Blood may also be submitted for PCR. If possible, samples should be collected from several affected animals. Trypanosomes are most likely to be found in the blood by direct examination during the early stages of the infection. They are less likely to be detected in chronically ill animals, and are almost never seen in healthy carriers. The use of trypanocidal drugs also decreases the probability of finding the parasites. Anticoagulated blood to be used for direct examination should be kept cool and sent to the laboratory as soon as possible. Tissues can be collected at necropsy. Serum can be collected for serology.

Recommended actions if African animal trypanosomiasis is suspected

Notification of authorities

Trypanosomiasis must be reported to state or federal authorities immediately upon diagnosis or suspicion of the disease.

Federal: Area Veterinarians in Charge (AVIC):
www.aphis.usda.gov/animal_health/area_offices/

State Veterinarians:

www.usaha.org/Portals/6/StateAnimalHealthOfficials.pdf

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Control

Most trypanosomes are transmitted by tsetse flies, and can only become established in areas where these vectors exist; however, *T. vivax* does not require tsetse flies and can become endemic in other areas. If the outbreak is detected early, this organism might be eradicated by quarantines, movement controls and the euthanasia of infected animals. Trypanosomes cannot survive for long periods outside the host, and disappear quickly from the carcass after death. Controlling arthropod vectors is important in preventing new infections.

In endemic areas of Africa, African animal trypanosomiasis can be controlled by reducing or eliminating tsetse fly populations with traps, insecticides and other means, and by treating infected animals with antiparasitic drugs. Drug resistance can be seen. The selection of trypanotolerant breeds of cattle can lessen the impact of trypanosomiasis. Animals given good nutrition and rested are more likely to recover rapidly than undernourished and stressed animals. No vaccines are available for trypanosomiasis.

A tsetse fly eradication campaign, the Pan African Tsetse and Trypanosomiasis Eradication Campaign (PATTEC), is being conducted in Africa. Its goal is to eliminate tsetse flies from the continent and, with them, to eliminate most animal trypanosomes. Because *T. vivax* in South America does not require tsetse flies, this technique would not be effective there.

Public Health

The trypanosomes that cause African animal trypanosomiasis are not considered to be pathogenic for humans; however, disease might be possible in people with certain genetic defects.

Internet Resources

Food and Agriculture Organization of the United Nations (FAO). Manual for the Recognition of Exotic Diseases of Livestock

<http://www.spc.int/rahs/>

The Merck Veterinary Manual

<http://www.merckvetmanual.com/mvm/index.jsp>

United States Animal Health Association.

Foreign Animal Diseases

<http://www.usaha.org/pubs/fad.pdf>

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>

OIE Terrestrial Animal Health Code

<http://www.oie.int/international-standard-setting/terrestrial-code/access-online/>

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