The trypanosomosis in the goat. Current status

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Background

Trypanosomosis is a major constraint on ruminant livestock production in Africa, including goat production. The impact of South American trypanosomosis on goats is largely unexplored and is only briefly discussed. Current knowledge on the important animal and human trypanosomes related to their pathogenicity for goats is summarized in Table 1.

Etiology

In Africa, cyclic transmission of the parasite to mammalian hosts occurs via numerous species of tsetse flies (*Glossina* spp.) during feeding by the flies. Elsewhere in the world, mechanical transmission by other species of biting flies is the primary mode of infection.
T. congolense is the most common trypanosome of goats in Africa. *T. vivax* is the second most common. Natural infection of goats with *T. brucei* is also sporadically reported. Goats are susceptible to *T. uniforme*, in Uganda and Zaire, but only mild infections occur. *T. simiae*, a trypanosome of swine and camels is transmissible to goats by either *Glossina* spp. or biting flies but causes mostly mild or subclinical disease (Smith and Sherman, 1994).

Goats and other domestic animals are relatively resistant to *T. brucei gambiense*. When infection does occur, the clinical course is chronic. *T. brucei rhodesiense* is an uncommon cause of caprine disease. A nonpathogenic trypanosome, *T. theodori*, was found incidentally in goats in Israel. It is transmitted by a hippoboscid fly, *Lipoptena caprina*. This organism is morphologically similar to the common, nonpathogenic sheep trypanosome, *T. melophagium*.
Information on the pathogenicity of the trypanosomes that occur outside of Africa, primarily in South and Central America is limited. *T. cruzi*, which is cyclically transmitted by reduviid bugs, as well as *T. evansi* and *T. equiperdum*, which are mechanically transmitted, cause disease in humans, camels and horses respectively (Smith and Sherman, 1994). Their infectivity for goats is presumed to be low. Kids infected experimentally with *T. cruzi* showed no clinical signs of disease and carried the infection for 38 days (Diamon and Rubin, 1958). The goat is a natural host for *T. evansi*, but reports of the disease, surra, in goats , are lacking (Levine, 1973). Only one non-African trypanosome, *T. vivax*, is primarily pathogenic for ruminants, especially in cattle. The pathogenicity for goats is not well investigated.

Epidemiology

Trypanosomosis in Africa follows the distribution and intensity of the various species of the tsetse fly. Approximately 10 million km^2 or 37% of the African continent is tsetse-infested. This area includes 38 countries. Various estimates suggest that the livestock-carrying capacity of such areas in West and Central Africa could be increased five- to seven-fold by eliminating or controlling animal trypanosomosis (Griffin, 1978).

There are approximately 200 million goats in Africa with as many as 50 million in the tsetse-infested regions of the continent. Natural infections with *T. congolense*, *T. vivax*, or *T. brucei* resulting in clinical disease have been known in African goats since the turn of the

century. Until recently, however, the perception has persisted that goats are highly resistant to infection, that caprine trypanosomosis is only sporadic, and that the disease in goats is of little economic consequence (Griffin, 1978). This opinion is currently undergoing a critical reappraisal. Regional differences do exist in the prevalence of caprine trypanosomosis, but it can be high in some areas (Griffin and Allonby, 1979; Kramer, 1966). In general, caprine trypanosomosis is more common in East than West Africa. This is attributed to differences in feeding preferences between riverine species of *Glossina* and savannah species; the latter are more inclined to feed on goats (Smith and Sherman, 1994).

Goats may serve as a reservoir of trypanosome infection for other species. In the Sudan, goats infected with *T. congolense* developed a chronic form of disease from which many spontaneously recovered. When the organism was passaged from goats into calves however, acute fatal bovine trypanosomosis occurred (Mahmoud and Elmalik, 1977). Goats also have been implicated as a reservoir of *T. brucei rhodesiense*, transmissible to man (Robson and Rickman, 1973).

The economic impact of trypanosomosis on goat production is beginning to be studied. A Kenyan analysis demonstrated that goats receiving monthly chemoprophylaxis against trypanosomosis had significantly decreased mortality rates, increased weight gains, and improved reproductive performance compared to untreated control goats. Differences in performance were also noted between breeds in the study with indigenous breeds performing better than non-indigenous cross breeds (Kanyari et al., 1983).

The existence of inherent trypanotolerance in certain goat breeds is controversial. It is generally accepted that trypanotolerant breeds of cattle exist, particularly the N'dama of West Africa and the West African Shorthorn. This inherent ability to control parasitemia and minimize disease has not been clearly demonstrated for specific goat breeds, despite the general observation that some breeds of goats will readily survive in tsetse infested areas. Dwarf West African goats have been considered inherently trypanotolerant, yet they can be readily infected experimentally (Murray et al., 1982). While earlier studies suggested that indigenous goat breeds of East Africa may show inherent trypanotolerance, no evidence of genetic resistance was observed in a subsequent study with either natural or experimental challenge in East African, Galla, or East African goats cross bred with Toggenburg, Nubian, or Galla breeds (Whitelaw et al., 1985). One factor contributing to the perceived trypanotolerance of various goat breeds under field conditions may be the feeding preferences of *Glossina* spp. Flies may select other livestock over goats when mixed animal populations are present (Murray et al., 1984). The existence of true trypanotolerance in goats deserves additional careful investigation.

Pathogenesis

Trypanosomes fall into two groups regarding their ability to produce disease. The hematic group, which includes *T. congolense* and *T. vivax*, remain confined to the circulation after introduction into the bloodstream by feeding *Glossina* spp. The disease produced in these infections is characterized by anemia. The humoral group, which includes *T. brucei*, is more invasive, with trypanosomes found in intercellular tissue and body cavity fluids after initial infection. Anemia in these cases is overshadowed by marked inflammatory, degenerative, and necrotic changes.

Anemia in trypanosomosis may be due to extravascular hemolysis and erythrophagocytosis, and also decreased erythropoiesis in chronic infections (Kaya et al., 1977). The destruction of red blood cells may result from both non-immune and immune-mediated mechanisms. Hemorrhage secondary to disseminated intravascular coagulation (DIC) may also contribute to anemia. Thrombocytopenia, microthrombus formation, and hemorrhage suggestive of DIC have been observed in caprine trypanosomiasis due to *T. vivax*

(Van der Ingh et al., 1976; Veenendaal et al., 1976). Anemia may be exaggerated by hemodilution because of expansion of blood and plasma volumes, which increased, respectively, 29% and 44% in goats with subacute *T. vivax* infection (Anosa and Isoun, 1976). The pathogenesis of inflammation and tissue damage by humoral trypanosomes such as *T. brucei* is complex and have been reviewed elsewhere (Soulsby, 1982). Immunosuppression can occur in trypanosomosis. *T. vivax* and *T. brucei* infection of goats resulted in depressed responses to mitogen stimulation in lymphocyte transformation tests (Diesing et al., 1983; van Dam, 1981). T. evansi Impaired immune function may aggravate the severity of concurrent infections. This was suggested by evidence of higher mortality rates and parasite loads in goats concurrently infected with *T. congolense* and *Haemonchus contortus* than in goats infected with only one or the other parasite (Griffin et al., 1981a; Griffin et al., 1981b).

One of the most notable features of trypanosomosis is the successive waves of parasitemia that occur every few days in animals that survive initial infection. Each wave of parasitemia is followed by an increase in circulating antibody that temporarily reduces the parasitemia. The effect is only temporary, however, because cyclically transmitted trypanosomes are capable of repeatedly altering their surface antigens (surface coat glycoproteins) and thereby evade the host immune system sufficiently to avoid total elimination of infection.

Clinical signs

Main clinical pictures produced by the different trypanosomes are summarized in Table 1.

Diagnosis

Anemia and emaciation in goats from endemic areas suggest the diagnosis of trypanosomosis. Definitive diagnosis is based on identification of trypanosomes in blood smears or tissues. Common parasitological detection tests for *Trypanosoma* spp. are also used in the goat. Wet film, blood smears examination (morphologic identification is better performed on thin smears), buffy coat, lymph node aspirates or mouse inoculation are commonly used. Serologic tests developed for the diagnosis of trypanosomosis include indirect hemagglutination test, a complement fixation test, an indirect fluorescent antibody test, ELISA and direct card agglutination test (CATT/*T. evansi*) and the indirect card agglutination test (LATEX/*T. evansi*).

Treatment

A variety of trypanocidal compounds are available for treatment, but no new drugs have been marketed for quite some time. Subsequently, drug resistance has become a significant problem. Compounds and dosages are formulated for single-dose use and treatment is usually on a herdwide basis because serial treatments on individual animals are difficult to carry out in the semi-nomadic livestock farming systems prevalent in endemic areas. Several of the drugs are locally irritating so subcutaneous injections should be given in areas of loose skin and intramuscular injections given deeply, avoiding vessels and nerves. Curative doses used in cattle are also appropriate for goats and sheep (Ilemobade, 1986). Diminazene aceturate is given intramuscularly as a 7% cold water solution at a dose of 3.5 mg/kg and is considered effective against the three major trypanosomes, as is quinapyramine dimethyl sulfate given intramuscularly as a 10% cold water solution at a dose of 10.0 mg/kg. Relapse of infection has been reported in goats treated with diminazene aceturate, presumably because of re-emergence of trypanasomes from the central nervous system where they were inaccessible to the drug during earlier treatment (Whitelaw et al., 1985).

Homidium chloride or homidium bromide are given in a 2% cold water solution at a dose of 1.0 mg/kg intramuscularly and are effective against *T. vivax* and *T. congolense*. Isometamidium chloride is also effective against the hematic trypanosomes when given at a dose of 0.25 to 0.75 mg/kg intramuscularly as a 1 or 2% cold water solution. This drug was shown to produce signs of shock or death in goats if given intravenously at doses greater than or equal to 0.5 mg/kg (Schillinger et al., 1985). Salicylhydroxamic acid with glycerol was found to be an unsatisfactory treatment for *T. vivax* in goats due to difficulties in administration and potential toxicity (van der Meer et al., 1980).

Control

Numerous constraints on control exist including reservoirs of infection in wild animal populations, the ability of trypanosomes to continuously alter their antigenic character thus confounding the development of suitable vaccines, a limited availability of effective drugs, the development of resistance to existing trypanocidal drugs, the difficult logistics of widespread tsetse control, lack of economic resources, poorly developed animal disease control programs, limited technical training programs, lack of international cooperation, and political instability (Murray and Gray, 1984; Doyle et al., 1984).

Currently, the major fronts in trypanosomosis control are reduction or elimination of tsetse populations and chemoprophylaxis of livestock. Tsetse fly control is accomplished by several methods, alone or in combination, including ground or aerial application of insecticides, such as chlorinated hydrocarbons and synthetic pyrethroids, tsetse trapping, and gamma-irradiated sterile fly release (Smith and Sherman, 1994).

Both isometamidium chloride and pyrithidium bromide will protect against infection with the three major goat trypanosomes for 2 to 4 months. The prophylactic dose of isometamidium is 1.0 mg/kg administered intramuscularly in a 1 or 2% cold water solution. Pyrithidium is given at an intramuscular dose of 2.0 mg/kg in a 2% solution of water that must be boiled. Quinapyramine chloride given subcutaneously in a 16.6% cold water solution at a dose of 7.4 mg/kg is prophylactic against *T. brucei* infection.

Despite intensive research, no effective vaccine is likely in the future because of the continuing problem of antigenic variation in trypanosomes. Given the obstacles to vaccination, there is a keen interest in the identification and promotion of tryanotolerant breeds of livestock in endemic areas, as discussed above in the section on epidemiology.

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Table 1. Trypanosomosis in goats. Adapted from Smith and Sherman, 1994.

Species	Major species affected	Geographic distribution	Vectors involved	Natural infection in goats	Experimental infection in goats	Clinical manifestation
Salivaria						
T. vivax	Domestic ruminants, camels, horses, antelope	Widespread in tropical Africa and South America	*Glossina* spp.	Common	Readily	Acute and chronic forms, mild to fatal
T. uniforme	Domestic ruminants, antelope	Zaire, Uganda	*Glossina* spp.	Yes	Not reported	Non-pathogenic or subclinical infection
T. congolense	All domestic animals, wild game	Widespread in Tropical Africa	*Glossina* spp.	Common	Readily	Acute, subacute, and chronic forms, mild to fatal outcome
T. simiae	Domestic pigs, camels, wild warthogs	Widespread in tropical Africa	*Glossina* spp. and *Stomoxys, Tabanus* flies	Uncommon	Not reported	mainly subclinical or mild clinical disease
T. evansi	Camels, equines, dogs, water buffaloes	India, Far East, Near East, Philippines, North Africa, Central and South America	various biting flies	yes	yes	Subclinical, moderate or acute disease.
T. brucei	Domestic ruminants, horses, dogs and cats	Widespread in tropical Africa	*Glossina* spp.	Common but with strain variation	Yes, with strain variation	Noninfective to fatal outcomes
T. b. gambiense (West African sleeping sickness	Humans	Tropical West and Central Africa	*Glossina* spp. and various biting flies	Uncommon Goats resistant	Very difficult	Noninfective or a chronic form leading to death or spontaneous recovery
T. b. rhodesiense (East African sleeping sickness)	Humans	East and Southern Africa	*Glossina* spp.	Uncommon	Yes	Experimental infections subacute and fatal
T. equiperdum	Horses	Southern Africa (Namibia, Botswana ,South Africa) , Ethiopia, Central Asia, , Middle East, Russia	Venereal reported	Not reported	Not reported	Not reported
Stercoraria						
T. cruzi (Chagas disease)	Humans	South and Central America, sporadic in USA	Reduviid blood-sucking bugs	Not reported	Yes	No