

ANIMAL TRYPANOSOMIASIS IN AFRICA: AETIOLOGY AND EPIDEMIOLOGY

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ABSTRACT

The aetiology and epidemiology of African trypanosomiasis in bovine species are comprehensively presented. In addition, a critical review of the history and transmission of the disease is exhaustively discussed. The mystery of other epizootiological factors associated with bovine trypanosomiasis is highlighted. Four major elements were identified as important in the epizootiology of African animal trypanosomiasis namely the trypanosome, the tsetse fly, the mammalian host and the environmental factors. It was concluded that the phenomenon of high rate of resistance referred to as trypanosotolerance has genetic correspondence.

Keywords: Trypanosomiasis, Aetiology, Epidemiology, Haemoprotozoan, Trypanosotolerance, *Trypanosoma*

INTRODUCTION

Animal trypanosomiasis is an economically devastating disease and a major constraint to livestock production in tropical Africa (Esiebo and Saror, 1991). Trypanosomiasis is a parasitic disorder caused by haemoprotozoan belonging to the genus, *Trypanosoma* of the family Trypanosomatidae, that multiply in the blood stream, lymphatic vessels and tissues including the cardiac muscles and the central nervous system. This highly fatal protozoan disease is virulent, inoculable but not contagious (except dourine, a venereal trypanosomiasis of equines). Trypanosomes are pathogenic, not only for animals but also for man where they cause sleeping sickness. Most species of domestic animals are to some degree susceptible to trypanosomiasis transmitted by various haematophagous insects, mainly *Glossina* species commonly known as tsetse flies. These are considered to be the true intermediate hosts of these parasites.

Tsetse flies occur exclusively in Africa over an area approximately 10 million km², extending on both sides of the equator from 15°N to 30°S. They are of primary importance in the spread and epidemiology of this economically and socially important disease (Houre, 1976; Ilard, 1989; Anene *et al.*, 2000). Despite all efforts and some impressive achievements, the problem of trypanosomiasis persists and in some respects is unchanged (Wilson *et al.*, 1968). The total area occupied by tsetse fly (one third of Africa) has reduced the areas of the continent which would otherwise support additional 125, 000, 00 heads or more of cattle and double the present number of cattle kept in Africa and cited by Killick-Kendrick and Geofrey (1963). If cattle in these areas are naturally challenged by this parasite, they come down with symptoms of the disease with corresponding impairment of performance in varying degrees. In particular, the *Bos taurus* cattle of West Africa are reputedly less susceptible than *Bos indicus* (Zebu) introduced much later to Africa - and the European *Bos taurus* breeds. The low susceptibility is defined as trypanotolerance. Ndama, Boule, Ghana

shorthorn, Somba and Muturu belong to trypanotolerant breeds with unusual natural resistance to trypanosomiasis. On the other hand, the zebu and European breeds are considerably highly susceptible to natural infection with trypanosomiasis, often giving rise to catastrophic morbidity and mortality making the rearing of these breeds difficult if not impossible in trypanosomiasis endemic regions of Africa (Epstein, 1975). The aim of this study is to pool together relevant and current information on aetiology and epizootiology of animal trypanosomiasis with particular emphasis on bovine trypanosomiasis to guide future investigation.

MATERIALS AND METHODS

A comprehensive literature search was made from the Internet and serial materials of Nnamdi Azikiwe Library, University of Nigeria, Nsukka. Various journal articles, proceedings of learned societies of veterinary parasitology, WHO documents and textbooks were consulted vis-à-vis of the aetiology and epidemiology of animal trypanosomiasis in Africa.

RESULTS AND DISCUSSION

Aetiology: The morphology of African trypanosome has been described in details (Leeflang, 1975; Ugochukwu, 1983). Trypanosomes are unicellular, microscopic and elongated protozoa that move by the help of a single flagellum at the base of which is found a characteristic structure known as kinetoplast. They are obligatory parasites usually having two hosts, they multiply in the body fluids especially blood vertebrate host (Table 1) and live in the digestive tract of invertebrate host which is generally a biting insect (Houre, 1976; Ilard, 1989).

The pathogenic trypanosomes have been classified either as Stercoraria (posterior station trypanosomes) *Trypanosoma theleri* which is mildly pathogenic to domestic and 20 wild ruminants (Table 1) and Salivaria (anterior station trypanosomes which is pathogenic to both domestic and wild animals (Table 1).

Table 1: Distribution of *Trypanosoma* Species among Vertebrate Host

Species	Host	Area	Reference
<i>T. congolense</i>	Goats	Nigeria	Ugochukwu (1983)
<i>T. vivax</i>	Cattle, Sheep, Goats, Horses	Nigeria East Africa	Roderick <i>et al.</i> (2004)
<i>T. simiae</i>	Pigs, Monkeys	Nigeria	Killick Kendrick and Geoffery (1963)
<i>T. gambiense</i>	Man	West Africa	ILARD (1990)
<i>T. rhodesiense</i>	Man	East Africa	ILARD (1990)
<i>T. brucei</i>	Dogs, Cats, Ruminants Monkeys	West Africa East Africa	Mulligan and Potts (1969) Nantulya (1990)
<i>T. suis</i>	Pigs	Nigeria	Killick, Kendrick and Geoffery (1963)
<i>T. evansi</i>	Camels, Horse	West Africa	Mulligan and Potts (1963)
<i>T. equiperdium</i>	Horse	West Africa East Africa	Mulligan and Potts (1963) Nantulya (1990)

Species of the genus, *Trypanosoma* are found in a wide variety of vertebrates. The majority are not pathogenic but some species are of considerable economic importance causing disease in man and animals. These trypanosomes mainly belong to three subgenera Trypanozoon, Duttonella, and Nanomas. The subgenus Trypanozoon contains *T. brucei*, two species of which *T. brucei gambiense* and *T. brucei rhodesiense* are responsible for sleeping sickness in man in Africa and one subspecies *T. brucei brucei* for infection in domesticated animals. *T. evansi* which is found in many parts of the world in a wide variety of animals. Also *T. equiperdium* and *T. suis* in the subgenera Nanomas *T. congolense* and *T. simiae* are the important members. The most important African trypanosomes species include: *T. vivax*, *T. congolense* and *T. brucei*. The important species causing diseases in cattle are *T. congolense*, *T. vivax* and *T. brucei* (Epstein, 1975; Clarkson, 1976; Obidike *et al.*, 2005).

Transmission: Animal trpanosomiasis has a sylvatic transmission cycle (Figure 1). The disease is maintained in ecological system which includes tsetse flies, woody vegetation and game or wild life. It is only when livestock is introduced into this system that tsetse fly will use the livestock as their food source and infect them with trypanosome. Trypanosomes except *T. equiperdium*, and *T. evansi* are transmitted cyclically by tsetse fly. Additionally, it is believed that biting flies including Tabanidae and Stomoxys also transmit the parasite mechanically. This activity is responsible for the persistence of *T. vivax* in areas of Africa free from tsetse flies as well

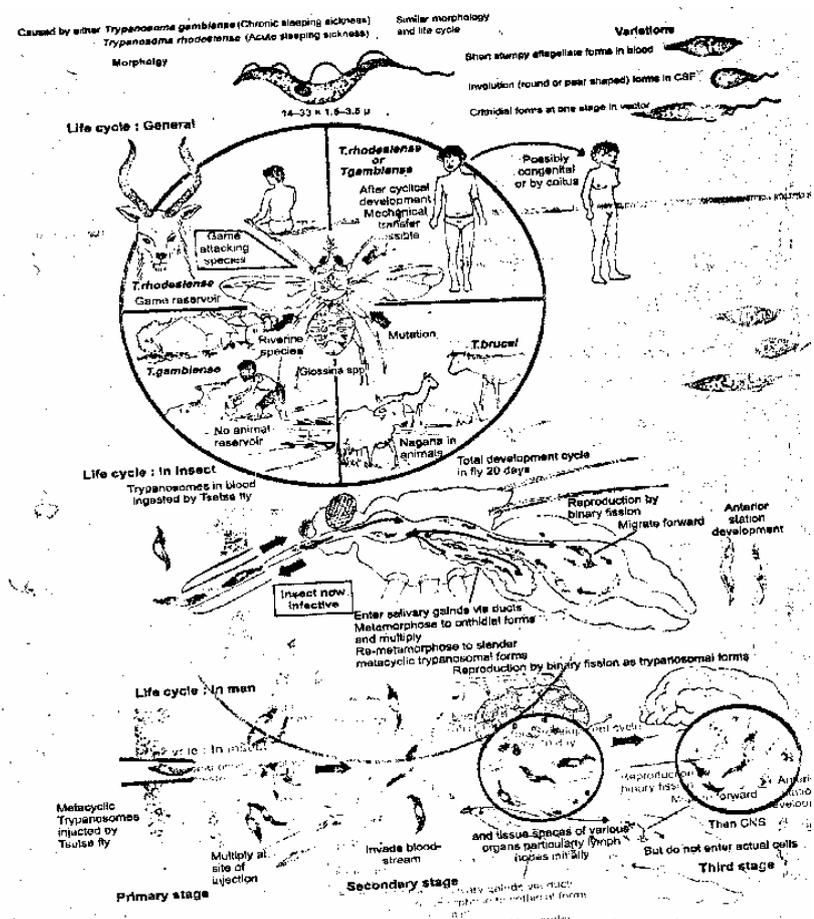


Figure 1: Life cycle of Trypanosomiasis (Sood, 2006)

as in several South American countries like Brazil, Colombia, and Guyana. For instance, two fatal outbreaks of bovine trypanosomiasis due to *T. vivax* were described in Maiduguri and Wadara both in Nigeria in closed herds maintained in the tsetse free Sahelian region (Maxie *et al.*, 1979). Also in Tanzania, pseudo-lumpy skin disease and acute *T. vivax* infections occurred simultaneously in a dairy herd which had no previous history of trypanosomiasis.

Where as tsetse fly might have introduced *T. vivax*, the failure to trap tsetse and the presence of large number of biting flies (*Stomoxys* and *Tabanus taenicola*) strongly suggested that in the outbreak both aetiological agents were transmitted mechanically (Conner and Mulkangi, 1986). The trypanosomes that cause disease in livestock and humans also infect some wild species which serve as a reservoir of infection that may in turn infect domestic animals and people. Many wild animals carry trypanosomes with no apparent ill-effect, in humans and most domestic livestock. However where such a harmless relationship with trypanosome, and their vector has not evolved, the pathogenic effects of infection are severe (Desowitz, 1960; Conner and Mulkangi, 1986). Wild life plays an important role as natural reservoir of trypanosome infection for domestic animals and man. It is generally believed that game animals can harbour trypanosomes and hardly suffer from the disease and the mechanism of this is unknown, although differences in the electrophoretic pattern of serum protein have been described (Desowitz, 1960). A similar tolerance is possessed by certain breeds of cattle, notably Ndama and Muturu which can be maintained in endemic areas where it is impossible to keep zebu cattle (ILARD, 1989). Blood meal analysis of tsetse flies caught in the wild shows that they feed on a wide variety of wild life host including primates (baboons, monkeys), suids, warthog, bush pig, red river hog, and giant bush pig) and various bovidae especially antelopes. Other studies have shown that pathogenic trypanosomes including *T. vivax*, *T. congolense* and *T. brucei* have been isolated from wild life in East Africa (Ashcroft, 1959; Anosa and Isoun, 1983).

However, various game animals such as red-buck, giraffe, bush pigs, kudu and bush buck are known reservoir hosts of pathogenic Africa trypanosome. Transmission is by bite of flies in the wild life. The trypanosome undergoes cyclical development in flies lasting 12 -35 days before they become infective. Though all species of tsetse fly are capable of cyclically transmitting trypanosomes, their importance as vectors depends on feeding habit, relative infectivity and distribution in relation to domestic stock. Additionally, it is believed that biting flies including tabanids and stomoxys also transmit the parasite mechanically. This is by direct transmission of infection by blood contaminating the mouth part of biting flies which are distributed during feeding.

The life cycle of the single-celled trypanosome is complex in both the tsetse fly vector and mammalian host, trypanosomes undergo a series of transformations into different forms As flies feed on animals infected with the parasite, they take up blood containing trypanosomes which then completes the life cycle (Houré, 1976; ILLARD, 1990).

Prevalence and Epidemiology: Large areas of Africa, approximately 4 million km² have been rendered unsuitable for livestock production by trypanosomes.

Climate and vegetation play a major role in the distribution of Tsetse fly. Other factors include presence of wild-life for food, types of soil for breeding, presence of predators and diversity of human population (Davis, 1977). The behaviour of tsetse flies varies from species to species in the same vegetational zone. The knowledge of tsetse ecology is important in the control measures most adopted in a particular zone or locality. Some workers have shown that as one moves from the north to the south, the feeding pattern of *G. tachinoides* changed from preference for man to one for domestic and wild animals. The genus, *Glossina* can be divided according to their habitat into (a) the forest species (*Fusca* group) comprising, of 12 species, (b) The riverine species (*palpalis* group) consisting of 5 species and (c) the savanna (the *mortisans* group) consisting of 5 species. The group name already give an indication of their distribution, the forest species are found in 500 km² wide coastal belt of tropical rainforest which stretches from Guinea Bissau to Nigeria in West Africa and further to the Cameroon, the central African Republic, large regions of Zaire and Angola as well as Congo, Gabon and Equatorial Guinea (Figure 2). The distribution of riverine species on one hand coincides, with that of forest flies, but on the other hand it clearly goes beyond the North and South along rivers on gallery forest, and even beyond the distribution of savanna species. Savanna species appear in the savanna belt which links up with the forest species region to the North and east especially, but primarily in the south-east.

The four most important species of *Glossina* in Nigeria are *G. palpalis*, *G. tachinoides*, *G. mortisans* a submortalans and *G. longipalis* (Davis, 1977). Surveys have shown that there is a wide geocological distribution of animal trypanosomiasis in Nigeria stretching from the mangrove forest to the Sudan savanna, owing to the presence of tsetse flies in these areas (Davis, 1977). Only about 1/5th of the northern Sahel savanna and the plateau of Mambilla, Jos and Obudu are free from tsetse fly (Davis, 1977; ILARD, 1990). However, cases of Trypanosomiasis have been reported on the Jos plateau (Anene *et al.*, 1991) and in the Sahel around Maiduguri (Maxie *et al.*, 1979). These unusual occurrences of animal trypanosomiasis have been attributed to movement of cattle from tsetse fly infested to tsetse fly free areas (Anene *et al.*, 1999).

Conclusion: Four major elements influence the epizootiology of African animal trypanosomiasis namely the trypanosome, the tsetse fly, the mammalian host and the environmental factors. Cattle are the primary victim of trypanosomiasis in West and East Africa and in South America. Sometimes outbreaks also occur in other species: horses, goat, dog, sheep and camel (Anosa, 1989). The phenomenon of high rate resistance to trypanosomiasis is termed trypanotolerance. Trypanotolerance has a genetic correspondence since crosses between trypanotolerant cattle and susceptible cattle show a high level of trypanotolerance (Anosa and Obi, 1980).

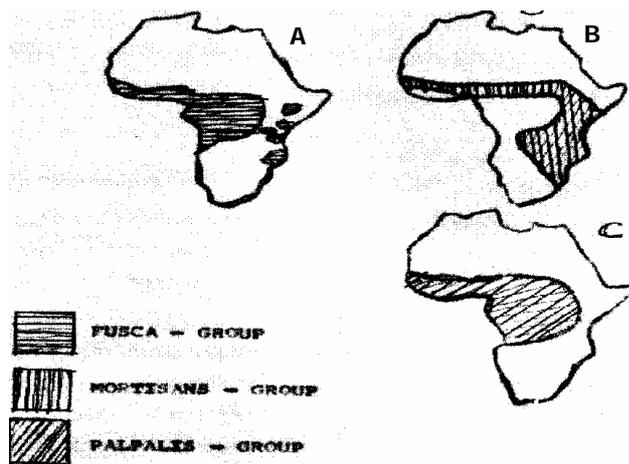


Figure 2: Testse fly occurrence in Africa

Trypanotolerance is however not absolute and in a sense represents only a potential, thus typanotolerant adults raised in tsetse fly free areas and subsequently infected by trypanosome have been shown to develop severe disease comparable to that shown by susceptible breeds (Desowitz, 1960; Ndoutamia *et al.*, 1993; Tabel *et al.*, 2000). Other factors which influence the prevalence of the disease in animal age and individual factors. Thus, young cattle are less frequently affected than older cattle. A study showed that prevalence decreased progressively from cattle over two years old 1 to 2 years, 9 months to 1 year, and under 3 months. Similarly, it has been shown in several experimental studies with mice, sheep, goats, cattle and monkeys that individual animals of the same breed and age exposed to the strain of trypanosomiasis show considerable variation in severity of disease (Mulligan and Potts, 1969; Henson and Noel, 1979; Anosa and Obi 1980; Anosa and Kaneko, 1983b). In conclusion, further work is anticipated to unravel the mystery of other epizootiological factors associated with bovine trypanosomiasis in the African continent in particular and animal trypanosomiasis in general.

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REFERENCES

- ANENE, B. M., CHIME, A. B., JIBIKE, G. I. and ANIKA, S. N. (1991). Prevalence of trypanosomiasis in Zebu cattle at Obudu ranch, a tsetse free zone in Nigeria. *Preventive Veterinary Medicine*, 10: 257 – 260.
- ANENE, B. M., ONAH D. N. and NAWA, Y. (2000). Drug resistance in pathogenic African

- trypanosomes: What hope for the future? *Veterinary Parasitology*, 96(2): 83 – 100.
- ANENE, B. M., OGBUANYA, C. E., MBAH, E. S. and EZEOKONKWO, R. C. (1999). Preliminary efficacy trial of cymelasan in dogs and mice artificially infected with *Trypanosoma brucei* isolated from dogs in Nigeria. *Review Elevage Medicine Pays*, 52(20): 23 – 29.
- ANOSA, V. O. and ISOUN, T. T. (1983). Pathology of experimental *T. vivax* infections in sheep and goats. *Bulletin of Veterinary Medicine*, 3: 685 – 700.
- ANOSA, V. O. (1989). Prevalence, pathogenesis and pathology of African trypanosomiasis. *Proceedings of a preparatory workshop held in Vom, Plateau State, Nigeria 5 - 9th June, 1989*.
- ANOSA, V. O. and KANEKO, J. J. (1983a). Pathogenesis of *T. brucei* infection in deer mice (*P. maniculatus*): ultrastructural pathology of the spleen, liver, heart and kidney. *Bulletin of Veterinary Medicine*, 27: 773 – 788.
- ANOSA, V. O. and KANEKO, J. J. (1983b). Pathogenesis of *T. brucei* infection in deer mice (*Peromyscus maniculatus*): Haematologic, erythrocytic, biochemical and ion metabolic aspect. *Bulletin of Veterinary Medicine B*, 30: 685 – 700.
- ANOSA, V. O. and OBI, T. U. (1980). Haematological studies on domestic animals in Nigeria. The effect of age, breed and haemoglobin type on bovine haematology and anaemia. *Bulletin of Veterinary Medicine*, 27:773 – 788.
- ASHCROFT, M. T. (1959). The importance of African wild animals as a reservoir of trypanosomiasis. *East African Medical Journal*, 36: 289 – 297.
- CLARKSON, M. J (1976). Trypanosomes. *Veterinary Parasitology*, 42: 9 – 29.
- CONNER, R. J. and MULKANGI, D. J. A. (1986). Concurrent outbreak of pseudo-lumpy skin disease and acute *T. vivax* infection in cattle. *Tropical Animal Health Production*, 18: 127 – 132.
- DESOWITZ, R. S. (1960). Studies on immunity and host parasite relationship 1. The immunological response of resistant and susceptible breeds of cattle on trypanosomes. *Annals of Tropical Medical Parasitology*, 54: 281 – 291.
- EPSTEIN, H. (1975). *The origin of the domestic animals of Africa*. Volumes 1 and 2. Africana, New York.
- ESIEVO, K. A. N. and SAROR, D. (1991). Immunochemistry and trypanosomiasis. *Veterinary Bulletin*, 61: 765 – 777.
- HENSON, J. B. and NOEL, J. C. (1979). Immunology and pathogenesis of African Animal trypanosomiasis. *Advance in Veterinary Science and Comprehensive Medicine*, 22: 20 – 35.

- HOURE, C. A. (1976). Trypanosomes. *Veterinary Parasitology*, 42: 9 – 29.
- ILARD (1989). Problems of current control method ILARD Reports, 9: 31 – 51.
- ILARD (1990). Problems of current control method ILARD Reports, 8: 31 – 51.
- IWUALA, O. E. M. and ALOZIE, C. C. (1980). Control of arthropod vectors of diseases in Nigeria. *Bulletin of Animal Production in Africa*, 28: 197 – 213.
- KILLICK-KENDRICK, R and GEOFFREY, D. G. (1963). Observation of a close association between *Glossina tachinoides* and domestic pigs near Nsukka. *Annals of Tropical Medicine and Parasitology*, 57: 222 – 231.
- LEEFLANG, P. (1975). Bovine trypanosomiasis and livestock industry and in Northern Nigeria. *Nigerian Veterinary Journal*, 10: 20 – 30.
- MAXIE, M. G., LOSOS, G. J. and TABEL, H. (1979). Experimental bovine trypanosomiasis (*T. vivax* and *T. congolense*) 1. Symptomatology and clinical pathology. *Tropical Medical Parasitology*, 30: 275 – 282.
- MULLIGAN, H. W. and POTTS W. H. (1969). *The African trypanosomiasis*. Aleu and Uwin, London.
- NANTULYA, N. N. (1990). Trypanosomiasis in domestic animals: Problem of diagnosis. *Review of Science and Technology on International Epizootiology*, 2: 357 – 367.
- NDOUTAMIA, G., MOLOO, S. K., MURPHY, N. B. and PEREGINE, A. S. (1993). Derivation and characterization of a quinapyramine resistant clone of *Trypanosoma congolense*. *Antimicrobial Chemotherapy*, 37: 1163 – 1166.
- OBIDIKE, I. R., AKA, L. MOMOH, C. V. and EZEOKONKWO, R. C. (2005). Effects of *Trypanosoma brucei* infections and diaminazone acetate treatment on serum activities of certain enzymes. *Sahel Veterinary Science*, 4: 17 – 23.
- RODERICK, S., STEVENSON, P., MWENDIA, C. and OKECH, G. (2000). The use of trypanocides and antibiotics by Massai pastoralists. *Tropical Animal Health Production*, 32: 361 – 374.
- SOOD, R. (2006). *Textbook of medical laboratory technologist*. Jaycee Brothers, India.
- TABEL, H., KAUSHIK, R. S. and UZONNA, J. E. (2000). Susceptibility and Resistance to *Trypanosoma congolense* infections. *Microbial infections*, 2(13): 1619 – 1629.
- UGOCHUKWU, E. I. (1983). Trypanosomiasis in goats caused by *T. congolense*. *Nigerian Veterinary Journal*, 12: 25 – 30.
- WILSON, S. G., MORRIS, K. R. S., LEWIS, I. J. and KROG, E. (1968). The effects of trypanosomiasis on rural economy. *Bulletin of World Health Organization*. 28: 595 – 613.